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Editorial Policy

The *DrexelMed Journal (DMJ)* features the scholarly activities of our graduate medical education trainees. This journal was created to highlight the many interesting and diverse scholarly activities and research at Drexel University College of Medicine and its participating affiliates (listed above). Recognizing that scholarly activity takes many forms, the *Journal* aims to publish all such efforts, and welcomes original research, reviews, case reports, and technical reports alike.

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The lead author must be in a current, Drexel-affiliated, graduate medical education program either as a resident or fellow, (i.e. PGY-1, etc.) and must be in good standing with the program. It is the responsibility of the lead author to review the submission(s) with all listed authors prior to the final electronic submission to *DMJ*. *DMJ* does not assume any responsibility for the addition or omission of authors. It is the responsibility of the lead author to verify all conflicts of interest for every author listed on the paper.

If residents from another institution would like to participate, they should request their designated institution official (DIO) to communicate directly with the Vice Dean of GME at DUCOM: Dr. Mark Woodland (215) 762-3500.

Please refer to the *DrexelMed Journal* website for further detailed instructions regarding submission guidelines:

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Please refer to the *DMJ* website for further instructions on how to submit your work for next year's edition.

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DREXEL UNIVERSITY
COLLEGE OF MEDICINE

EDITORIAL COMMENTS

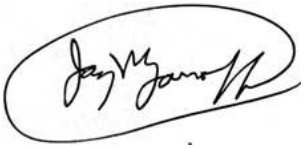
We are happy to present the seventh issue of the *DrexelMed Journal*, featuring the scholarly activities of the graduate medical education trainees of Drexel University College of Medicine, Hahnemann University Hospital, Abington Memorial Hospital, Allegheny General Hospital, Easton Hospital, Mercy Health System, Monmouth Medical Center, St. Christopher's Hospital for Children, St. Peter's University Hospital, and Virtua Health.

This edition of the *DMJ* includes again an impressive scope and range of types of projects, subject matter, and depth of research. Residents and fellows working with faculty in over fifteen specialties from eight institutions, not including collaborative physicians from outside of our GME scope, have taken the time to present their scholarly activity to us. This year's edition also includes a thoughtful poem about the heart.

We are very proud to bring you this edition of the *DrexelMed Journal*. We hope you take the time to peruse and appreciate the diverse richness of this year's edition and encourage you to submit your work for next year's edition!

David Berkson, MD
Editor-in-Chief
Associate Professor, Program Director, Family Medicine

Mark B. Woodland, MS, MD
Program Director, OB-GYN
Vice Dean, GME
Clinical Professor, OB-GYN



Jay M. Yanoff, EdD
Chief GME Officer, DIO
Hahnemann University Hospital

DEAN'S RECOGNITION

I offer my congratulations to Dr.'s Berkson, Woodland, and Yanoff for the 7th Edition of the DrexelMed Journal. Seven years ago when Drs. Yanoff and Woodland initiated this effort, it was whole heartedly supported by the College of Medicine to emphasize the scholarly activities of our residents. At the time it was in line with the Strategic Plan of the College of Medicine and the research mission of the College of Medicine. Since then, the challenge has been to continue to expand beyond the halls of our primary GME affiliates and I am pleased to see the participation of our affiliates continues to expand in this edition.

Finally, my personal appreciation to the many residents represented in this journal and to those of you in training who have ongoing scholarly activities. At Drexel, academic inquiry through research and innovation is part of our basic mission. We hope throughout your training programs and your professional careers that you continue your endeavors to move medicine forward.

Daniel V. Schidlow, MD
Annenberg Dean and Senior Vice President, Medical Affairs
Drexel University College of Medicine

Abstract: A Tough Act to Swallow

Nonihal Singh, MD, Mahesh Krishnamurthy, MD, Shelini Sooklal, MD
Easton Hospital: Department of Medicine

INTRODUCTION
Intentional foreign body ingestion in adults is commonly seen in those with psychiatric conditions, developmental delays and substance abuse.

CASE
A 34-year-old male ex-soldier presented to the emergency room (ER) with abdominal pain. The patient was previously diagnosed with an anxiety disorder, depression, bipolar disorder and posttraumatic stress disorder. The pain was described as epigastric, sharp and 10/10 in severity. An abdominal x-ray showed a foreign body in the stomach, which appeared to be a metallic butter knife (Fig.1). He denied any recollection of swallowing this object or any suicidal intent. Prior records showed that the patient was admitted on two occasions, each for swallowing a pen. The pens were successfully extracted via esophagogastroduodenoscopy (EGD).

Under general anesthesia, EGD was performed. Two half-knives were visualized lying on the greater curvature of the stomach; half of a butter knife and half of a regular kitchen knife. A snare and Magill forceps were used to retrieve the knives. The patient made an uneventful recovery.

Four days later he again presented to the ER with abdominal pain, and was found to have a large metallic spoon handle at the lesser curvature of stomach (Fig.2). This was similarly removed via EGD. A psychiatric evaluation was requested.

CONCLUSION
Impulse control disorders are often diagnosed when the patient is unable to refrain from performing an act that is harmful. Endoscopic removal is preferred. Perforation of the gastrointestinal tract is rare. Collaboration between medicine and psychiatry is vital to prevent these recurrent, costly admissions.

REFERENCES
1. Palese C, Al-Kawas FH. Repeat intentional foreign body ingestion: the importance of a multidisciplinary approach. Gastroenterol Hepatol (N Y). 2012 Jul;8(7):485-6
2. Huang BL, et al. Intentional swallowing of foreign bodies is a recurrent and costly problem that rarely causes endoscopy complications. Clin Gastroenterol Hepatol. 2010 Nov;8(11):941-6
3. Weiland ST, Schurr MJ. J. Conservative management of ingested foreign bodies. Gastrointest Surg. 2002 May-Jun;6(3):496-500

Figure 1. Abdominal X-ray showing two large metallic half knives in the stomach

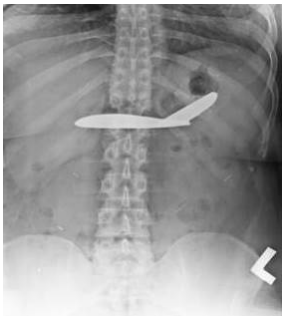
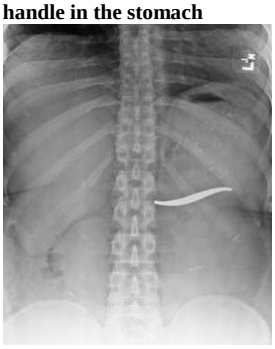


Figure 2. Abdominal X-ray showing large metallic spoon handle in the stomach



Abstract: An Unusual Cause of Vertebral Osteomyelitis

Ramya Mallareddy, MD, Harvey Hakim, MD
Easton Hospital: Department of Medicine

CASE
A 62-year-old male came to our hospital with fever, chills, mid-back pain and hypotension. An open appendectomy was performed 2 weeks prior to admission due to ruptured appendix. Post-operative course was complicated by E. coli septicemia. MRI of thoracic spine showed discitis at T9-T10 and vertebral osteomyelitis (Figs 1,2). ESR and CRP levels were elevated. No neurological manifestations were noted. The patient was treated with IV doripenem for 6 weeks. CT abdomen and pelvis was done which showed no intra-abdominal abscesses. Other sources of infection were ruled out.

DISCUSSION
Vertebral osteomyelitis was first recognized by Hippocrates and Galen. Pathophysiology includes hematogenous spread, direct inoculation, and contiguous spread. Vertebral bone is highly vascular thus prone to infection. Staphylococcus aureus is the most common organism followed by enteric gram negative bacilli. The most common symptom is back pain. Fever is present infrequently. Lack of treatment leads to contiguous spread to the epidural space causing neurologic deficits. Elevated ESR and CRP are sensitive indicators for infection. CRP level correlates to clinical response to therapy. MRI is the preferred modality for diagnosis. CT guided or open bone biopsy is warranted if blood cultures are negative. Optimal duration of antibiotic therapy is 6 weeks, but a longer course may be justified.

CONCLUSION
Vertebral osteomyelitis may develop in any bacteremic state and should be considered when a patient has suggestive signs and symptoms. Early diagnosis and prompt treatment is essential to avoid the development of epidural abscess and neurologic complications.

Figure 1

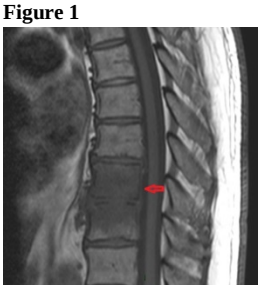
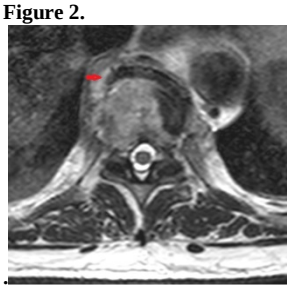


Figure 2.



Abstract: An Unusual but Critical Aneurysm in the Heart

Thein Aung, MD, Narpinder Singh, MD
Easton Hospital: Department of Medicine

CASE
57 year-old male with longstanding heart murmur was admitted for left lower extremity cellulitis. Apart from leg pain and swelling he denied chest pain and palpitations. He was febrile, tachycardic, had bilateral lower limb erythema, and had a pan-systolic, grade 3/6 murmur over the right upper parasternal region. Transthoracic echocardiography (TTE) showed a dilated aortic root with a right aneurysmal Sinus of Valsalva (SVA). Transesophageal echocardiography (TEE) showed a large 3x4 cm right SVA compressing the right ventricular outflow tract (RVOT) causing obstruction. He was immediately referred for corrective surgery.

DISCUSSION
SVA is a dilatation of aortic root caused by the lack of continuity between the middle layer of the aortic wall and the aortic valve. Incidence of SVA is between 0.15 to 1.5% in patients undergoing cardiac bypass surgery. It is most commonly found in the right coronary sinus, followed by noncoronary sinus, and rarely in the left coronary sinus. TTE and TEE have diagnostic accuracy of 75% and 90% respectively. SVA are asymptomatic unless they compress adjacent structures or form a thrombus. Unruptured SVA needs continuous monitoring as complications such as endocarditis, malignant arrhythmias, and acute coronary artery obstruction can occur. The most common complication is the formation of aorto-cardiac fistula, mostly into right heart chambers.

CONCLUSION
SVA is an uncommon but not infrequent finding on echocardiography. It is important to understand and recognize SVA on the routine echocardiography since the timely referral to corrective surgery is important to prevent disastrous complications.

REFERENCES
1. Ott D. Aneurysm of the sinus of Valsalva. Cardiac Surg Ann 2006; 9:165-176
2. Moustafa S, et al. Sinus of Valsalva aneurysms--47 years of a single center experience and systematic overview of published reports. Am J Cardiol. 2007 Apr 15;99(8):1159-64
3. Steves W. Congenital heart surgery nomenclature and database project: aortic aneurysm, sinus of valsalva aneurysm, and aortic dissection. Ann Thoracic Surg 2000; 69:S147-S163
4. Galicia-Tornell MM, et al. Sinus of Valsalva aneurysm with rupture. Case report and literature review. Cir. 2009 Nov-Dec; 77(6):441-5
5. Harkness J, et al. A 32 year experience with surgical repair of sinus of Valsalva aneurysms. J Cardiac Surg 2005; 20:198-204

Figure 1. Color doppler image of aortic insufficiency with Sinus of Valsalva Aneurysm

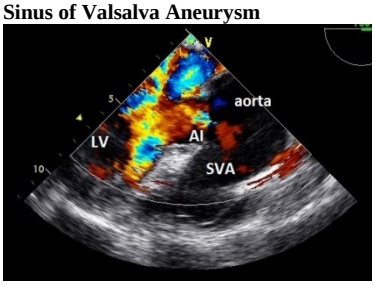
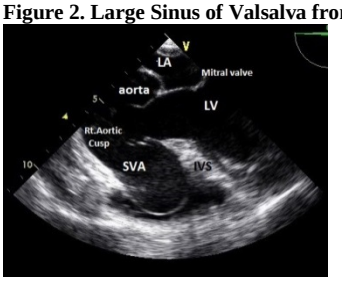


Figure 2. Large Sinus of Valsalva from right aortic cusp



Abstract: Comparison of Bronchoscopic and Non-bronchoscopic Methods of Obtaining Distal Airway Cultures in Tracheostomized Children

Olufunke Afolabi-Brown, MD*, Mikhail Kazachkov, MD**, Michael Marcus, MD**, Murali Pagala, PHD***, Jason Perlman, MD****, Peter Speciale*****
*St. Christopher's Hospital for Children: Pediatric Pulmonology
**Maimonides Infants and Children Hospital: Department of Medicine, Division of Pediatric Pulmonology
***Maimonides Medical Center: Department of Surgery
****Maimonides Infants and Children Hospital: Department of Medicine, Division of Pediatric Infectious Diseases
*****Maimonides Medical Center: Department of Medicine

INTRODUCTION
Distal airway secretions can be sampled by bronchoscopic bronchoalveolar lavage (B-BAL), blind protected BAL (BP-BAL) and tracheal aspirates (TA). We quantitatively compared cultures of distal airway secretions from BP-BAL, B-BAL, and TA and assessed efficacy of the three methods in diagnosing bronchitis in tracheostomized children.

METHODS
Children with tracheostomies scheduled for their surveillance bronchoscopy were enrolled in the study. Patients underwent BP-BAL using a double lumen plugged catheter followed by B-BAL and TA. Samples were sent for quantitative bacterial cultures. Amount of secretions present in the tracheobronchial tree was assessed visually based on a validated grading system. Diagnosis of bronchitis was made based on this grading score as well as on positive quantitative culture in the BAL fluid. Diagnostic agreement between cultures obtained by the three methods and visual grading scores was determined by kappa analysis.

RESULTS
20 patients were enrolled in the study. Diagnosis of bronchitis by visual grading score had substantial agreement (Kappa 0.7, concordance 85%) with BP-BAL , moderate agreement (Kappa 0.5, 75%) with B-BAL, and fair agreement (Kappa 0.3, 65%) with TA. Within the three techniques, the diagnostic agreement was substantial between BP-BAL and B-BAL (Kappa 0.76, 90%), but only moderate between BP-BAL and TA (Kappa 0.49, 80%). One-way ANOVA showed that BP-BAL had significantly lower pathogenic colonies ($P<0.05$) than either B-BAL or TA.

CONCLUSION
BP-BAL is potentially more accurate in diagnosis of bronchitis and allows for more accurate sampling of lower airway secretions in tracheostomized children.

REFERENCES
1. British Thoracic Society Bronchoscopy Guidelines Committee, a Subcommittee of Standards of Care Committee of British Thoracic Society. British thoracic society guidelines on diagnostic flexible bronchoscopy. Thorax. 2001;56 Suppl 1:i1-21

2. Chang AB, et al. A bronchoscopic scoring system for airway secretions--airway cellularity and microbiological validation. Pediatr Pulmonol. 2006;41(9):887-892
3. Pang JA, Cheng AF, Chan HS, French GL. Special precautions reduce oropharyngeal contamination in bronchoalveolar lavage for bacteriologic studies. Lung. 1989;167(5):261-267
4. Respiratory Care Services. Bronchoalveolar lavage using combicath. Kaleida Health.8
5. Hughes J, Jones AS, Morar P, Singh V, Van Saene R. Impact of tracheotomy on colonization and infection of lower airways in children requiring long-term ventilation: A prospective observational cohort study. . 1998;113(1):77+

Abstract: Correlation of the Pretest Clinical Score (4Ts) and the Appropriate use of Laboratory Studies to Diagnose Heparin-Induced Thrombocytopenia (HIT)

Jennifer Hawkins, DO, Fernando Garcia, MD, Joseph Jaworski, MD, Vanlila Swami, MD
Drexel University College of Medicine: Department of Pathology and Laboratory Medicine

INTRODUCTION
HIT is diagnosed with ELISA screening test and a confirmatory serotonin release assay (SRA). 4Ts scoring system is utilized to assess probability of heparin as a cause of thrombocytopenia. ELISA results with optical density (OD) >0.4 units are positive (0.40-1.00: weak positive).

OBJECTIVE
Use the 4T scoring system to evaluate the appropriateness of HIT testing in patients with heparin induced thrombocytopenia.

METHODS
55 patients' HIT test requisitions were reviewed between 08/01/2012 to 12/07/2012 and the 4Ts scoring system were obtained. The 4Ts clinical scoring system is based on four categories: thrombocytopenia, timing of thrombocytopenia, thrombosis and other causes for thrombocytopenia. Each category is given a score from 0 to 2, with a maximum score of 8. Pretest probability is classified as low <3 points; intermediate 4-5 points; or high 6-8 points.

RESULTS
46 patients (83.6%) had a low probability, 9 (16.4%) had an intermediate probability and 0 had a high pretest probability. 5 of 46 patients with low probability and 1 of 9 patients with intermediate probability had a weak positive ELISA (Table 1).

DISCUSSION
HIT is over utilized as other causes of thrombocytopenia should be ruled out first. Recommendations are: 1) All HIT testing requisitions should be accompanied with the 4Ts scoring evaluation. 2) HIT testing is indicated for intermediate and high pretest probability. 3) HIT testing is not indicated with low pretest probability. 4) SRA is reflexed if ELISA OD is >1.00 units. 5) Weak positive ELISA is repeated if HIT testing is warranted.

REFERENCES
1. Cuker A, Gimotty PA, Crowther MA, Warkentin TE. Predictive value of the 4Ts scoring system for heparin-induced thrombocytopenia: a systematic review and meta-analysis. Blood. 2012 Nov 15;120(20):4160-7
2. Lo GK, et al. Evaluation of pretest clinical score (4 T's) for the diagnosis of heparin-induced thrombocytopenia in two clinical settings. J Thromb Haemost. 2006 Apr;4(4):759-65

Table 1 . Pretest Probability and ELISA HIT Lab Results

	Low Pretest Probability Score ≤3 # of patients	Intermediate Pretest Probability Score (4-5) # of Patients	High Pretest Probability Score (6-8) # of patients	Total # of patients
ELISA Negative OD: <0.4 units	46 (83.6%)	9 (16.4%)	0	55
ELISA Positive OD: 0.40-1.00 units	5 (10.9%)	1 (11.1%)	0	6
SRA Positive	0 (0%)	0 (0%)	0	0

Abstract: Dilated Cardiomyopathy - Importance of Adherence to Medical Management

Haider Shamsulddin, MD, koroush khalighi, MD, Suman Thapamagar, MD
Easton Hospital: Department of Medicine

INTRODUCTION
Patients with dilated cardiomyopathy can be asymptomatic, but most present at some point in the spectrum of heart failure.[1]

CASE
A 47 year-old female with no significant past medical history presented with shortness of breath and leg swelling several weeks after severe upper respiratory tract infection, and was found to be in acute congestive heart failure (CHF). Echocardiography showed evidence for dilated cardiomyopathy and an estimated left ventricular ejection fraction (LVEF) of 5-10%. After ruling out secondary causes and giving her aggressive medical therapy with diuretics, beta blockers, and ACE inhibitor, she became asymptomatic with her LVEF gradually improving to 45% within six months and 55% within two years.

Due to a change in her medical insurance, and since she was asymptomatic, she stopped her medications. Three years later she presented with similar symptoms of shortness of breath, swelling, edema, and flu-like symptoms. Repeat cardiac evaluation, including echocardiography and coronary angiography, was significant for a reduction in her LVEF to 20-25%, resulting in recurrent CHF. She was restarted on her pervious medical regimen and within 3-months her LVEF improved to 40%.

DISCUSSION
The unclear etiology of dilated cardiomyopathy and the reversibility of LVEF with medical management are unique but not uncommon in this syndrome, since patients may present at some point in the spectrum of heart failure. A smaller number can deteriorate, requiring more invasive intervention including biventricular pacing with defibrillator, ventricular assist devices, or cardiac transplant.[1]

1. Mohan S, Parker M, Wehbi M, Douglass P. Idiopathic dilated cardiomyopathy: A common but mystifying cause of heart failure Cleveland clinic journal of medicine 2002; 69(6):481-487

Abstract: Educational Colonoscopy Video Enhances Bowel Prep Quality and Comprehension in an Inner City Population

Ajish Pillai, MD*, Asyia Ahmad, MD**, Noemi Baffy, MD*, Seth Kaufer, DO**, Radha Menon, MD**, David Oustecky, MD**
*Drexel University College of Medicine: Internal Medicine
**Drexel University College of Medicine: Department of Medicine, Division of Gastroenterologyand Hepatology

INTRODUCTION
Bowel preparation quality and patient comprehension are barriers to CRC screening, and video instruction may help. Few studies

have explored improvements involving interactive computer programs, pictures, or brochures except two, which focused on compliance and anxiety as endpoints.

METHODS
We conducted a single blind prospective study of inner city patients undergoing their first colonoscopy within 30 days. Experimental variable: 6 minute colonoscopy video (CV) or GERD video (GV) at initial visit. Upon colonoscopy, a 14 question quiz assessed comprehension, while blinded endoscopists graded preparations using the Ottawa scale.

RESULTS
104 subjects: 56 CV, 48 GV, 12 excluded. 48% male, 52% female; 90% had < HS education, 76% AA, and 67% used split preparation. These categories were identical between groups. CV had more excellent bowel preps (53% vs 31%; p<0.01), less inadequate bowel preps (9% vs 23%; p<0.01) and higher quiz scores (91% vs 79%; p<0.001). CV excelled in: "What is a polyp?" (80% vs 50%; p<0.001), "If a polyp is found, what happens?" (100% vs 77%; p=0.001), "What do patients feel after colonoscopy?" (79% vs 50%; p<0.002), "How often is colonoscopy repeated?" (100% vs 72%; p<0.001). In all patients, quiz scores positively correlated with preparation (odds ratio 2.31 (CI 1.35-3.94); P<0.001). Previous physician instruction spared results (85%).

CONCLUSION
Despite prior explanations of colonoscopy, videos improve comprehension and prep quality and may impact patient compliance. They should be considered as a necessary tool in gastroenterology practices.

REFERENCES
1. Lichtenstein GR, Cohen LB, Uribarri J. Review article: Bowel preparation for colonoscopy--the importance of adequate hydration. Aliment Pharmacol Ther. 2007 Sep 1;26(5):633-41
2. Piegal BMR,et al. Developmental and Validation of a Novel Patient Educational Booklet to Enchance Colonoscopy Preparation. Am J Gastroenterology 2011; 106:875-883

Abstract: Effect of HAART-Induced Immune Reconstitution Inflammatory Syndrome with Fatal Outcome in a Patient with Progressive Multifocal Leukoencephalopathy (PML)

Srividhya Lakshmanan, MD, Saman Kannangara, MD, Rashid Makhdomi, MD
Easton Hospital: Department of Medicine

INTRODUCTION
Immune reconstitution inflammatory syndrome (IRIS) is a collection of inflammatory disorders associated with the paradoxical worsening of pre-existing infectious processes following initiation of highly active antiretroviral therapy (HAART) in immunocompromised individuals.[1] PML-IRIS occurs in up to 18% of HIV-infected patients with PML.[2] Usually this condition is self-limited but when neurological structures are involved, the outcome may be fatal.

CASE
A 44-year-old male was admitted with complaints of incoherent speech and confusion during a court hearing. The physical examination was normal. A CT scan showed mild global cerebral atrophy. On day seven, he developed fever and an X-ray of the chest showed bilateral basal infiltrates and he was started on antibiotics. On day ten, the HIV rapid test was positive and the bronchoalveolar lavage was positive for pneumocystis carinii. An MRI of the brain showed features suggestive of PML (Fig 1). The patient was started on HAART. The patient's CD4 count improved but his neurological symptoms worsened. An MRI was repeated which showed worsening PML(Fig 2) and lumbar puncture was positive for JC virus. Later he was diagnosed with IRIS, started on corticosteroids, but he deteriorated and died.

DISCUSSION
Antiretroviral therapy in AIDS patients leads to dramatic improvement in CD4+ T-cell counts, however some patients experience a clinical deterioration resulting in IRIS. Early and prolonged treatment with corticosteroids may be useful in these patients but requires further investigation.[3] Unfortunately, in our patient ,he clinically deteriorated in spite of corticosteroid therapy.

REFERENCES
1. Riedel DJ, Pardo CA, McArthur J, Nath A. Therapy Insight: CNS manifestations of HIV-associated immune reconstitution inflammatory syndrome. Nat Clin Pract Neurol 2006;2:557–565
2.Cinque P, et al. The good and evil of HAART in HIV-related progressive multifocal leukoencephalopathy. J Neurovirol 2001;7:358–363
3. Tan K, Roda R, Ostrow L, McArthur J, Nath A PML-IRIS in patients with HIV infection: clinical manifestations and treatment with steroids. Neurology. 2009 Apr 28;72(17):1458-64

Figure 1. MRI showing fairly extensive patchy asymmetric white matter abnormality characteristic of PML

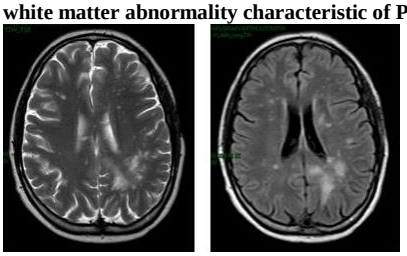
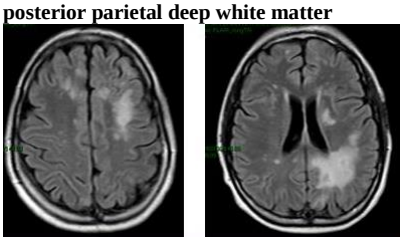


Figure 2. MRI showing PML with worsening in the left posterior parietal deep white matter



Abstract: Evaluation of Eating Disorders is necessary for Patients referred for Motility Testing

Nishita Patel, MD*, Asyia Ahmad, MD**, Karissa Russell**
*Drexel University College of Medicine: Internal Medicine
**Drexel University College of Medicine: Department of Medicine, Division of Gastroenterologyand Hepatology

INTRODUCTION
Eating disorders (EDs) are associated with disturbing gastrointestinal sensitivity and physiology and in turn cause functional gastrointestinal disorders (FGIDs). However, to our knowledge, no study has evaluated the prevalence of underlying EDs in patients with dysmotility symptoms. We performed a prospective study of patients referred for motility testing.

METHODS
Enrolled patients were screened for an underlying ED with a self-reporting questionnaire that contained ED risk factors and 3

validated diagnostic scales:1) Eating Attitude Test(EAT), 2) DSMIV Criteria for Anorexia Nervosa (AN) and Bulimia Nervosa (BN), 3)ED Diagnostic Scale (EDDS) for AN, BN, Binge ED. Significant findings were defined as Eat Score=19, 3 of 4 DSMIV criteria for AN, 5 of 6 DSMIV criteria for BN, 3 of 4 EDDS criteria for AN, 5 of 6 EDDS criteria for BED.

RESULTS
30 patients were enrolled. Mean age 51.2± 15.7, 26 (86%) were female and 16 (53%) were non-Hispanic white. Of the 30 patients, 5 (17%) patients were found to have a previously undiagnosed ED. Of these ED patients, all were female with a mean age of 50.6±14.1 and 3 (60%) non-Hispanic white. No difference noted in the demographics between the ED and non ED group. There was a significant increase in compensatory behaviors (diet pill, laxatives, diuretics, self-induced vomiting, laxative, fasting) amongst patients with EDs than those without EDs (80% vs 16%; p=.01).

CONCLUSION
We find that patients with dysmotility symptoms and an ED have an increased prevalence of ED risk factors such as exhibiting compensatory behaviors. These risk factors are important to ascertain in patients presenting with dysmotility symptoms as they do not respond to conventional FGID treatments. Resuming normal feeding and proper psychosocial counseling should become a key component of the treatment plan for these patients.

REFERENCES
1. Robinson PH, Clarke M, Barrett J. Determinants of Delayed Gastric Emptying in Anorexia Nervosa and Bulimia Nervosa. Gut 1988:29(4); 458-64
2. Hutson WR. Gastric Emptying in Patients with Bulimia Nervosa and Anorexia Nervosa. Am J of Gastroenterology. 1990: 85:41-46
3. Szmukler GI, Young GP, Lichtenstein M, and Andrews JT. A Serial Study of Gastric Emptying in Anorexia Nervosa and Bulimia. Internal Medicine Journal. 1990;20(3):220-25
4. Robinson PH, Stephenson JS. Dietary Restriction Delays Gastric Emptying in Rats. Appetite. 1990;14(3):193-201
5. Hadley S and Walsh B. Gastrointestinal Disturbances in Anorexia Nervosa and Bulimia Nervosa. Current Drug Targets-CNS & Neurological Disorders. 2003;2(1):1-9

Abstract: Incidental Diagnosis of Pulmonary Cryptococcosis in a Cardiac Transplant Recipient

Sukhraj Balhan, MD*, Raymond Benza, MD**, Nitin Bhanot, MD***, Tejpreet Lamba, MD****, Anil Singh, MD*
*Allegheny General Hospital: Department of Medicine, Division of Pulmonary Disease/Critical Care Medicine
**Allegheny General Hospital: Department of Medicine, Division of Cardiology
***Allegheny General Hospital: Department of Medicine, Division of Infectious Diseases
****Allegheny General Hospital: Department of Medicine

INTRODUCTION
Pulmonary Cryptococcosis in immunosuppressed individual may present without overt respiratory signs and symptoms. Diagnosis of invasive fungal infections should be entertained in the right scenario.

CASE
A 55-year-old man, non-smoker, s/p orthotropic heart transplant two years prior, was found to have multiple unilateral nodular infiltrates on a routine chest x ray. He denied any respiratory or cardiac symptoms. He did admit to some fatigue and progressive weight loss for the past couple of months. Physical examination was unrevealing. HRCT confirmed multiple pulmonary nodules. Concern for an indolent infection or malignancy such as post-transplant lymphoproliferative disorder was entertained. Navigational bronchoscopy with biopsies were performed. Cytopathology results were negative for malignancy and gram, silver, and AFB stains were negative. Cultures, however, grew Cryptococcus neoformans. His serum cryptococcal antigen was 1:128.

Lumbar puncture did not reveal evidence of meningeal involvement. Given significant pulmonary involvement in an immunocompromised host, an initial antimicrobial regimen of liposomal amphotericin with flucytosine, followed by fluconazole was initiated. The patient's fatigue gradually improved. His immunosuppression with medications was decreased and long term treatment with antifungal therapy is planned.

DISCUSSION
Cryptococcosis is most commonly identified in immunodeficient hosts such as patients with AIDS, malignancy, post- transplant recipients, and those on long term steroids.[1] Literature suggests that up to 40% of solid-organ transplant recipients with pulmonary cryptococcosis may not have symptoms and may be diagnosed incidentally through imaging[2], as was the case in our patient. Invasive testing proved valuable in making a timely diagnosis and initiating appropriate therapy.

REFERENCES
1. Casadevall A, Perfect J. Human crytococcosis. In: Casadevall A, Perfect J, eds. Cryptococcus neoformans. Washington, DC: ASM Press, 1998: 407–56
2. Singh N, et al. Pulmonary cryptococcosis in solid organ transplant recipients: clinical relevance of serum cryptococcal antigen. Clin Infect Dis. 2008;46(2):e12

Figure 1. H&E Stain

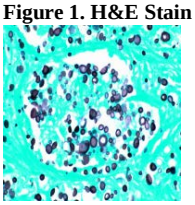
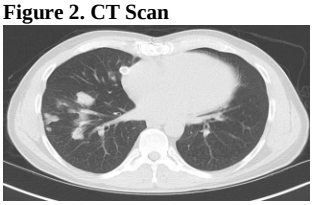


Figure 2. CT Scan



Abstract: Management of Implantable Cardiac Devices in patients with Breast Cancer requiring Radiation Therapy

Nader Omar, MD, Rahul Bhagat, MD, Koroush Khalighi, MD
Easton Hospital: Department of Medicine

CASE
A 75-year-old woman with a history of severe ischemic cardiomyopathy and ventricular tachycardia, who underwent an ICD implantation six years prior, was diagnosed with left breast carcinoma. She had an uneventful mastectomy, chemotherapy and adjuvant radiotherapy, requiring device removal with insertion in the contralateral chest wall.

A 56-year-old woman underwent uncomplicated left pectoral pacemaker insertion for sick sinus syndrome. One year later she developed a left breast mass requiring lumpectomy and left axillary lymph node resection, followed by chemotherapy and radiation therapy for suspected breast carcinoma. Unfortunately she developed a postoperative skin infection and pacemaker pocket infection which did not respond to antibiotic therapy. She underwent pacemaker and lead extraction with implantation of a new device on the contralateral side.

A 74-year-old woman with history of severe non-ischemia cardiomyopathy had refractory congestive heart failure despite maximized medical therapy. She underwent left pectoral biventricular ICD therapy with significant improvement of her heart failure class from NYHA class III-VI to II. Five years later she was diagnosed with left breast carcinoma, required left mastectomy, adjuvant radiotherapy, and radiation therapy. Her ICD pulse generator was overlying the radiation target site, requiring removal of the device with reinsertion of a new system in the contralateral chest.

DISCUSSION
The aforementioned cases outline the necessity for cardiac device explantation in patients requiring subsequent chest wall manipulation and localized radiation therapy. Inherent risks are local wound infection, arrythmogenicity, and direct cardiac or device circuit injuries.

Abstract: Neuraxial Blockade is a Viable Option for Arthrogryposis Multiplex Congenita during Pregnancy

Lisa Lee, MD, Mian Ahmad, MD, Kesavan Sadacharam, MD
Drexel University College of Medicine: Department of Anesthesiology

INTRODUCTION
Arthrogryposis multiplex congenita (AMC) is a rare condition, occurring 1:3000 live births.[1] Patients with AMC have multiple structural abnormalities including shortening of limbs, craniofacial malformation, joint contractures, and significant scoliosis of the spine. Though the etiology is unclear, animal studies have shown that intrauterine fetal akinesia will lead to contractures at birth.[2] The aforementioned problems make administration of anesthesia challenging in these patients. AMC is well-studied in children. However, it is uncommon for parturients to present with this disorder.

CASE
A 28 year-old female, G1P0, was admitted for induction of labor at term. Her medical history was significant for AMC, gestational diabetes mellitus, seizures, and GERD. Physical examination revealed severe scoliosis of thoracic and lumbar spine, and contractures in all extremities. After conferring with the patient, a combined spinal-epidural was performed. Good analgesia was achieved for both labor and subsequent c-section.

DISCUSSION
In patients with AMC, regional anesthesia is difficult due to vertebral abnormalities. The action of local anesthetics are unpredictable because of abnormalities in CSF production and reabsorption,[4] leading to the possibility of failed epidural due to unilateral block.[5] Because the risks of general anesthesia outweighed the benefits, we proceeded with regional anesthesia. For labor analgesia in this patient, we used a combined spinal-epidural technique. The spinal portion helped indirectly confirm the epidural space while using the loss-of-resistance technique. Continuous spinal infusion was not attempted because of the risk of postdural puncture headache. In our patient, epidural supplementation during cesarean delivery worked well, despite the severe structural abnormalities of the spine.

REFERENCES
1. Bamshad M, Van Heest AE, Pleasure D. Arthrogryposis: a review and update. J Bone Joint Surg Am 2009; 91 Suppl 4:40-6
2. Hall JG. Arthrogryposis multiplex congenita: etiology, genetics, classification, diagnostic approach, and general aspects. J Pediatr Orthop B. 1997; 6:159-66
3. Rozkowski A, Smyczek D, Birnbach DJ. Continuous spinal anesthesia for cesarean delivery in a patient with arthrogryposis multiplex congenita. Regional anesthesia 1996; 21(5):477-9
4. Spooner L. Caesarean section using a combined spinal epidural technique in a patient with arthrogryposis multiplex congenita. Int J Obstet Anesth 2000;9: 282-5
5. Quance DR. Anaesthetic management of an obstetrical patient with arthrogryposis multiplex congenita. Can J Anaesth;1988; 35:612-4.

Abstract: Nine Year Gap without an Implantable Cardioverter Defibrillator (ICD)

Nay Tun, MD, Koroush Khalighi, MD
Easton Hospital: Department of Medicine

CASE
A 77-year-old woman with a history of hypertension, severe obstructive sleep apnea, and non-ischemic cardiomyopathy presented with recurrent lightheadedness and palpitations, lasting a few seconds in the previous week. Nine years ago she was diagnosed with non-ischemic cardiomyopathy with ejection fraction (EF) of 30% after she presented with shortness of breath and palpitations. At that time, her left ventricular dysfunction persisted despite a maximized conventional medical therapy for over six months. Since she was no longer symptomatic, she declined the recommended prophylactic ICD therapy. Now, her repeat cardiac work-up, including echocardiography, coronary angiography, chest X-ray and laboratory data were unremarkable, except for the persistent severe cardiomyopathy and estimated EF of 30%. During her hospitalization, she had several runs of symptomatic sustained monomorphic ventricular tachycardia of up to 45 seconds. Therefore, she underwent an ICD system implantation and remained asymptomatic at 8 months.

DISCUSSION
In patients with non-ischemic cardiomyopathy, conventional medical therapy with beta-blockers, ACE inhibitors, and spironolactone have shown to halt or delay the progression of myocardial dysfunction. Although there are few cases where cardiomyopathy may persist, and progress to congestive heart failure or malignant cardiac arrhythmias, cardiac resynchronization with ICD therapy remains as the gold standard. However, in selected cases, careful observation with optimal medical therapy can safely postpone the early use of ICD therapy.

REFERENCES
1. Eshaghian S. The Role of Implantable Cardioverter Defibrillators in Ischemic and Non-Ischemic Cardiomyopathy and Effect on Mortality and Sudden Death. Einstein J. Biol. Med. 2007; 23:12-20
2. Tung R, Boyle NG, Shivkumar K. Catheter Ablation of Ventricular Tachycardia. Circulation. 2011;123:2284-2288
3. Katritsis DG, Camm A. J,Nonsustained Venticular Tachycardia: where do we stand? European Heart J. 2004; 25(13):1093-1099
4. Ladwig KH, Ronel J, Baumert J, Kolb C. Psychological comorbidity and quality of life in patients with an implantable cardioverter/defibrillator. Herzschrittmachertherapie & Elektrophysiologie. 2010:21(2):129
5. Buxton AE, et al. Nonsustained ventricular tachycardia. Cardiol. Clin. 2000;18:327–336

Abstract: Quality Improvement Project: Identifying Barriers that Hinder Patient Office Visit Compliance for Desired Long Acting Reversible Contraception at an Urban Womens Health Center

Sherron Colgram, MD
Drexel University College of Medicine: Department of Obstetrics and Gynecology

INTRODUCTION
Long acting reversible method of contraception (LARC), which includes intrauterine and subdermal implants, are underutilized despite their proven safety, use and cost effectiveness. It has been shown that once financial and knowledge barriers are removed women who receive contraception counseling before use demonstrate higher rates of method satisfaction, continuation rates, and acceptance. There are no current studies that identify differences between women who decide to use LARC and those who actually obtain this form of birth control.

OBJECTIVE
To identify any predictors of non-return visits for desired LARC insertion. Our hypothesis is that the level of comprehensive counseling regarding LARC may differ from women who present for LARC insertion.

METHODS
Retrospective electronic medical record chart review was undertaken for all patients from an ambulatory academic teaching clinic from January to November 2012 who were scheduled for LARC insertion but did not present to scheduled visit. Demographics included age, race, marital status and parity. Identification of barriers to LARC insertion including but not limited to level of LARC counseling, current contraception at time of visit, recent delivery, and recent STD screening were identified.

RESULTS
The majority of women were found to be within their postpartum period, received contraceptive counseling by ancillary staff, but did not receive counseling during antepartum care by their obstetrical provider.

DISCUSSION
There is need for improvement in contraceptive counseling before LARC insertion visits. Counseling should occur throughout the antepartum period and LARC should preferably be placed at postpartum visits to prevent future unintended pregnancies.

Abstract: Sonographic Assessment of Hyaline Cartilage Thickness in the Knee at different Views Distinct from the Standard View with the Knee fully Flexed

Jatin Patel, MD
Drexel University College of Medicine: Department of Medicine, Division of Rheumatology

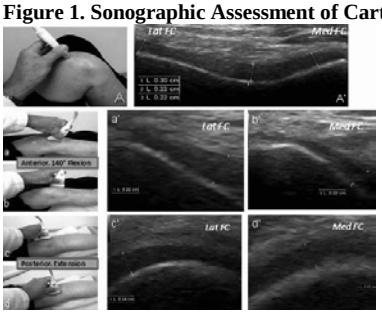
INTRODUCTION
Studies of cadavers or pathological specimens from postsurgical knee arthroplasty patients have shown that the measurement of femoral articular cartilage thickness (FACT) is accurate and reproducible by ultrasound (US). The aim of this study was to find an effective alternative method to measure the FACT by US, using a minimally flexed or an extended knee.

METHODS
Twenty right knees from volunteers with a mean age of 32.6 (range 22-52) years were evaluated by US. The FACT was measured in transverse view with the knee fully flexed. Two views of the medial and lateral femoral condyles were obtained at the anterior aspect with the knee flexed at 140° and the posterior aspect with knee extended.

RESULTS
The correlations of other views with the fully flexed view were good, 0.85 or higher for both raters with the exception of the lateral FC in the posterior view of the extended knee. The mean of thickest FACT was 20 mm for the lateral FC and 20.4 mm for the medial FC at the standard view. In a longitudinal view with the knee flexed at 140° it was 20.3 mm for the lateral FC and 20.3 for the medial FC. In the posterior view with the extended knee the mean of the FACT for lateral and medial FC was 19.1 mm and 20.7 mm respectively.

CONCLUSION
US measurement of FACT using the longitudinal view anterior aspect with the knee at 140° of flexion & at posterior aspect with extended knee at the medial FC demonstrated good comparability to standard fully flexed knee.

Figure 1. Sonographic Assessment of Cartilage



Abstract: Takotsubo Cardiomyopathy (Broken Heart Syndrome): an unusual association with Depression

Nonihal Singh, MD, Elmi Farhad, MD, Suman Thapamagar, MD
Easton Hospital: Department of Medicine

INTRODUCTION
Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy, apical ballooning syndrome, and broken heart syndrome, is an uncommon, recently recognized cardiac syndrome.[1] It is associated with physical or emotional stress and is usually seen in elderly postmenopausal females.[2]

CASE
A 79-year-old-caucasian-widower nursing home (NH) resident presented to the emergency room (ER) for evaluation of chest pain. On arrival her EKG showed paced rhythm and her troponins were mildly elevated 1.1ng/mL. Transthoracic echocardiography showed hypokinesis/akinesis of the apex, lower two-thirds of the apical portion of the septum, and apical portion of the lateral wall with apical ballooning (Fig 1), a finding which was not seen in the prior study of 2008. Ejection fraction was reduced to 40%-45%. Subsequently cardiac catheterization was performed which showed no major blockages (Fig 2). She was treated conservatively for heart failure. Three months later a follow up echocardiogram showed normalization of the ejection fraction and resolution of the apical ballooning.

DISCUSSION
The patient had a long history of depression for which she was on citalopram, but had been taken off citalopram due to tremors a month prior to presentation. She was also admitted to NH one year ago by her daughter. TCM appears to be associated with untreated depression. There is a possibility that this catastrophe could have been prevented if she was not taken off the citalopram or if it was replaced with another antidepressant. Physicians should be well

aware of this association especially while dealing with elderly females on antidepressants.

Figure 1. Left ventriculogram showing apical ballooning

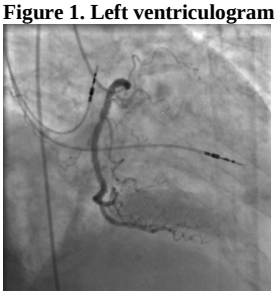
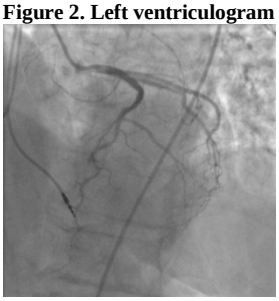


Figure 2. Left ventriculogram showing no major blockages



REFERENCES

1. Akashi Y, Goldstein D, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. Circulation. 2008;118:2754–2759

2. Wittstein I, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005;352:539–548

Case Report: Allopurinol-Induced Hepatotoxicity- A Rare And Potentially Lethal Side Effect

Bhavinkumar Patel, MD
Easton Hospital: Department of Medicine

CASE REPORT

A sixty-year male was admitted for CHF exacerbation. Initial laboratory tests revealed total bilirubin 1.4 mg/dl, alkaline phosphatase (ALP) 193 IU/liter, aspartate aminotransferase (AST) 135 IU/liter, alanine aminotransferase (ALT) 204 IU/liter and serum creatinine of 1.53 mg/dL. Elevated liver function tests were presumed to be secondary to congestive hepatopathy and atorvastatin which was discontinued. The patient's uric acid levels were high at 11.7 and he was started on allopurinol for prophylaxis against gout. Four days later, the patient started complaining of fatigue and severe anorexia. Physical examination was remarkable for mild icterus and laboratory tests revealed total bilirubin 5.2 mg/dl, ALP 268 IU/liter, AST > 4000 IU/liter, ALT 2605 IU/liter and INR 2.0. Hepatitis panel and liver sonogram were normal. Patient deteriorated rapidly, was diagnosed with acute fulminant hepatic failure and was transferred to the intensive care unit. All of his medications were stopped except for insulin. The patient was started on supportive therapy with intravenous n-acetylcystine and monitored closely. After withholding the medications, his liver function tests gradually improved, jaundice resolved, and he was discharged home. Allopurinol was identified as the responsible etiology for the acute worsening of liver functions. Except allopurinol and atorvastatin, all other medications were re-instituted without any complications.

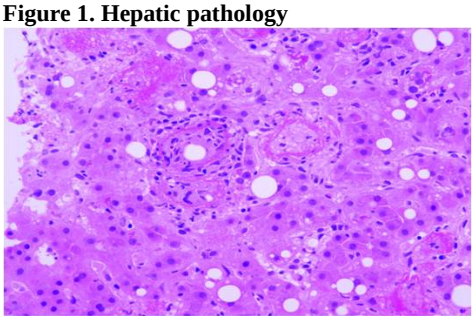
DISCUSSION

Physicians prescribing allopurinol should be aware of the manifestations of the hypersensitivity syndrome and allopurinol-induced hepatic and renal toxicity. Caution is recommended, especially in patients with underlying renal dysfunction and diabetic nephropathy. Concomitant use of amoxicillin, non-steroidal anti-inflammatory drugs, and ACE inhibitors should be avoided.

REFERENCES

1. Umpierrez A, et al. Successful desensitization of a fixed drug eruption caused by allopurinol. J Allergy Clin. Immunaol 1998; 101(2):286-287
2. BarrancoVP. Clinically significant drug interactions in dermatology. J An Acad Dermatol 1998;38:599-612
3. Gittin N. Drug induced hepatic vascular abnormalities. Clin in Liver Dis 1998;2:591-606
4. Pennell DJ, Numan TO, O' Doherty MJ. Fatal Steven Johnson syndrome in a patient on captopril and allopurinol. Lancet 1984;2:463
5. Boston Collaborative Drug surveillance Program. Excess of ampicillin rashes associated with allopurinol or hyperuricemia. NEJM 1972;286:505-507

Figure 1. Hepatic pathology



Case Report: Amyand Hernia

Wesley Vosburg, MD, Phoebe Abraham, MD, H. Kenneth Williams, MD
Allegheny General Hospital: Department of Surgery - Division of General Surgery

CASE REPORT

In 1753 a case report was published by Claudius Amyand that described an eleven year old boy who was found to have an inguinal hernia that contained his appendix.[1] Later, the term Amyand hernia was given to this condition. Today, inguinal hernias are found to contain the appendix about 1% of the time and acute appendicitis within an Amyand hernia occurs in approximately 0.1% of inguinal hernias.[2] We present a case of an 87 year-old male found to have acute appendicitis within an Amyand hernia. Diagnosis was supported with abdominal CT (Figs 1,2). The patient underwent appendectomy via an inguinal incision and hernia repair using the Bassini technique.

DISCUSSION

A classification scheme that divides Amyand hernias into one of four groups may be determined based on pre-op CT results.[3] Type one Amyand hernias demonstrate a normal appendix within an inguinal hernia and may be managed on an elective basis. Type two shows characteristics of acute appendicitis within the inguinal hernia but no signs of intra-abdominal sepsis. These hernias should be repaired more urgently and include appendectomy. Type three involves

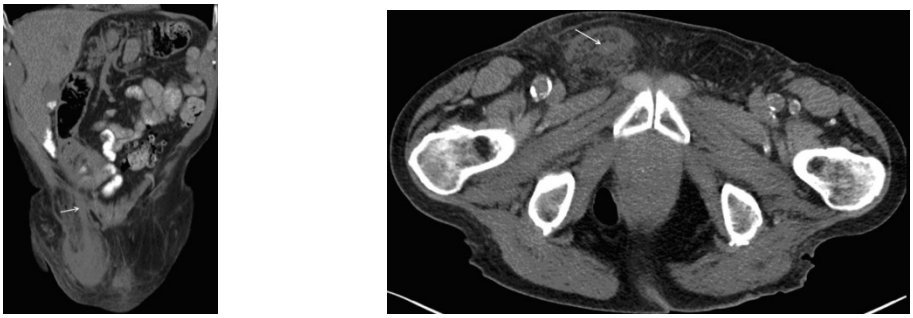
acute appendicitis within the inguinal hernia and abdominal wall or peritoneal sepsis. Laparotomy to address the other intra-abdominal process is advocated. Type four involves acute appendicitis within the inguinal hernia and another finding of intra-abdominal pathology that is either related or unrelated, such as diverticulitis.[3,4] Type four hernia management is similar to the other three and individualized to each patient.[4]

REFERENCES

1. Amyand C. Of an Inguinal Rupture, with a Pin in the Appendix Coeci, Incrusted with Stone; And Some Observations on Wounds in the Guts. Philosophical Transactions of the Royal Society of London 1753; 39:329-42
2. Llullaku, et al. A Pin in Appendix within Amyand`s Hernia in a Six-year-old Boy: Case Report and Review of Literature. World Journal of Emergency Surgery 2010; 5:1-3
3. Constantine S. Computed Tomography Appearances of Amyand Hernia. Journal of Computer Assisted Tomography 2009; 33:359-62
4. Losanoff J, Basson M. Amyand Hernia: What Lies Beneath - A Proposed Classification Scheme to Determine Management. The American Surgeon 2007; 73:1288-290

Figure 1. CT images of appendix (arrow) and acute inflammatory process contained within a right sided inguinal hernia

A. Coronal **B. Transverse**



Case Report: An Unusual Case of "Code Blue"

Yanfang Guan, MD, Koroush Khalighi, MD, Suman Thapamagar, MD
Easton Hospital: Department of Medicine

INTRODUCTION

Whereas coronary artery spasm (CAS) is not an uncommon event and spontaneous spams might occur in young patients as well as during percutaneous coronary intervention in elderly patients, extensive multivessel spasm rarely occurs.[1,2] Recognizing and rescuing massive multivessel CAS is paramount because it may cause devastating hemodynamic embarrassment and even death.[3,4] The aim of this study was to improve diagnosis and treatment of multivessel CAS.

CASE REPORT

When a 66-year-old female with recurrent chest pain, normal cardiac enzymes, and an unremarkable EKG was catheterized into the left coronary artery, she suddenly became bradycardic, lost pulse, and developed electromechanical cardiac dissociation, apparently due to CAS despite the pretreatment with sublingual nitroglycerin. Angiography showed total occlusion of the entire left descending and left circumflex artery (Fig 1). Standard cardiopulmonary resuscitation protocol was immediately initiated. Upon three times of intra-coronary 400 mcg nitroglycerin injection the patient regained spontaneous circulation, heart rate and blood pressure recovered, and left coronary artery angiography revealed complete reversal of the spasm (Fig 2). No potentially lethal ischemia-induced ventricular arrhythmias were noted. The patient's vital signs then remained stable, and coronary angiography was continued as planned.

DISCUSSION

We conclude that CAS, especially multivessel spasm, prompts for accurate diagnosis, and the immediate treatment with intracoronary artery injection of high doses of nitroglycerin may relieve the spasm and restore spontaneous circulation. This measure may be warranted in high risk patients both prior and during catheterization.

REFERENCES

1. Ahooja V, Thatai D. Multivessel coronary vasospasm mimicking triple-vessel obstructive coronary artery disease. J Invasive Cardiol. 2007;19:E178-81
2. Moukarbel GV, Dakik HA. Diffuse coronary artery spasm induced by guidewire insertion. J Inv Cardiol. 2003;15:353-4
3. Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (coronary spastic angina JCS 2008): digest version. Circ J. 2010;74:1745-62
4. Park YM, et al. Diffuse multi-vessel coronary artery spasm: Incidence and clinical prognosis. Int J Cardiol. Epub 2012 Jan 24

Figure1. Occlusion of LAD and Circumflex

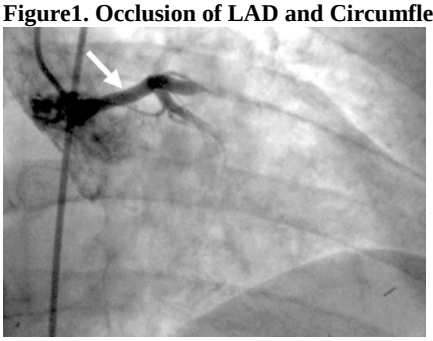
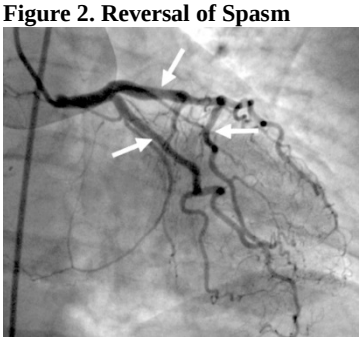


Figure 2. Reversal of Spasm



Case Report: Beware, 'I' can appear anywhere!

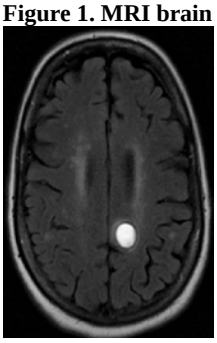
Nagakrishnal Nachimuthu, MD, Hesham Elmergawy, MD, Jeffrey Felzenberg, MD, Rosemary Fiore, MD, Sara Wallach, MD
Monmouth Medical Center: Department of Medicine

CASE REPORT

We present the case of a patient with occult primary melanoma who presented with multiple metastatic lesions to brain and bone.

A 64 year-old lady with history of chronic back pain status-post lumbar laminectomy done eighteen years ago presented with worsening back pain for 2 weeks. She denied fever, chills, bowel or urinary incontinence, tingling or weakness. She was experiencing worsening back pain with radiation to the left knee. Physical examination and labs were normal. MRI lumbar and thoracic spine showed enhancing lesions on T6, T10, T11, L2-L3, right sacral ala, and pathological fracture T11, suspicious for metastatic disease. MRI brain revealed hyperintense lesions in the left cerebral and cerebellar hemisphere (Fig 1). The patient underwent kyphoplasty of T11 compression fracture and bone biopsies were obtained. The final biopsy reported a high grade metastatic malignant melanoma. A thorough examination of her skin and fundus revealed no primary lesions suggestive of melanoma.

Figure 1. MRI brain with contrast



DISCUSSION

Melanoma is an aggressive lesion with a 5-year survival rate of 5% in patients with distant metastasis.[1,2,3] Very few cases have been reported of melanoma metastasizing to the bone (especially spine). In such cases prognosis is very poor.[2] Even though rare, melanoma should be considered part of the differential diagnosis in evaluating bony lesions with no overt primary cancer.

REFERENCES

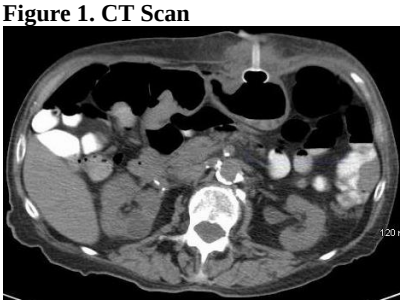
1. Donaldson WF Jr, et al. Symptomatic metastatic melanoma to the spine. J Spinal Disord. 1993; 6(4):360-363
2. Roses DF, et al. Survival with regional and distant metastases from cutaneous malignant melanoma. Surg Gynecol Obstet. 1991;172(4):262-268
3. Rigel DS, Friedman RJ, Kopf AW. The incidence of malignant melanoma in the United States: issues as we approach the 21st century. J Am Acad Dermatol. 1996;34:839–847

Case Report: Buried Bumper Syndrome: a Rare but Preventable Complication of Percutaneous Endoscopic Gastrostomy Tube Placement

Hadie Razjouyan, MD
Monmouth Medical Center: Department of Medicine

CASE REPORT
An 81 year-old male with a history of laryngeal carcinoma status post laryngectomy and chemoradiation presented with abdominal pain after percutaneous endoscopic gastrostomy (PEG) tube placement. He received the PEG tube because of progressive dysphagia. The pain was at the PEG site, 7-8/10 in severity, crampy, constant without radiation, not associated with nausea, vomiting, diarrhea or constipation. After the procedure, he had been having pain and noticed the PEG site was getting edematous and red. He was having tube feeding and denied any constitutional symptoms. He was admitted with possible infection of PEG tube insertion site and a CT scan of the abdomen and pelvis without IV contrast was performed for further investigation (Fig 1). Then he underwent upper GI endoscopy 8 days after placement which showed the dislocated bumper, a large defect in the gastric wall with necrotic-appearing tissue and pus consistent with buried bumper.

Figure 1. CT Scan



DISCUSSION
The buried bumper syndrome is a rare complication of PEG tube placement with an incidence of 0.3-2.4%.[1] The main reason seems to be squeezing of the soft tissue specifically stomach wall between the internal and external bumper.[1] Obesity, chronic severe cough, manipulation, and frequent inadvertent tube traction by caregivers were reported as risk factors. Avoiding excessive traction force while routing the tube through the abdominal wall, allowing an additional 1.5-2.0 cm between the external bumper and skin, visualizing the internal bumper immediately following the PEG placement, and periodic measurement of the tube external portion length are measures which can help to avoid such complications.[2]

REFERENCES
1. Wong R and Ladabaum U. An uncommon complication of percutaneous endoscopic gastrostomy tubes. Clin Gastroenterol Hepatol, 2013;11(2):A25
2. Pop GH. Buried Bumper Syndrome: Can We Prevent It? Practical Gastroenterology. 2010:8-13

Case Report: Calcification Everywhere: a Rare Case of Pseudopseudohypoparathyroidism (PPHP)

Nonihal Singh, MD, Mahesh Krishnamurthy, MD, Dhaval Sureja, MD, Pradip Toshniwal, MD
Easton Hospital: Department of Medicine

CASE REPORT
A 55-year-old alcoholic, hypertensive male presented with bilateral lower extremity numbness and frequent falls for two months. Physical examination revealed short stature, hand muscle atrophy, severe weakness, spasticity, brisk reflexes, bilateral knee clonus with extensor plantar reflexes and left homonymous hemianopia. Subsequently, he developed altered mental status, hypoxia and was transferred to the ICU. CT brain showed extensive scalp and basal ganglia calcification (Fig 1). MRI confirmed acute right occipital lobe infarct with hemorrhagic conversion and chronic spinal cord compression with myelomalacia at C6-T1 and T9-T10 secondary to degenerative disc disease. The hand x-rays confirmed short 4th metacarpal bone and extensive soft tissue calcification (Fig.2). The patient's serum calcium, phosphorus, and PTH levels were normal. Subsequently, he underwent laminectomy and foraminotomy.

DISCUSSION
The findings, including extensive calcification and normal laboratory data, suggest PPHP. The target organ unresponsiveness to parathyroid hormone (PTH) is called pseudohypoparathyroidism (PHP). When it is associated with short stature, short fourth metacarpal bone, subcutaneous calcification, lumbar stenosis and developmental delay, it is called Albright hereditary osteodystrophy (AHO).[1] When the mutated gene allele is paternally transmitted, phenotypic features are present without renal resistance to PTH.[2] This extremely rare entity, with normal biochemical parameters, is called PPHP.[1,2,3,4]

Although basal ganglia, dentate nucleus, and subcutaneous calcifications are described in PPHP, to the best of our knowledge we report the first case of extensive scalp calcification. The fact that our patient was undiagnosed until the age of 55 is likely due to physicians' unawareness of this rare condition.

REFERENCES
1. Kottler ML, Linglart A, Carol JC. Albright hereditary osteodystrophy. Orphanet encyclopedia. January 2004. Available at: http://www.orpha.net/data/patho/GB/uk-AHO.pdf. Accessed June 23, 2012

2. Mantovani G. Pseudohypoparathyroidism: diagnosis and treatment.J Clin Endocrinol Metab. 2011;96(10):3020-30
3. Pseudopseudohypoparathyroidism. Online Mendelian Inheritance of Man. November 2010 Available at: http://www.ncbi.nlm.nih.gov/omim/612463. Accessed June 23, 2012
4. Pseudopseudohypoparathyroidism. Office of rare diseases research. Available at: http://rarediseases.info.nih.gov/GARD/Condition/7860/Pseudopseudohypoparathyroidism.aspx. Accessed June 23, 2012

Figure 1. CT scan showing extensive scalp calcification.

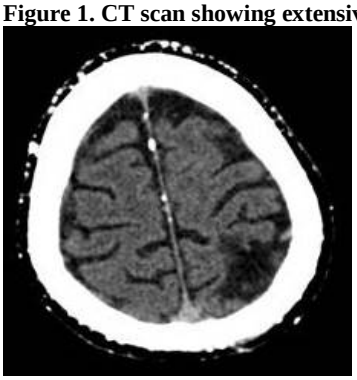
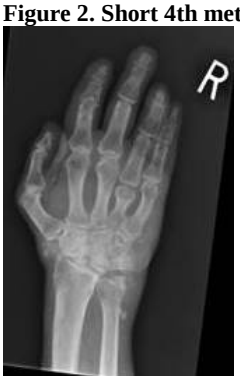


Figure 2. Short 4th metacarpal, extensive soft tissue calcification.



Case Report: Caseating Granulomas in the Skin: TB or not TB

Julieta Gilson, MD, Mahesh Krishnamurthy, MD, Jolanta Zelaznika, MD
Easton Hospital: Department of Medicine

CASE REPORT

A 63 year-old male presented with a 2 month history of diffuse myalgias, arthralgia, fatigue and night sweats. He developed swelling, tenderness and erythema of his right ankle, which eventually ulcerated. Subsequently, he developed multiple nodules involving his legs, arms, and trunk. Two of the nodules evolved into painful ulcers. There was no other rash. He reported no dry mouth or eyes, oral or genital ulcers, cardiac, respiratory or genitourinary symptoms. Laboratory evaluation showed ESR 50mm/h, leukocytosis, mild eosinophilia, anemia, increased platelet count. CRP 3.3. CPK, P-ANCA, C-ANCA, ANA, ACE levels, and RF were negative. Bone marrow biopsy revealed mild hypercellularity. Extensive bacteriological studies, including TB and fungal infections, yielded negative results. Skin biopsy of the nodules demonstrated chronic inflammation with caseating granulomas. There was no evidence of vasculitis. Since extensive investigation failed to reveal an infectious etiology, a provisional diagnosis of sarcoidosis was made. The patient was started on steroids, resulting in dramatic improvement of the skin lesions (Figs 1,2). Adalimumab was introduced as a steroid-sparing medication. Subsequently all lesions, including ulcerations, resolved.

DISCUSSION

Sarcoidosis is a systemic inflammatory disorder known as one of the “great imitators” clinically and histopahtologically, with skin involvement in about 20-35% of the patients.[4] This is an unusual presentation of granulomatous dermatitis with caseating granulomas. After infectious causes were ruled out, sarcoidosis was the most likely diagnosis. To the best of our knowledge, this is the first reported case of non-infectious granulomatous dermatitis with these histopathological findings.

REFERENCES

1. Mukhopadhyay S, et al. Causes of pulmonary granulomas: a retrospective study of 500 cases from seven countries. J Clin Pathol. 2012 Jan;65(1):51-7
2. Fernandez-Faith E, McDonnell J. Cutaneous sarcoidosis: differential diagnosis. Clin Dermatol. 2007 May-Jun;25(3):276-87
3. Gibson LE, Winkelmann RK. The diagnosis and differential diagnosis of cutaneous sarcoidosis. Clin Dermatol 1986;4:62-74
4. Rabinowitz LO, Zaim MT. A clinicopathologic approach to granulomatous dermatoses. J Am Acad Dermatol 1996;35:588-600
5.Tchernev G. Cutaneous sarcoidosis: the "great imitator": etiopathogenesis,morphology, differential diagnosis, and clinical management.Am J Clin Dermatol.2006;7(6):375-82

Figure 1. Skin ulcer

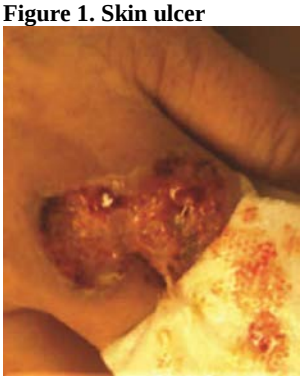
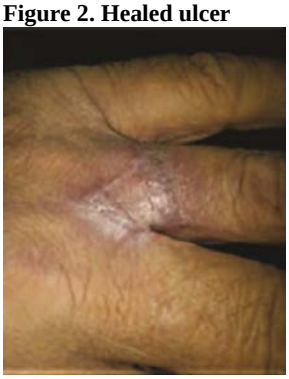


Figure 2. Healed ulcer



Case Report: Cholestasis in Pregnancy status post Cholecystectomy

Monica Suliman, MD, Kimberly Dong, Ross Myers
Monmouth Medical Center: Department of Obstetrics and Gynecology

CASE REPORT

32 year-old G3P2 Hispanic female at 35 weeks gestational age presents with generalized pruritus worse at night and on the palms and soles. Patient was found to have elevated bile acids (total bile acids>17umol/L). Further, the patient had a history of cholecystectomy in 2011 and denied history of cholestasis in prior pregnancies. Patient was diagnosed with intrahepatic cholestasis of pregnancy, started on ursodeoxycholic acid and frequent fetal monitoring.

DISCUSSION

Intrahepatic cholestasis in pregnancy (ICP) is defined as pruritus in pregnancy with elevated bile acids which resolves after delivery.[1] This case presents a paradoxical picture of a Hispanic patient with a history of cholecystecomy presenting with classical signs and symptoms of ICP. There have been few studies with patients with ICP after cholecystectomy. Interestingly, it has been shown that women with prior gallbladder disease including cholecystectomies, surprisingly, have an increased risk for ICP.[2] The exact etiology in pregnancy is unknown; however, it also has been associated with environmental and genetic factors including an increased risk in South American populations and a decreased incidence of 1/500-1000 pregnancies in North America.[3] In fact, ICP has been seen with a threshold for total bile acids in the Latina population as low as 8.5umol/L instead of the laboratory standard of 10umol/L.[4] ICP is important to identify because of its high risk of complications to the fetus including sudden death. Therefore, close surveillance is critical, especially in Latina populations with other risk factors.

REFERENCES

1. Joshi D, James A, Quaglia A, Westbrook RH, Heneghan MA. Liver disease in pregnancy. Lancet. 2010;375:594-605
2. Glantz A, Marschall HU, Mattsson LA. Intrahepatic cholestasis of pregnancy: relationships between bile acids levels and fetal complication rates. Hepatology. 2004;467-474
3. Cunningham FG, et al. Chapter 50. Hepatic, Gallbladder, and Pancreatic Disorders. In: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY, eds. Williams Obstetrics. 23rd ed. New York: McGraw-Hill;2010
4. Lee RH, et al. Bile acid concentration reference ranges in a pregnant latina population. Am J Perinatology. 2012

Case Report: Just Another Case of Gastroenteritis?

Chintan Gandhi, MD
Saint Peter's University Hospital: Department of Pediatrics

CASE REPORT

A 4 month-old previously healthy female presented with complaints of 5-6 episodes of non-bilious, non-bloody vomiting with 1 episode of brownish diarrhea in ER. The child had decreased activity and PO intake. Her older sister was having the same complaints. No fever or decreased urine output. Vitals: temperature 99.3 F, pulse 157/min, BP 90/60 mm Hg and respirations 36/min. The child was fussy but consolable. Skin turgor was poor. Mucous membranes were dry. Capillary refill was < 2 seconds. Abdomen was soft, non-tender with no masses. The rest of the examination, CBC and BMP were normal. She failed PO challenge in ER and was admitted for moderate dehydration with a working diagnosis of acute gastroenteritis. She then had 2 episodes of bloody diarrhea and was crying inconsolably. Stat ultrasound of the abdomen showed ileocolic intussception which was reduced by air enema immediately (Figs 1,2).

DISCUSSION

Intussusception can present with the same symptoms as acute gastroenteritis, but a change in the pattern of illness,

in the character of pain, or in the nature of vomiting should alert the physician. The 'classic' triad of intussusception (abdominal pain, bloody/red currant jelly stool and abdominal mass) were reported in only 29 to 33% of presentations[1] so high degree of suspicion is required for diagnosis. Abdominal ultrasound, the gold standard for diagnosis, shows classic doughnut or target appearance in transverse images. Delays still occur in the diagnosis of intussusception because of the non-specific nature of symptoms and signs in some patients. The success rate of radiologic hydrostatic reduction under ultrasonic guidance is ~50% if symptoms are present >48 hours and 70–90% if reduction is done in the first 48 hours.[3] Undiagnosed intussusceptions can cause tissue ischemia, intestinal perforation and peritonitis. If left untreated, it can be fatal. It is an abdominal emergency and it is of utmost important for the physician to diagnose this condition as soon as possible.

REFERENCES

1. Blanch AJ, Perel SB, Acworth JP. Pediatric intussusception: epidemiology and outcome. Emerg Med Australas. 2007;19:45–50
2. Bines J, Ivanoff B. Vaccines and Biologicals. Acute intussusception in infants and children: a global perspective. WHO/V&B/02.19. Geneva:World Health Organization;2002
3. Kleigman et al. Nelson's Pediatrics, 19th Edition. Philadelphia: Saunders Elsevier. 2011;1760

Figure 1. US classic doughnut or target appearance

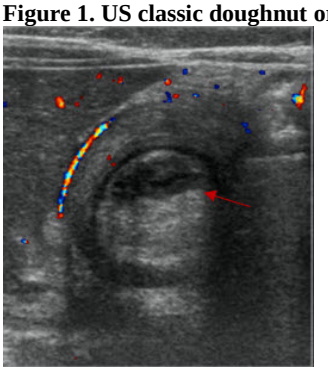
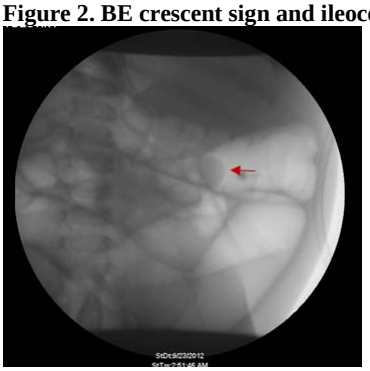


Figure 2. BE crescent sign and ileocolic intussusception



Case Report: Management of Olecranon Bursitis using Elbow Device in a Taxi-driver with Maladaptive Posture while Driving

Angel Checa, MD*, Reginato Anthony, MD**
*Drexel University College of Medicine: Department of Medicine, Division of Rheumatology
**Rhode Island Hospital and Alpert School of Medicine at Brown University at Providence: Department of Medicine

CASE REPORT

A healthy 60 year-old male taxi-driver for the last 7 years, drives supporting his elbows over the steering wheel. He presented complaining of pain and swelling at the left elbow. On clinical exam, a fluctuant, moderately tender swelling at the olecranon was observed. Similar episode in the right elbow was reported two years before. The possibility of gout, chondrocalcinosis, penetrating injury, or infection were ruled out. A large effusion characterized by anechoic area, surrounding multiple hypoechoic nodules consistent with hypertrophy of the synovium was noted (Fig 1).

DISCUSSION

Traumatic olecranon bursitis has been related with different occupational risk factors, which all have a common pathogenic mechanism: repetitive trauma.[1] This is a characteristic recurrent traumatic olecranon bursitis. It was treated by aspiration and a corticosteroid intrabursal injection and NSAIDs for two weeks. The patient was additionally advised to discontinue the positioning of the elbow on the steering wheel. In order to prevent repositioning of his elbows on the steering wheel, a band with a plastic bar was developed to wear on his forearms (Fig 2). A padding bar (one inch height by one inch) adjustable to the forearm 5 cm front olecranon, was used to decondition the inappropriate habit. He returned asymptomatic six months later. Additionally, the maladapted position at the workplace was corrected. We have demonstrated that the temporary use of an inexpensive device prevented the maladaptive posture at the work place and removed the pathogenic mechanism of olecranon bursitis in this taxi-driver.

REFERENCES

1. Matsumoto T, et al. Massive nonspecific olecranon bursitis with multiple rice bodies. J Shoulder Elbow Surg 2004;13(6):680- 683

Figure 1. US with anechoic area surrounding hypoechoic nodules

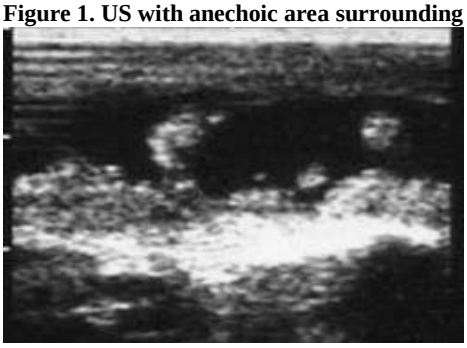
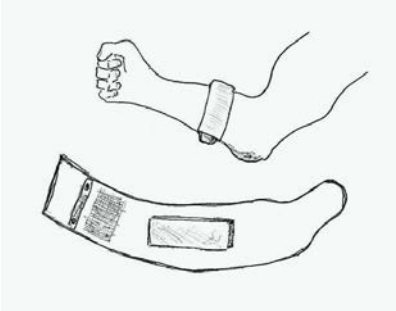


Figure 2. Device for forearm



Case Report: Mycobacterium riyadhense causing Pulmonary Infection in Human

Jason Henderson, DO*, Sandy Genes**, Peter Kaplan, MD*, Zachary Young, MD*
*Allegheny General Hospital: Department of Medicine, Division of Pulmonary and Critical Care Medicine
**Alleghney County Health Department: Department of Medicine

CASE REPORT

A 29 year-old man originally from Saudi Arabia presented with complaints of weight loss, productive cough, night sweats, and fever. He was initially treated with Levaquin for pneumonia. Although he initially improved, his symptoms progressed and a few months later he represented. A chest X-ray (Fig 1) at that time showed bilateral upper lobe infiltrates and he was again treated with Levaquin. A CT chest (Fig 2) was obtained which revealed a cavitary lesion in the right upper lobe, bronchiectasis, and bilateral upper lobe "tree-in-bud" opacities. His sputum was positive for Acid-fast bacilli and a PPD was positive with a reaction of 25mm. A qualitative Quantiferon-TB gold was also positive. Mycobacterium cultures were negative for Mycobacterium tuberculosis by DNA probe and Mycobacterium avium. Pending culture data, he was started on emperic tuberculosis treatment. Culture data subsequently identified Mycobacterium riyadhense based on DNA sequencing. Drug senstivities showed intermetidate sensitivy to isoniazid and resistance to levofloxacin, kanamycin, cycloserine, and doxycycline. The patient's clinical symptoms improved and his radiographic abnormalities also improved during the course of treatment. His sputum cultures turned negative after four months of treatment. Treatment with isoniazid 300 mg daily, rifampin 600 mg daily, pyrazinamide 1000 mg daily, and ethambutol 900 mg daily was continued for one year following the conversion of his sputum cultures.

DISCUSSION

To our knowledge, this is the first reported case of M. riyadhense causing pulmonary disease in the United States, and only the fourth case of M. riyadhense described in the literature

REFERENCES

1. Van Ingen J, et al. Mycobacterium riyadhense sp. nov.; a non-tuberculous species identified as Mycobacterium tuberculosis by a commercial line-probe assay. Int J Syst Evol Microbiol 2009;59:1049–1053
2. Godreuil S, et al. Mycobacterium riyadhense pulmonary infection, France and Bahrain [letter]. Emerg Infect Dis. 2012 Jan. http://dx.doi.org/10.3201/eid1801.110751
3. Andersen P, Munk ME, Pollock JM, Doherty TM. Specific immune-based diagnosis of tuberculosis Lancet. 2000;356:1099–1104

Fig 1: Chest X-ray showing cavitary lesion

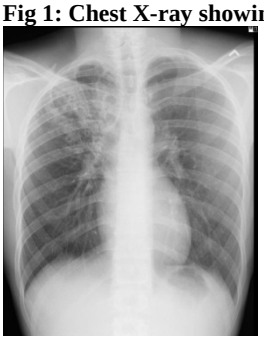
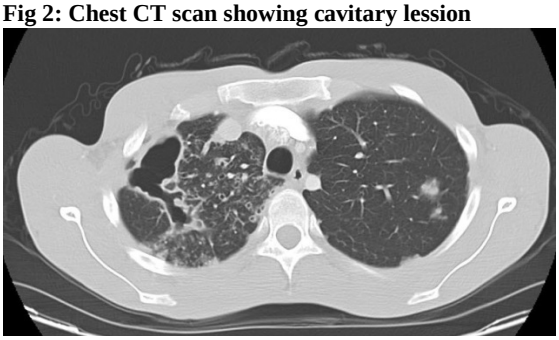


Fig 2: Chest CT scan showing cavitary lession



Case Report: Osseous Metaplasia within a Renal Oncocytoma, a Rare Occurrence

Rachel Musial, MD, Xingcao Nie, MD, PhD, J Steve Hou, MD
Drexel University College of Medicine: Department of Pathology and Laboratory Medicine

CASE REPORT

We report here a rare case of a renal oncocytoma with osseous metaplasia arising in a 45 year-old female. The mass was found incidentally on a CT of the abdomen and pelvis which revealed a 6.1 cm heterogeneous enhancing mass arising in the right kidney. She underwent a right total nephrectomy. Gross examination of the kidney revealed a circumscribed, pink-maroon to brown, focally hemorrhagic mass, measuring 5.2 x 4.8 x 3.5 cm, with central fibrous and firm areas (Fig 1). Light microscopy identified cells with abundant eosinophilic cytoplasm and osseous metaplasia (Fig 2). The osseous metaplasia consisted of multifocal areas of well-formed cancellous bone with osteoclasts, osteoblasts, adipose, and hematopoietic elements. Immunohistochemistry revealed that the tumor cells stain focal positive for vimentin and negative for CK7 and colloidal iron. Electron microscopy revealed abundant uniform mitochondria, confirming the diagnosis of an oncocytoma.

DISCUSSION

Osseous metaplasia has been reported in a variety of sites, including kidney, liver, gastrointestinal tract, and lung. Osseous metaplasia is a rare occurrence within renal neoplasms, occurring in approximately 4% of renal oncocytomas. In renal neoplasms, osseous metaplasia may be related to ischemia, necrosis, or inflammation. Several mechanisms have been proposed including a metaplastic or reparative response within the tumor or surrounding tissue, the production of bone by tumor cells, or ossification of pre-existing mucin or calcification. The prognostic significance of ossification in renal cell neoplasms is unclear. However, osseous metaplasia may indicate slow growth and a longer duration of tumor, which may suggest a less aggressive biology.

REFERENCES

1. Funai K, et al. Thymoma with osseous metaplasia. Journal of Computer Assisted Tomography. 2001;25(6):897-899
2. Perez-Ordonez B, et al. Renal oncocytoma: A clinicopathologic study of 70 cases. Am J of Surg Pathol. 1997;21(8): 871-883
3. Kefeli M, Yildiz L, Aydin O, Kandemir B, Yilmaz AF. Chromophobe renal cell carcinoma with osseous metaplasia containing fatty bone marrow element: A case Report. Pathology Research and Practice. 2007;203:749-752
4. Hoorweg JJ, Loftus BM, Hilgers FJM. Osteoid and bone formation in a nasal mucosal melanoma and its metastasis. Histopathology. 1997;31:465–468
5. Cribbs RK, et al. Renal cell carcinoma with massive osseous metaplasia and bone marrow elements. Ann Diagn Pathol. 1999;3:294-299

Figure 1. Gross examination of the bivalved kidney

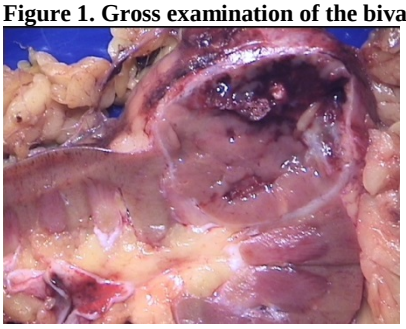
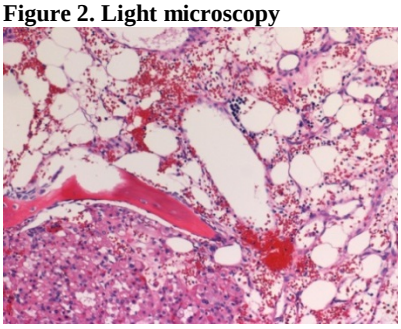


Figure 2. Light microscopy



Case Report: Palpitations as an Unusual Manifestation of a Multisystemic Disease

Julieta Gilson, MD, Koroush Khalighi, MD, Mahesh Krishnamurthy, MD
Easton Hospital: Department of Medicine

CASE REPORT

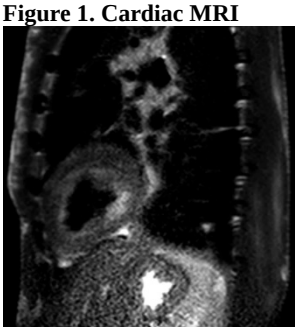
A 29 year-old African-American male presented with a three-month history of palpitations, weight loss, fatigue, dyspnea and night sweats. His physical exam revealed left conjunctival congestion and bilateral crackles in his lungs. EKG showed non-sustained ventricular tachycardia and CT chest revealed severe bilateral fibrosis of lung parenchyma. Transbronchial biopsy revealed non-caseating granulomas, confirming the diagnosis of sarcoidosis. Several episodes of non-sustained VT were documented. Cardiac MRI showed delayed gadolinium enhancement in the myocardium (Fig 1), highly suggestive of cardiac sarcoidosis. The patient was started on systemic steroids and methotrexate. An ICD was placed due to inducible sustained polymorphic ventricular tachycardia on cardiac electrophysiology.

DISCUSSION

Cardiac sarcoidosis is present in only 5% of cases of clinical sarcoidosis.[1,2,4] However autopsy reports indicate subclinical cardiac involvement in up to 20–30% and in populations like Japanese up to 60%.[4,5] Conduction abnormalities and ventricular arrhythmias are the most common causes of sudden death in these patients. There are no established criteria for specific diagnosis of cardiac

sarcoidosis.[3] The Japanese Ministry of Health proposed guidelines for diagnosis based either in histological confirmation by endomyocardial biopsy, which has a low sensitivity due to the patchy involvement, or histological confirmation of extra-cardiac sarcoidosis plus presence of conduction abnormalities or ventricular arrhythmias.[4,5] Our patient met the diagnostic criteria for the latter. Early recognition of cardiac sarcoidosis is challenging but vital because of high risk and lack of predictability for sudden cardiac death. Any patient with sarcoidosis should be evaluated for presence of conduction abnormalities and other arrhythmias.

Figure 1. Cardiac MRI



REFERENCES

1. Lannuzzi MC, Rybicki BA, Teirstein AS. Sarcoidosis. N Engl J Med. 2007 Nov 22;357(21):2153-65
2. Nery PB, Leung E, Birnie DH. Arrhythmias in cardiac sarcoidosis: diagnosis and treatment. Curr Opin Cardiol. 2012;27(2): 181-9
3. Morgenthau AS, Iannuzzi MC. Recent advances in sarcoidosis. Chest. 2011;139(1):174-82
4. Hamzeh N. Sarcoidosis. Med Clin North Am. 2011;95(6):1223-34
5. Yeboah J, Lee C, Sharma OP. Cardiac sarcoidosis: a review 2011. Curr Opin Pulm Med. 2011;17(5):308-15

Case Report: Persistent Left Superior Vena Cava Syndrome: What we should be aware of...

Nay Tun, MD, Thein Aung, MD, Swarnalatha Kanneganti, MD, Koroush Khalighi, MD
Easton Hospital: Department of Medicine

CASE REPORT

An 81-year-old woman with history of paroxysmal atrial fibrillation and hypertension presented with palpitations and shortness of breath. Due to recurrent symptomatic bradycardia, pauses and asystole preceded by transient episodes of non-sustained ventricular tachycardia, she was referred for a permanent pacemaker placement. While inserting the guide wire for pacemaker implantation, the left subclavian vein was found to have an unusual course. The guide wire crossed medially behind the heart, suggesting the presence of persistent left superior vena cava syndrome. Venography confirmed the presence of a large persistent left superior vena cava. Although a right ventricular pacing lead could still be placed, an atrial lead could not be positioned superiorly in an optimal position using this approach. Therefore, left cephalic vein approach was abandoned and the pacemaker was successfully inserted via the right pectoral approach through the right cephalic vein.

DISCUSION

Persistent left superior vena cava (PLSVC) is a rare accessory thoracic vein from embryological life that can be present with or without right superior vena cava. In general, PLSVC is incidentally identified during intra-thoracic procedures. It is found in 0.3-0.5% of the general population and in 3-5% in association with congenital heart diseases.[1] PLSVC should be considered when experiencing difficulties with central venous access since it is one of the most common thoracic vein anomalies. It is important to notice and recall the abnormalities of the anatomic structures that could be accidentally found during thoracic operations or accessing central lines.

REFERENCES

1. Bhatti S, et al. Persistent left superior vena cava (PLSVC) with anomalous left hepatic vein drainage into the right atrium: role of imaging and clinical relevance. Vascular Medicine 2007;12:319–324
2. MacDonald ST, et al. Absent Superior Vena Cava Multimodality Imaging of Upper Body Venous Drainage via Left-sided Superior Vena Cava and Azygous Venous System. Circulation: Cardiovascular Imaging. 2009;2:e34-e36 doi:10.1161/CIRCIMAGING.108.828558
3. Muster AJ, et al. Is surgical ligation of an accessory left superior vena cava always safe? Pediatr Cardiol 1998;19:352–354
4. Santoscoy RS, et al. Coronary sinus ostial atresia with persistent left superior vena cava. Ann Thorac Surg 1996;61:879 –882

Case Report: Radiographic Assessment of Craniocervical Injury with Computed Tomography

Blachy Davila, MD*, Robert Koenigsberg, DO*, Ripple Sheth, DO***
*Drexel University College of Medicine: Department of Radiologic Sciences
**Philadelphia College of Osteopathic Medicine: Department of Medicine

CASE REPORT
An unresponsive adult male presented to the ED after MVA. Cervical CT demonstrated comminuted fracture of C1, left atlanto-occipital dislocation and bilateral atlanto-axial dislocation. Spinal cord compression, subarachnoid hemorrhage and a large prevertebral hematoma were also found and the patient subsequently expired.

DISCUSSION
Craniocervical injury is a leading cause of death and disability in trauma. Cervical injury occurs in 5-10% of unconscious ED patients presenting with fall or MVA. Approximately 20% of traffic fatalities involve upper cervical spine injury. Craniocervical injury can range from ligamentous to isolated fractures and ultimately, atlanto-occipital or atlanto-axial dislocation. The CCJ is formed by articulations and ligaments involving the occiput, atlas, and axis. Stabilizing ligaments include anterior and posterior atlanto-occipital membranes, alar ligaments, transverse ligament, and tectorial membrane. The most accurate, reproducible measurements to evaluate the CCJ are obtained sagitally: basion-axial (BAI), basion-dental (BDI), atlanto-dental (ADI) and atlanto-occipital (AOI) intervals. Abnormal BAI and BDI (>12 mm) imply injury to a stabilizing ligament. Abnormal ADI (>3mm) and AOI (>2 mm) imply transverse ligament and atlanto-occipital articulation with tectorial membrane/alar ligament injury, respectively. Craniocervical junction injury is uncommon but prompt diagnosis is essential for prevention of further trauma and early management, given the possibility of instability and neurovascular compromise.

REFERENCES
1. Bono, et al. Measurement Techniques for Upper Cervical Spine Injuries: Consensus Statement of the Spine Trauma Study Group. Spine. 2007;32(5):593-600
2. Davenport, et al. (2011, August 15). Cervical Spine Fracture in Emergency Medicine. Medscape Reference. Retrieved from http://emedicine.medscape.com/article/824380-overview
3. Deliganis, et al. Radiologic Spectrum of Distraction Injuries. October 2000 RadioGraphics, 20, S237-S250
4. Harris, et al. Radiologic Diagnosis of Traumatic Occipitovertebral Dissociation: 2. Comparison of Three Methods of Detecting Occipitovertebral Relationships on Lateral Radiographs of Supine Subjects. AJR. 1994;162(4):887-892
5. Rojas, et al. Reassessment of the Craniocervical Junction: Normal Values on CT. Am. J. Neuroradiol. 2007;28(9):1819-1823

Figure 1. Saggital CT images

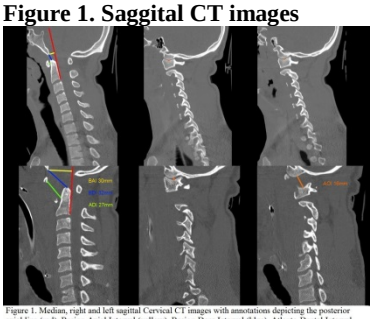


Figure 1. Median, right and left sagittal Cervical CT images with annotations depicting the posterior axial line (red), Basion-Axial Interval (yellow), Basion-Dens Interval (blue), Atlanto-Dental Interval (green) and Atlanto-Occipital Interval (orange). Normal comparison images above, case images below.

Figure 2. Coronal CT images and 3D rendering

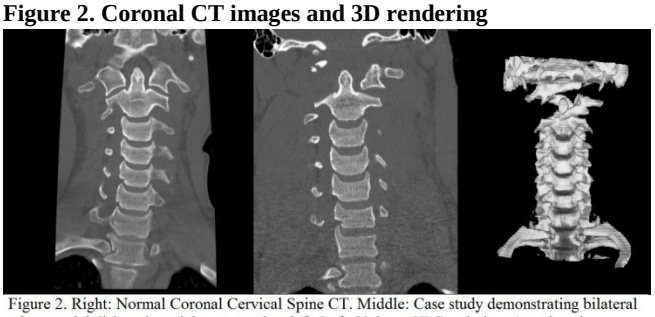


Figure 2. Right: Normal Coronal Cervical Spine CT. Middle: Case study demonstrating bilateral atlanto-axial dislocation, right greater than left. Left: Volume 3D Rendering, Anterior view.

Case Report: Rare but Serious Complication of a Benign Procedure

Hadie Razjouyan, MD
Monmouth Medical Center: Department of Medicine

CASE REPORT
A 54 year-old female was found unresponsive and admitted to the ICU under impression of septic shock and started on broad spectrum antibiotic therapy. Later the course became associated with diarrhea which was negative for C. difficile colitis as well as enteric pathogens and was diagnosed as antibiotic-associated diarrhea. Since the patient was morbidly obese and sedated because of intubation, to avoid stool soiling and more complications like decubitus wounds, a rectal tube was placed. In 4 days, she developed heavy lower gastrointestinal fresh bleeding. She underwent urgent colonoscopy and found to have two proximal rectal ulcers one of which was actively bleeding. The ulcers were in opposite rectal walls.

DISCUSSION
Although rectal tube placement is safe, painless,[1] technically easy and usually done by registered nurses specifically in patients with active diarrhea,[1,2] it may have serious complications as mentioned in our case and recent

reports.[3] Simple steps before the procedure may help avoiding unwanted complications. First, look for the reason of diarrhea or stool soiling and avoid the predisposing agents or factors. Second, if placing a tube in an obese patient and the finger guide will not be long enough for proper placement, it is better to advance it before inflating the balloon and not continuing to inflate more than usual resistance occurs. Third, perform daily surveillance to look for tube necessity and eventually the sooner it is removed, the fewer complications occur.

REFERENCES

1. Channick R, Irwin RS. Indications for and complications of rectal tube in critically ill patients. Journal of Intensive Care Medicine, 1988;3:321-3
2. Benoit RA, Jr. and Watts C. The effect of a pressure ulcer prevention program and the bowel management system in reducing pressure ulcer prevalence in an ICU setting. J Wound Ostomy Continence Nurs, 2007;34(2):163-75
3. Popek S and Senagore A. Indwelling rectal tubes: an unusual cause of significant rectal bleeding in two critically ill patients. Am Surg, 2013;79(2)219-20

Case Report: Rare Case of Acute Cholecystitis due to Transitional Cell Carcinoma Metastasis from the Urinary Bladder to the Gallbladder

Uchechukwu Stanley Ogu, MD, Robert Bloch, MD, Scott Caganap, Sonia Jasuja
Easton Hospital: Department of Surgery

INTRODUCTION

Metastatic disease of the gallbladder rarely causes signs and symptoms of gallbladder disease.[1,2] This case is the first and only report of symptomatic urinary bladder cancer metastasis to the gallbladder.

CASE REPORT

A 79-year-old male receiving chemotherapy for bladder cancer presented to the emergency department with nausea, emesis, and right upper quadrant pain for 3 days. The patient had been diagnosed 9 months prior to presentation with high-grade invasive transitional cell carcinoma. Physical exam elicited positive Murphy's sign. Laboratory findings and imaging suggested acute cholecystitis. The patient received an uncomplicated laparoscopic cholecystectomy. The staining and histological characteristics of the pathology specimen showed the tumor penetrating inward from the serosal surface of the gallbladder, most consistent with metastatic bladder carcinoma.

DISCUSSION

Urinary bladder cancer is the fifth most common cancer in men. Approximately 73,510 new cases of bladder cancer were diagnosed in the U.S. in 2012.[4] Bladder cancer spreads to distant sites in 4% of cases.[4] The most common metastatic site is to the lymph nodes, followed by bone, lung, liver and peritoneum.[5] One case of distant metastasis to the biliary tree has been identified describing bile duct wall metastasis from bladder cancer.[3] Little data exists on secondary tumors of the gallbladder. One study revealed a 5.8% incidence of metastasis to the gallbladder.[1] The most common primary tumor is melanoma, followed by renal cell carcinoma and breast cancer.[2] The most common presentation is right upper quadrant or epigastric pain imitating cholecystitis followed by jaundice.[2]

REFERENCES

1. Abrams HL, Spiro R, Goldstein N. Metastases in carcinoma; analysis of 1000 autopsied cases. Cancer. 1950;3(1):74-85
2. Dong XD, DeMatos P, Prieto VG, Seigler HF. Melanoma of the gallbladder: a review of cases seen at Duke University Medical Center. Cancer. 1999;85(1):32-39
3. Hong SP, et al. Bile duct wall metastasis from micropapillary variant transitional cell carcinoma of the urinary bladder mimicking primary hilar cholangiocarcinoma. Gastrointestinal Endoscopy. 2002;56(5):756-758
4. American Cancer Society. Cancer Facts & Figures 2012. Atlanta, Ga: American Cancer Society. 2012
5. Shinagare AB, et al. Metastatic pattern of bladder cancer: correlation with the characteristics of the primary tumor. AJR. 2011; 196:117-122

Case Report: Refractory Orthostatic Hypotension in a patient with Small Cell Lung Cancer treated with Radiation

WuQiang Fan, MD, Thein Aung, MD, Mahesh Krishnamurthy, MD
Easton Hospital: Department of Medicine

CASE REPORT

A 58 year-old male with small cell carcinoma of the right upper lung received 2 cycles of Cisplatin and Etopside–based chemotherapy concurrently with a total dose of 4640 cGy radiation to the lower neck, right lung, and mediastinum. Following the radiotherapy, he developed transient hypertension followed by persistent orthostatic hypotension (supine: 130/71; upright: 73/46) which was refractory to intravenous fluids. A diagnosis of baroreflex failure (BF) was made based on the history of extensive radiotherapy. The patient was started on midodrine, fludrocortisones and TED Stockings. Physical maneuvers were also advised to decrease fluctuations in blood pressure. His orthostatic hypotension has since improved

on follow-up. Differential diagnosis includes paraneoplastic autonomic neuropathy, which is often seen in small cell lung cancer, and platinum-induced neuropathy.

DISCUSSION

Baroreceptors are specialized neurons in the aortic arch (innervated by CN X) and carotid sinuses (innervated by CN IX) to monitor changes in blood pressure. Baroreflex can be measured by methods involving vasoactive drugs, Valsalva maneuver, and neck chamber.[2] BF is an under-recognized cause of labile blood pressures with orthostatic changes.[3] Patient may present with labile hypertension, orthostatic hypotension, and syncope. BF as a late sequela of neck irradiation has been reported.[4] Our present case suggests that radiation-related damage of the baroreceptors in aortic arch has similar consequences. Clonidine is the antihypertensive of choice for supine hypertension. Fludrocortisone, midodrine and TED stocking are helpful in orthostatic hypotension. Physical maneuvers like periodic squatting help maintain blood pressure when upright.

REFERENCES

1. Koike H, Tanaka F, Sobue G. Paraneoplastic neuropathy: wide-ranging clinicopathological manifestations. Curr Opin Neurol. 2011;;24(5):504-10
2. La Rovere MT, Pinna GD, Raczak G. Baroreflex sensitivity: measurement and clinical implications. Ann Noninvasive Electrocardiol. 2008;13(2):191-207
3. Timmers HJ, Wieling W, Karemaker JM, Lenders JW. Cardiovascular responses to stress after carotid baroreceptor denervation in humans. Ann N Y Acad Sci. 2004;1018:515-9
4. Sharabi Y, Dendi R, Holmes C, Goldstein DS. Baroreflex failure as a late sequela of neck irradiation. Hypertension. 2003;42(1):110-6

Case Report: Silicone Injections

David Swanson, MD, Avrumi Zlochower, MD Candidate
Drexel University College of Medicine: Department of Radiologic Sciences

CASE REPORT

A 46 year-old genotypically male, phenotypically female patient reported a history of injection of ~1200 cc of silicone into the buttocks during a single session for cosmetic enhancement. Since the procedure the patient has experienced erythema, nodularity, induration, and migration of injected material. Abscesses developed and were drained. The patient was admitted with persistent fever and a CT of the abdomen and pelvis with contrast was obtained, showing extensive chronic inflammatory changes relating to the injections (Fig 1).

DISCUSSION

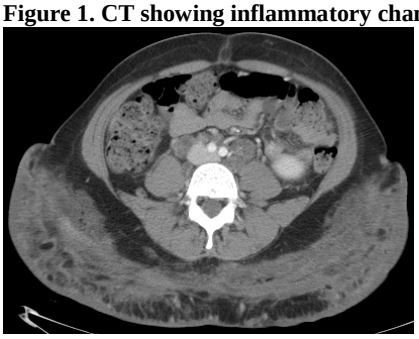
Liquid silicone is a group of polymers containing silicon conjugated with oxygen and methane that renders the substance theoretically physiologically inert and non-immunogenic. Silicone injections have been performed by both medical and non-medical personnel in a variety of sites since the 1940's.[2] As a result of improper technique, severe complications such as granulomatous disease, pneumonitis, silicone pulmonary embolism, and fatalities have occurred.[1,2,3] When performed by trained personnel, miniscule droplets of pharmaceutical grade silicone are implanted at appropriate depths and locations using microdroplet technique. These droplets become encapsulated by a network of fibrodysplasia. This requires adequate time between injections. The result is a structure that is not palpable, has the texture of adjacent tissue, and will typically not migrate.[1]

Non-medical injections in particular have been troubling as they often involve injection of a large volume of impure material by unlicensed practitioners.[1] This case is presented to highlight the unintended effects that often accompany cosmetic injections performed utilizing improper technique.

REFERENCES

1. Narins RS, Beer K. Liquid injectable silicone: a review of its history, immunology, technical considerations, complications, and potential. Plast Reconstr Surg. 2006;118(3 Suppl):77S-84S
2. Restrepo CS, et al. Silicone pulmonary embolism: report of 10 cases and review of the literature. J Comput Assist Tomogr. 33(2):233-7
3. Paredes vila S, et al. Pneumonitis caused by silicone gel following breast implant rupture. Ir J Med Sci. 2010;179(1):141-5
4. Altmeyer MD, Anderson LL, Wang AR. Silicone migration and granuloma formation. J Cosmet Dermatol. 2009;8(2):92-7
5. Pasternack FR, Fox LP, Engler DE. Silicone granulomas treated with etanercept. Arch Dermatol. 2005;141(1):13-5

Figure 1. CT showing inflammatory changes.



Case Report: Simple as it appears, is never simple!

Nagakrishnal Nachimuthu, MD, Alan Haratz, MD, Peter Lapmann, MD, Jeffrey Lederman, MD, Sara Wallach, MD
Monmouth Medical Center: Department of Medicine

CASE REPORT

A 73 year-old Caucasian lady came to the emergency room with complaints of diarrhea and abdominal pain for 1 day. She was seen during previous admission for dizziness and imbalance for which CT brain showed lacunar infarct posterior aspect of right internal capsule. Physical examination revealed diffuse lower abdominal tenderness. Patient was started on antibiotics and intravenous fluids. She improved, however it was noticed that her leukocytosis persisted and platelet count trended up. Given the thrombocytosis, lacunar infarct, malignancy was included on the differential. PET scan was unremarkable. She was empirically started on high dose steroids. Kidney biopsy was reported as focal segmental necrotizing and cresenteric glomerulonephritis consistent with diagnosis of microscopic polyangiitis (MPA).

DISCUSSION

Table 1. Lab and study results

WBC	31.7, 7% BANDS
HB	11.7
PLATELETS	598 - 700 - 900
BUN	49 BASELINE-20
CREATININE	3.41 BASELINE -0.5
URINE ANALYSIS	MODERATE PROTEIN AND BLOOD
ANA	POSITIVE WITH 1:160 DIFFUSE PATTERN
C4	LOW
PROTEINASE 3	HIGH
LFT	NORMAL
STOOL FOR C DIFF	PENDING RESULTS
CT ABDOMEN	CECAL WALL THICKENING SUGGESTING FOCAL COLITIS

MPA is associated with symptoms that affect kidneys, nervous system, lungs, and skin. When a patient presents with a constellation of symptoms that involves ears, skin, lungs, kidneys, anemia, thrombocytosis, cerebral infarcts, conduction abnormalities, and uveitis, differential diagnosis should include ANCA associated vasculitis.[1,2,3] Biopsy is required to make a definitive diagnosis. However, if clinical suspicion is high, patients should be treated empirically. Prompt diagnosis and treatment can be organ and/or life-saving.[1]

REFERENCES

1. Jayne D. The diagnosis of vasculitis. Best Pract Res Clin Rheumatol 2009;23:445
2. Jennette JC, Falk RJ. Small-vessel vasculitis. N Engl J Med 1997;337:1512
3. Hoffman GS, et al. Wegener granulomatosis: an analysis of 158 patients. Ann Intern Med 1992;116:488

Case Report: Takotsubo Syndrome: Atypical presentation

Abraham Tacang, MD, Mohamed Sheta, MD
Monmouth Medical Center: Department of Medicine

CASE REPORT

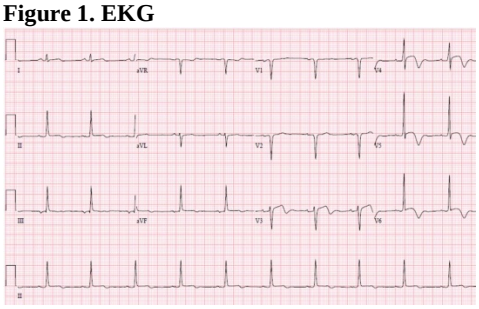
A previously healthy 60 year-old female presented to her PMD after she developed an attack of chest heaviness lasting for two hours, radiating to her left shoulder with associated diaphoresis. She felt fine in the evening but the next morning she felt fatigued. EKG showed ST elevation in V2-3 with T-wave inversion in antero-lateral leads pointing to septal infarction (Fig 1). Initial Troponin-I was 0.62 ng/mL. Cardiac catheterization showed no significant coronary artery disease with apical and septal akinesis, findings consistent with Takotsubo cardiomyopathy. The patient admitted that her husband has some financial difficulties over the last year but she denies any acute emotional or physiologic stressors. The patient was admitted to the ICU and conservative management was initiated.

DISCUSSION

Takotsubo cardiomyopathy is increasingly recognized among patients initially presenting with acute coronary syndrome. Characteristic clinical features include acute chest pain, dynamic EKG changes usually ST-segment elevations and eventual T-wave inversions, apical ballooning and absence of obstructive coronary artery disease. These clinical features are mainly reversible. Most patients are women over the age of 50, whose presentations are associated with an inciting stressful event. Pathophysiology remains unclear. We are presenting an atypical case of Takotsubo cardiomyopathy where the chest pain was transient and persisted for only two hours, making the prediction of the diagnosis very remote. We strongly believe that increasing the awareness of milder forms of the disease will help early diagnosis and hence proper and timely management.

REFERENCES
1. Wittstein I, et al. Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress. NEJM. 2005;362:539-48
2. Samardhi H, et al. Takotsubo cardiomyopathy: an Australian single centre experience with medium term follow up. Int Med J. 2012;42:35-42
3. Sealove B, Tiyyagura S, and Fuster V. Takotsubo Cardiomyopathy. JGIM. 2008;23:1904-8

Figure 1. EKG



Case Report: Thyroid Dysgenesis as a Cause of Congenital Hypothoidism (CH)

Ashish Gupta, MD, Kanika Shanker, MD
Saint Peter's University Hospital: Department of Pediatrics

CASE REPORT
The full term baby born to G2P2 mother had a birth weight 2.995 kg, length 49 cm and head circumference 35 cm. Neonatal examination was completely normal. The newborn was found to be jaundiced on day 6 of life at the pediatrician visit. Bilirubin was found to be 24.5mg/dl with direct <10%. The infant was immediately admitted to the NICU and was started on phototherapy. On day 7 of life, the state newborn screening laboratory called regarding elevated level of TSH of 600mU/l. The baby was started on levothyroxine. The repeat labs showed TSH >100mU/l. Later the technetium thyroid scan showed complete non-visualization of thyroid gland.

DISCUSSION
According to world-wide data obtained from neonatal thyroid screening programs CH occurs with an incidence of 1:3000 to 1:4000. Thyroid dysgenesis, which includes aplasia, hypoplasia, and an ectopic gland, is the most common cause of congenital hypothyroidism, accounting for 85% of cases. The most common form of thyroid dysgenesis is an ectopic gland, demonstrated by a thyroid scan or ultrasonography. 10% of dysgenesis are caused by an inborn error of thyroxine synthesis and the remaining 5% are the result of transplacental maternal thyrotropin-receptor blocking antibodies. The clinical features during the first two postnatal weeks are enlarged head circumference due to brain myxedema and prolonged neonatal jaundice. Severe hypothyroidism before birth and a delay in treatment after birth are associated with an impaired intellect and neuro-psychological abnormalities. Therefore treatment should be started as soon as possible. The drug of choice is levothyroxine at a dose of 10-15 mcg/kg/day with goal of maintaining the FT4 1.2-2.3 ng/dL and TSH <6 mU/L. Monitoring of thyroid function should be obtained during the initiation phase of treatment and with any dosage change.

REFERENCES
1. Fisher DA. Second International Conference on Neonatal Thyroid Screening: progress report. JPediatr 1983;102(5):653-654
2. Kaplan SA. Clinical pediatric endocrinology. 2nd edition. Edited by Solomon A Kaplan. Philadelphia. Saunders;1990

Case Report: Unwinding Life In The Heart; A Case of Internal Cardioverter-Defibrillator Lead Malfunction

Swarnalatha Kanneganti, MD, Thein Aung, MD, Koroush Khalighi, MD
Easton Hospital: Department of Medicine

CASE REPORT
A 63 year-old, pacemaker dependent woman presented with complaint of hearing “beeping sounds” from her internal cardioverter-defibrillator (ICD). Interrogation of her defibrillator revealed a gradual increase in the lead impedance of over 2000 ohms from the past five months prior to this presentation. The ICD lead fluoroscopy showed externalization of the ICD lead components between proximal and distal coils, a rare finding that has been recently reported in the literature with St. Jude Medical Riata ICD leads.[2,3,4] She subsequently underwent a successful ICD lead replacement.

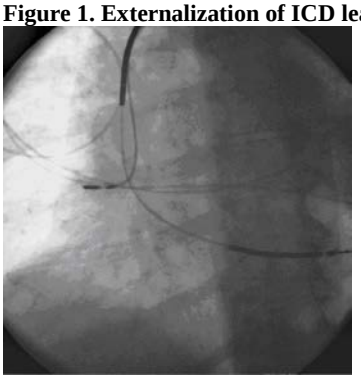
DISCUSSION
ICD lead malfunctions such as disintegration and externalization of the coils are rare but serious

complications noted with Riata ICD leads1, and are of growing concern. Although extremely uncommon, few pacemaker and ICD lead manufacturers had malfunctioning leads resulting in a series of advisories and recalls.[5] A rare possible risk exists for serious injuries and death resulting from inappropriate sensing, failure to capture or to deliver shock, and perforation of the heart. As a result, the medical community has faced serious medical, psychological and financial burden. Most recent examples for such serious problems are Telectronics Accufix pacing leads, Medtronic's Sprint Fidelis lead, and St. Jude Medical Riata ICD lead malfunctions. Externalization of the ICD lead is rare, but is a potentially serious complication of the newer ICD systems. There is no consensus if patients who already have these leads implanted should have them replaced. Due to significant medico-legal risks involved, a clear communication between patients and their physicians is essential.

REFERENCES

1. MedPage Today; Higher Failure Rates with Riata Leads Affirmed; http://www.medpagetoday.com/MeetingCoverage/HRS/32673 (Accessed on 28 January 2013)
2. Vlay SC. Concerns about the Riata ST (St. Jude Medical) ICD lead. Pacing Clin Electrophysiol. 2008;31(1):1-2
3. Dorman HG, van Opstal JM, Stevenhagen J, Scholten MF. Conductor externalization of the Riata internal cardioverter-defibrillator lead: tip of the iceberg? Report of three cases and review of literature. Europace. 2012 Mar 19
4. Parvathaneni SV, Ellis CR, Rottman JN. High prevalence of insulation failure with externalized cables in St. Jude Medical Riata family ICD leads: Fluoroscopic grading scale and correlation to extracted leads. Heart Rhythm. 2012 Mar 23
5. US Food and Drug Administration; FDA Classifies Voluntary Physician Advisory Letter on Riata and Riata ST Silicone Defibrillation Leads as Class I Recall (URGENT MEDICAL DEVICE ADVISORY) http://www.fda.gov/Safety/Recalls/ucm283879.htm (Accessed on 28 January 2013).

Figure 1. Externalization of ICD lead wire between superior vena caval and right ventricular coils



Case Report: Wernicke's Encephalopathy: A life-threatening complication of thiamine deficiency

Haseeba Shahzad, MD, Swarnalatha Kanneganti, MD, Jekaterina Tretyakov, MD
Easton Hospital: Department of Medicine

CASE REPORT

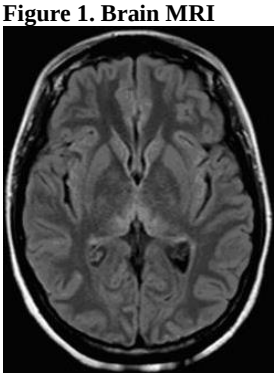
A 31 year-old woman with a history of morbid obesity, status post gastric bypass surgery three months ago, presented with one week history of diplopia, blurred vision, and gait instability. She also noticed numbness and weakness in both lower extremities. On physical exam eyes were convergent with vertical nystagmus and movements were restricted to downward gaze. Sensation was intact. In both lower extremities power was decreased and deep tendon reflexes were absent. Mild ataxia was noted. One month prior to this presentation, the patient stopped taking medications including vitamin supplements due to intractable vomiting. Wernicke's encephalopathy was strongly suspected[1,2] due to history of gastric bypass,[3] medication, noncompliance and clinical features of nystagmus, ataxia, and ophthalmoplegia.[3] Immediately, intravenous thiamine replacement was initiated. MRI of the brain showed restricted diffusion in medial thalami extending towards the periaqueductal gray matter and quadrigeminal plate, suggesting Wernicke's encephalopathy (Fig 1). Eventually, with thiamine supplementation, the patient's symptoms improved.

DISCUSSION

Wernicke's encephalopathy is an acute life-threatening neurologic disorder caused by thiamine deficiency.[1] Thiamine acts as a cofactor for the key enzymes in metabolism. Its deficiency may cause neuronal injury in the areas of brain with high thiamine requirement, especially surrounding the aqueduct, third and fourth ventricles. Mamillary bodies are involved in virtually all cases, and dorsomedial thalamus, locus ceruleus, periaqueductal gray matter, occulomotor and vestibular nuclei are commonly affected. Wernicke's encephalopathy should be strongly considered in patients presenting with acute delirium or ataxia. Failure to treat can lead to permanent neurologic damage or death.

REFERENCES
1. Sechi G, Serra A. Wernicke's encephalopathy: new clinical settings and recent advances in diagnosis and management. Lancet Neurol. 2007;6(5):442-55
2. Donnino MW, Vega J, Miller J, Walsh M. Myths and misconceptions of Wernicke's encephalopathy: what every emergency physician should know. Ann Emerg Med. 2007;50(6):715-21
3. Mrsic V, et al. Wernicke's encephalopathy as a complication of bariatric surgery. Acta Med Croatica. 2012;66(1):17-22

Figure 1. Brain MRI



Article: A Novel Biologic Prosthetic Patch for the Repair of Congenital Diaphragmatic Hernia of Bochdalek in Infants

Leah Sieren, MD, L. Grier Arthur, MD, Noor Kassira, MD, Michael Katz, MD, Rajeev Prasad, MD
St. Christopher's Hospital for Children: Department of Surgery

ABSTRACT

Introduction: The surgical repair of a congenital diaphragmatic hernia (CDH) in infants can be challenging, particularly when a primary repair is not feasible and a prosthetic patch must be used. We present our experience with CDH repair utilizing XenMatrix™, a biologic non-cross-linked acellular porcine dermal collagen matrix.

Methods: Under IRB protocol #19940, we retrospectively reviewed the charts of all infants who underwent repair of a CDH with XenMatrix™. Patient gestational age and age and weight at the time of surgery were noted. Any intraoperative or postoperative complication and length of follow-up were recorded.

Results: Between April 2009 and April 2011, XenMatrix™ was used for the repair of a CDH in three patients. All patients had a left-sided CDH. The XenMatrix™ patch was placed via a thoracoscopic approach in one patient for an initial repair. Two patients underwent repair of a recurrent CDH, one completed utilizing a thoracoscopic approach and the other via an open abdominal approach. Of the two patients with a recurrent CDH, one originally had a primary closure via an open approach and the other originally had a Gore-Tex patch placed thoraco-scopically. The mean gestational age was 37±3 weeks. The mean age at surgery was 143 days (range 4-260 days), and the mean operative weight was 5±2 kg. There were no intra-operative or post-operative complications. After a mean follow-up of 31 months (range 21-45 months), no patient in whom XenMatrix™ was used has had a recurrence of the CDH.

Discussion: In each of the three cases of CDH, a prosthetic patch was required because the diaphragmatic defect could not be closed primarily. We chose to utilize XenMatrix™, a biologic material with a high burst strength that does not require rehydration, has no sidedness and is easy to manipulate, especially when a minimally invasive approach is utilized. Thus far in our experience there have been no complications or recurrences. We believe that XenMatrix™ should be considered for the repair of a congenital diaphragmatic hernia when a patch is necessary for closure.

BACKGROUND

The surgical repair of a congenital diaphragmatic hernia (CDH) was first successfully performed in a child in 1902 via an open abdominal incision.[1] Subsequently, repair via a thoracotomy was described with the advantages being the avoidance of an abdominal exploration and the ability to push rather than pull the abdominal contents through the hernia defect.[2] In 1995 the first thoracoscopic repair of a CDH was reported.[3] This coupled the advantages of a thoracic approach to the repair, listed above, with the advantages of a minimally invasive approach, including the potential for decreased postoperative pain and better cosmesis while maintaining an equivalent hospital stay.[4] Regardless of the approach, when a diaphragmatic defect cannot be closed primarily, a prosthetic patch is required. Historically, Gore-Tex® has been utilized most often.[5] In recent years, many other types of prosthetic materials, both synthetic and biologic, have been introduced as options.[6-11] We describe our experience in patients with CDH, both initial and recurrent, successfully repaired using XenMatrix™, a biologic non-cross-linked acellular porcine dermal collagen matrix graft.

METHODS

Under IRB protocol #19940, we retrospectively reviewed the charts of three infants who underwent repair of a CDH with XenMatrix™. Patient gestational age and age and weight at the time of surgery were noted. Any intraoperative or postoperative complication and length of follow-up were recorded. Statistical analysis was performed using InStat Version 3.10 (Graphpad Software, La Jolla, CA). Results are presented as mean±SD.

RESULTS

Three patients underwent a repair of a CDH using XenMatrix™. These 3 operations were performed by 2 surgeons. One of the 3 patients underwent an initial repair, which was done thoracoscopically, and 2 of the patients required repair of a recurrent CDH with one done thoracoscopically and the other completed via an open abdominal approach.

The first case was that of an 8-month old male with a history of a left-sided CDH repair done at another institution in the newborn period via an open abdominal approach. This patient's postoperative course was complicated by an episode of severe necrotizing enterocolitis necessitating a second laparotomy and bowel resection. The patient was eventually discharged to home, but he was admitted to our hospital with respiratory syncytial virus pneumonia. A chest radiograph done at the time of admission to our hospital demonstrated a recurrent CDH (Fig 1a). A CT scan confirmed intestine and the left kidney to be in the left hemithorax (Fig 2). After an appropriate period of medical management to optimize his respiratory status, the patient was taken to the operating room for a thoracoscopic repair of his recurrent left-sided CDH using XenMatrix™. The patient was placed in a lateral position. Three 5mm trocars and a 30 degree telescope were utilized. Upon entry into the chest, significant adhesions were encountered. Lysis of these adhesions revealed the colon and left kidney in the left hemithorax. The viscera were gently reduced into the abdomen. The medial aspect of the defect was closed primarily with interrupted sutures of 2-0 silk. However, due to excessive tension and the absence of an adequate diaphragmatic rim laterally, a graft was necessary to complete the closure of the defect. A XenMatrix™ patch was trimmed to an appropriate size and passed into the chest. Several interrupted sutures of 2-0 silk were used to secure the patch in place to the remnant of the diaphragm. The 5 most lateral sutures, where there was no diaphragmatic rim, were passed around adjacent ribs (Fig 3). A single chest tube was left to drain the pleural space. Thus far, after a 45 month period of follow-up, the patient remains asymptomatic without evidence of recurrence, either clinically or by chest radiograph (Fig 4a).

The second patient was a 3.2 kg full term male transferred to our institution because of respiratory distress in the delivery room and a subsequent chest radiograph demonstrating bowel loops in the left hemithorax (Fig 1b). The patient remained hemodynamically stable, and so on day of life 4 he underwent a thoracoscopic CDH repair with a XenMatrix™ patch. To accomplish this, the patient was placed in a lateral decubitus position. Three 5mm trocars and a 30 degree telescope were utilized. The defect was noted to be large. Medially, the diaphragm was approximated primarily using 2-0 braided polyester sutures. Laterally, the gap was too wide and so a XenMatrix™ patch was utilized to bridge the defect. As was done in the first case, the patch was sewn to the rim of the diaphragm medially and to the chest wall musculature laterally with the sutures passed around adjacent ribs. A single chest tube was left to drain the pleural space. Thus far, after a 27 month period of follow-up, the patient remains asymptomatic without evidence of recurrence, either clinically or by chest radiograph (Fig 4b).

The third patient was a 5-month old girl who previously underwent a thoracoscopic repair of a large left-sided CDH in the newborn period utilizing a Gore-Tex® patch. Subsequently, the patient required a laparoscopic Nissen fundoplication and gastrostomy for severe gastroesophageal reflux and failure to thrive. At 5 months of age, the patient was readmitted to the hospital because of respiratory distress. A chest radiograph suggested a recurrent CDH (Fig 1c). A CT scan confirmed the suspicion as intestine was found to be in the left hemithorax. Following a period of medical optimization, the patient was taken to the operating room for a repair of the recurrent CDH utilizing XenMatrix™. A thoracoscopic approach was initially used for this recurrent CDH. The patient was positioned in a lateral decubitus position. As in the first 2 cases, a 3 trocar approach and a 30 degree telescope were utilized. Significant thoracic adhesions were encountered and lysed, and the abdominal contents were reduced. The previously placed Gore-Tex® patch was noted to have pulled away laterally and posteriorly, and so it was excised. Because some abdominal viscera, including the colon, appeared to be closely adherent to the abdominal side of the diaphragm (perhaps related to the previous fundoplication), we converted to an open abdominal procedure. The colon was in fact densely adherent to the diaphragm just medial to the defect. Following lysis of adhesions, a Xen-Matrix™ patch was utilized to close the defect using 2-0 braided polyester sutures (once again passed around the ribs posterolaterally where there was no diaphragmatic rim). Thus far, after a 21 month period of follow-up, the patient remains asymptomatic without evidence of recurrence, either clinically or by chest radiograph (Fig 4c).

DISCUSSION

In our series, we utilized both thoracoscopic and open techniques to repair all 3 left-sided diaphragmatic hernias using XenMatrix™, which is approved by the Food and Drug Administration for the repair of diaphragmatic herniation, to bridge the defect as a primary closure was impossible in all 3 instances. The XenMatrix™, which does not require rehydration and has no sidedness, is relatively easily manipulated and sutured into place without concern regarding the adjacent viscera which remains juxtaposed to the patch. In the 2 patients with recurrent CDH we felt it to be advantageous to avoid a synthetic patch and the associated risk of infection as we expected to have to lyse adhesions and, in the case of the patient with a history of necrotizing enterocolitis, mobilize previously inflamed intestine. For this reason, we opted to use a biologic patch. Other commercially available biologic patches are rigid, even after rehydration, making it difficult to manipulate and suture these materials.[12] Also, that some of these

patches have sidedness poses a distinct problem with CDH repair as vital structures lie on both sides of the repair. XenMatrix™ is an acellular structurally intact non-cross-linked porcine dermal collagen matrix. This dermal matrix has been shown to induce neovascularization and tissue remodeling and regeneration by two weeks, and have shown greater tissue integration and less inflammation than cross-linked collagen implants or porcine small intestine submucosal implants.[13,14] The burst strength of the material also may be superior to other porcine grafts.We believe that XenMatrix™ should be considered as an option for the repair of a congenital diaphragmatic hernia when a patch is necessary for closure of the defect.

REFERENCES

1. Heidenhain L.Geschichte eines Falles von chronischer Incarceration des Magens in einer angeborenen Zwerchfellhernie welcher durch Laparotomie geheilt wurde mit anschliessenden Bemerkungen uber die Moglichkeit das Kardiacarcinom der Speiserohre zu reseszіren. Deutsch Z Chir 1905;76:394-403
2. Hume JB. Congenital Diaphragmatic Hernia. Br J Surg 1922;10:207-215
3. Silen ML, Canvasser DA, Kurkchubasche AG, Andrus CH, Naunheim KS. Video-assisted thoracic surgical repair of a foramen of Bochdalek hernia. Ann Thorac Surg 1995;60:448-50
4. Cho SD, et al. Analysis of 29 consecutive thoracoscopic repairs of congenital diaphragmatic hernia in neonates compared to historical controls. J Pediatr Surg. 2009;44:80-6
5. Keijzer R, et al. Thoracoscopic repair in congenital diaphragmatic hernia: patching is safe and reduces the recurrence rate. J Pediatr Surg 2010;45:953-7
6. Geisler F, Grotlieb A, Fried D. Agenesis of the diaphragm repaired with Marlex. J Pediatr Surg 1977;12:587-588
7. Riehle KJ, Magnuson DK, Waldhausen JH. Low recurrence rate after Gore-Tex/Marlex composite patch repair for posterolateral congenital diaphragmatic hernia. J Pediatr Surg. 2007;42:1841-4
8. Newman BM, et al. Prosthetic materials and muscle flaps in the repair of extensive diaphragmatic defects: an experimental study. J Pediatr Surg 1985;20:362-367
9. Atkinson JB, Poon MW. ECMO and the management of congenital diaghragmatic hernia with large diaphragmatic defects requiring a prosthetic patch. J Pediatr Surg 1992;27:754
10. Grethel EJ, et al. Prosthetic patches for congenital diaphragmatic hernia repair: Surgisis vs Gore-Tex. J Pediatr Surg 2006;41:29-33
11. Mitchell IC, et al. Permacol: a potential biologic patch alternative in congenital diaphragmatic hernia repair. J Pediatr Surg. 2008;43:2161-4
12. Gourlay DM, Cassidy LD, Sato TT, Lal DR, Arca MJ. Beyond feasibility: a comparison of newborns undergoing thoracoscopic and open repair of congenital diaphragmatic hernias. J Pediatr Surg. 2009;44:1702-7
13. Petter-Puchner AH, et al. Adverse effects associated with the use of porcine cross-linked collagen implants in an experimental model of incisional hernia repair. J Surg Res. 2008;145:105-10
14. Petter-Puchner AH, et al. Adverse effects of porcine small intestine submucosa implants in experimental ventral hernia repair. Surg Endosc. 2006 Jun;20(6):942-6

Figure 1. Preoperative chest radiographs of all 3 patients demonstrating a left-sided CDH in each case.

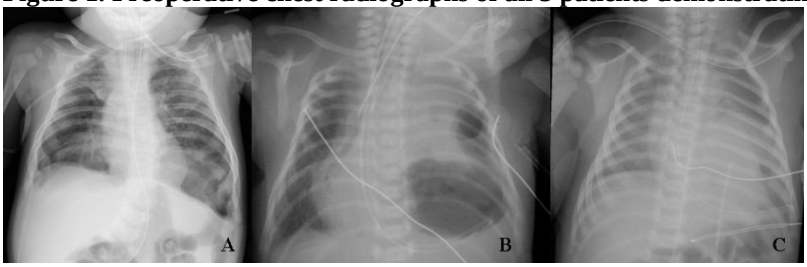


Figure 2. CT of patient #1 confirming intestine and left kidney in the left hemithorax.

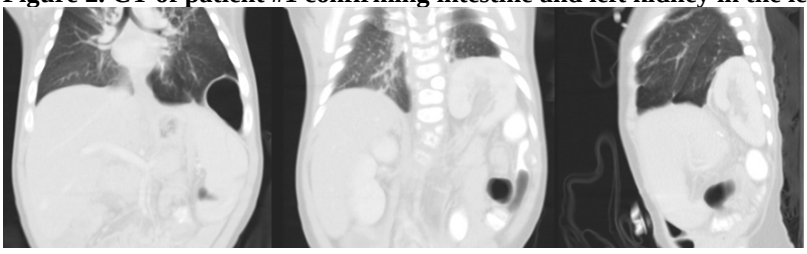


Figure 3. Intraoperative images in patient #1. A. Adhesions/viscera in the left hemithorax. B. Herniated viscera bulging into left hemithorax. C. Reduction of the herniated viscera. D. Primary closure of the medial aspect of the hernia. E. XenMatrix™ patch being sutured. F. Completed repair using XenMatrix™ patch.

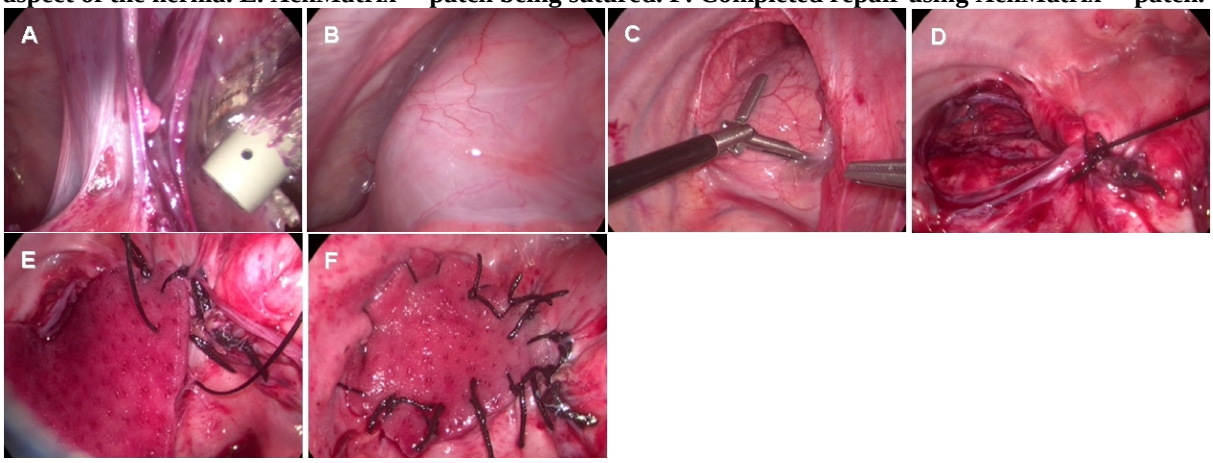
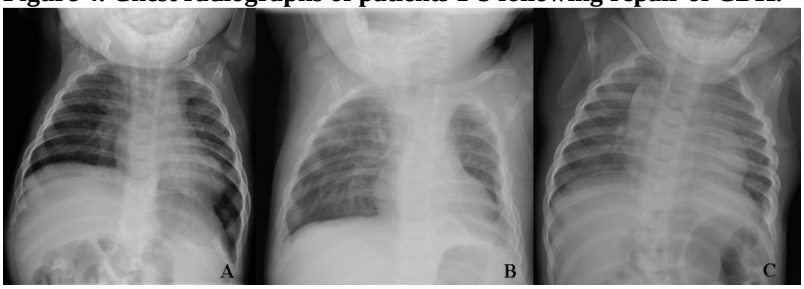


Figure 4. Chest radiographs of patients 1-3 following repair of CDH.



Article: Allograft Nerve Reconstruction for Digital Nerve Loss

Nimit Patel, MD*, Nirav Amin, MD*, Lucy McCabe**, John Taras, MD*
*Drexel University College of Medicine: Department of Orthopaedic Surgery
**Philadelphia Hand Center

ABSTRACT

Purpose: Reliable reconstruction of nerve gaps in the hand and digits remains a challenge to the hand surgeon despite the availability of graft options suitable to repair the deficit. This prospective study was designed to analyze the outcomes of digital nerve gaps in the hand measuring 30 mm or less reconstructed with processed nerve allograft.

Methods: Seventeen patients with 21 digital nerve lacerations in the hand underwent digital nerve reconstruction with processed nerve allograft. Outcome data for 14 patients with 18 digital nerve lacerations was available for analysis. The average nerve gap measured 11 mm (range 5 - 30 mm). Outcome measures included postoperative sensory examination as assessed by Semmes-Weinstein monofilaments and static- and moving-2 point discrimination. Pain was graded using a visual analog scale. In addition, patients completed the QuickDASH survey pre- and post-operatively to qualify their pain perception and functional impairment.

Results: Seven of 18 (39%) digits had an excellent result, 7 of 18 (39%) had good results, 4 of 18 (22%) digits had fair results, and 0 of 18 (0%) had poor results. Initial QuickDASH scores recorded at the patient's first postoperative visit averaged 44.8 (range 2.3-79.5), and final QuickDASH scores averaged 26.1 (range 2.3-43.2). There were no signs of infection, extrusion, or graft reaction.

Conclusions: The data suggest that processed nerve allograft provides a safe and effective alternative for the reconstruction of peripheral sensory nerve deficits in the hand measuring up to 30 mm.

INTRODUCTION

Restoration of peripheral nerve function continues to challenge surgeons despite advances in manufactured and traditional graft options and refined surgical techniques.[1-4] Segmental nerve loss often accompanies traumatic injury of the hand and digits. A tension-free, end-to-end repair often is not possible. In many cases, traumatic nerve injury requires debridement of the cut nerve ends, which further compromises tension-free repair.[5] In cases in which a secure, tension-free repair is not possible, the most common recommendation has been to graft the defect with a segment of autologous nerve. Autografts provide a reliable structure and environment through which axons can regenerate across a deficit; however, disadvantages have been described, including increased anesthesia and operating times, higher facility costs, and donor site morbidity such as pain, scarring, neuroma formation, and sensory loss.[6-12] Autograft alternatives include artificial nerve conduits and allografts, which eliminate donor site morbidity. Several clinical studies have documented the effectiveness of nerve conduits for short gaps;[13] however, the clinical value of nerve conduits to bridge larger gaps remains unclear.[13,14] Recent advances in allograft tissue processing have eliminated the need for immunosuppression, and allografts are becoming an attractive option.[6-9] Several authors have reported success with processed nerve allografts.[1,3,17-19] One such allograft to gain recent attention is the Avance® processed nerve graft (AxoGen, Inc., Alachua, FL). This type of graft consists of human nerve that has been decellularized, gamma irradiated, and subjected to enzymatic degradation of chondroitin sulfate proteoglycans (CSPG) with chondroitinase. As a result, the allograft maintains its 3-dimensional structure while remaining nonimmunogenic.[18,20] Studies in animal models have shown this material to mimic autografts in macrostructure, 3-dimensional microstructural scaffolding, and the protein composition inherent to nerve tissue. Theoretically, these qualities render allograft an effective material to span peripheral nerve defects.[1,17,19] A recent review of the literature however, disclosed few clinical studies evaluating the efficacy of processed allografts to reconstruct digital nerve defects. In a retrospective review, Karabekmez et al. reported on the efficacy of processed allografts for restoring adequate sensation in nerve defects ranging from 5 to 30 mm, and Cho et al reported on a subset of digital nerve defects up to 40 mm from an ongoing nerve registry. The goal of this prospective study was to evaluate the clinical and functional outcomes of digital nerve gaps in the hand measuring 30 mm or less reconstructed with processed nerve allograft.

METHODS

After receiving institutional review board approval, a prospective study was conducted on patients older than 18 years of age with digital nerve lacerations felt not amenable to primary repair (Table 1). Nerve reconstruction was performed using commercially available process nerve allograft (Avance® Nerve Graft, AxoGen, Inc., Alachua, FL) When a nerve laceration was identified during surgery, the gap between the debrided nerve ends was measured with the hand and digits in a neutral posture (Fig 1). If the gap was gauged to be 5 mm or greater, then a nerve allograft was used to reconstruct the defect. The injured nerve's diameter was measured and observed to be 2-3 mm for the proper digital nerves in this series. Each process nerve allograft was prepared following the packaged instructions for use. Under loupe magnification, the allograft was trimmed to match the defect, and 8-0 nylon suture was used to secure the graft junctures. Typically, 3 simple sutures secured each juncture (Fig 2). After wound closure, a sterile dressing was applied, and a plaster splint was fabricated to maintain the hand in a protected posture.

Seventeen patients with 21 digital nerve reconstructions met this study's inclusion criteria. All surgeries were performed by one hand surgeon. An independent observer collected the outcome data. Primary outcome measures included postoperative sensory examination using Semmes-Weinstein monofilaments, and static- (S2PD) and moving- (M2PD) 2-point discrimination using the Disk-Criminator (Sensory Management Services LLC, Lutherville, MD). Patients completed the QuickDASH (Disabilities of the Arm, Shoulder, and Hand)[21] survey pre- and postoperatively to qualify their pain perception and functional impairment. In addition, pain was recorded on a 10-point visual analog scale, where a score of 0 denoted no pain, and a score of 10 signified extreme pain. Secondary outcome measures included patient demographics, comorbidities, hand dominance, and location and mechanism of injury. Postoperative complications were reported.

RESULTS

Seventeen patients with 21 digital nerve lacerations underwent digital nerve allograft reconstruction. Three patients did not complete the study's 12-month minimum follow-up requirement. As a result, the study cohort included 14 patients with 18 digital nerve reconstructions. Ten men and 4 women with an average age of 39 years (range 18–76

years) returned for follow-up examination and outcome reporting through at least 12 months after surgery. Eleven injuries involved the dominant hand, and 7 injuries affected the non-dominant hand. Mechanisms of injury included a saw or power saw (7), knife (2), tape measure/sheet metal (3), glass (3), ceramic (2), and steel cable (1). There were concomitant fractures of the involved digit in 7 patients and 9 tendon lacerations in the involved digit in 7 patients. The average interval from injury to surgery was 29 days (range 2–262 days). The average nerve defect measured 11 mm in length (range 5 - 30 mm). The average time to final follow-up extended 15 months (range 12-20 months). At final follow-up at a minimum of 12 months postoperative, Semmes-Weinstein monofilament testing averaged 3.78 (range 2.83 – 6.65). Average S2PD results were 7.11 mm (range 5-8 mm), and average M2PD results were 5.44 mm (range 2-8 mm). Results were graded according to the scale reported by Taras.[13] According to this scale, 7 of 18 (39%) digits had an excellent result, 7 of 18 (39%) had good results, 4 of 18 (22%) digits had fair results, and 0/18 (0%) had poor results. Initial QuickDASH scores recorded at the patient's first postoperative visit averaged 44.8 (range 2.3-79.5) and final QuickDASH scores averaged 26.1 (range 2.3-43.2). There were no signs of infection, extrusion, or graft reaction. Using the visual analog scale, pain averaged 2 at final follow-up compared to the average initial pain score of 5. One patient underwent additional surgery for a 2-stage tendon reconstruction.

DISCUSSION

Despite the options available to reconstruct nerve defects, treatment of peripheral nerve gap injuries remains challenging. Traditionally, nerve autografts have been advocated to reconstruct peripheral nerve defects not amenable to primary repair, but donor site comorbidities, increased operative times, and limited harvest sites have presented considerable limitations.[6-10] Early results of processed allografts are encouraging and provide an attractive alternative to autografts.[1,17,19] Manufactured grafts alleviate donor site morbidity issues while retaining the elements that promote cell migration into the graft.[6,7] Published studies have presented alternatives to autografts for nerve defects extending up to 20 mm. In 2000, Weber et al. conducted a randomized, controlled study to examine the outcomes of nerve conduits compared with direct suture and autograft for sensory digital nerve repairs. For nerve gaps 4 mm or less, they reported mean M2PD 3.7 +/- 1.4 mm for conduit repair and 6.1 +/- 3.3 mm for end-to-end repairs ($p = 0.03$). The mean M2PD for nerve gaps greater than 8 mm for conduits was 6.8 +/- 3.8 mm.[18] Taras et al. also demonstrated the efficacy of purified type 1 bovine collagen conduits in a prospective study of 22 isolated digital nerve lacerations at a mean gap of 12 mm. They reported mean M2PD and S2PD of 5.0 and 5.2 mm, respectively, for digits with measurable recovery at the mean final follow-up 20 months (range, 12-59 months) after surgery; however, 6 nerve repairs failed to recover S2PD and were excluded from the mean analysis of S2PD. According to the grading criteria used in the Taras study, 13 of 22 (59%) digits achieved excellent results, 3 of 22 (14%) digits had good results, 6 of 22 (27%) digits had fair results, and there were no (0%) poor results. Rinker et al. showed no difference between polyglycolic nerve conduits and autogenous vein grafts for repair of peripheral nerve defects at a mean gap of 10 mm. A Multicenter Retrospective Study of Avance® Nerve Graft Utilization, Evaluations and Outcomes in Peripheral Nerve Injury Repair (RANGER) represents the largest retrospective review of processed allografts to date. The multicenter, multisurgeon review showed that use of processed allografts is a reliable method to restore nerve function across nerve gaps 5-50 mm in length.[1] The current study presents the results of a prospective series demonstrating the clinical efficacy of processed nerve allografts to repair peripheral nerve defects in the digits measuring up to 30 mm (average 11 mm) in length. The results of the current study are similar to others in the literature[1,3,6,7,12,13,16-18,20,21] but are expanded to include the patient's perspective of their functional limitations. In the current study, postoperative QuickDASH scores improved by an average of 18 points, representing a 42% reduction from preoperative scores. A limitation of the QuickDASH rating instrument is that the score reflects concomitant injuries, which in many cases accounted for a greater degree of disability than did the nerve injury. No postoperative complications such as infection or graft rejection occurred. One patient underwent an additional surgery for a 2-stage tendon reconstruction, which was unrelated to and did not impact the nerve reconstruction with allograft. Debate exists about the best material and technique to repair digital nerve defects. One advantage of allografts compared to nerve conduits is the similarity between allografts and autografts in terms of 3-dimensional macro- and microstructure. Whitlock et al. compared Avance® processed allografts to type 1 collagen conduits and isografts (autograft) in a rat model. They demonstrated that processed allografts are superior to conduits for short (14 mm) and long (28 mm) nerve gaps.[4] Nerve conduit provides a hollow macrostructure and allows for collection of fibrin within the inner chamber to serve as the scaffold for axonal regeneration towards the distal stump; yet it lacks the internal microstructure and extracellular matrix of native nerve tissue. Whitlock et al. suggest that the basal laminal internal structure retained by advances in allograft processing enhances its nerve regeneration potential. A subsequent study quantified the nerve fiber density between allograft, nerve conduits, and isograft. Nerve conduits exhibited a consistent decrease in midgraft density compared to allografts and isografts for both short and long graft models. In addition, the nerve fibers were distributed in an unorganized pattern for

conduits, whereas allografts and isografts demonstrated evenly distributed nerve fiber regeneration.[20] Limitations of the current study include small sample size and short-term follow-up. Future investigators should design prospective randomized studies to compare allografts to nerve conduits. In addition, the performance of nerve allografts to bridge motor nerve gaps longer than 20 mm would further delineate the indications for this type of graft. In conclusion, this prospective study demonstrates the efficacy of processed nerve allografts for the treatment of peripheral sensory nerve injuries up to 30 mm in length. Processed nerve allografts provide a reliable off-the-shelf alternative to autografts and nerve conduits.

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REFERENCES

1. Brooks DN, et al. Processed nerve allografts for peripheral nerve reconstruction: a multicenter study of utilization and outcomes in sensory, mixed, and motor nerve reconstructions. Microsurgery 2012;32(1):1-14
2. Ducic I, Fu R, Iorio ML. Innovative treatment of peripheral nerve injuries: combined reconstructive concepts. Ann Plast Surg 2012;68(2):180-187
3. Ijpma FF, Nicolai JP, Meek MF. Sural nerve donor-site morbidity - Thirty-four years of follow-up. Ann Plas Surg 2006;57:391–395
4. Whitlock EL, et al. Processed allografts and type I collagen conduits for repair of peripheral nerve gaps. Muscle Nerve 2009;39:787–799
5. Weber RA, Breidenbach WC, Brown RE, Jabaley ME, Mass DP. A randomized prospective study of polyglycolic acid conduits for digital nerve reconstruction in humans. Plast Reconstr Surg 2000;106(5):1036–1045
6. Hudson TW, Liu SY, Schmidt CE. Engineering an improved acellular nerve graft via optimized chemical processing. Tissue Eng 2004;10:1346–1358
7. Hudson TW, et al. Optimized acellular nerve graft is immunologically tolerated and supports regeneration. Tissue Eng 2004;10(11-12):1641-1651
8. Mackinnon SE, Doolabh VB, Novak CB, Trulock EP. Clinical outcome following nerve allograft transplantation. Plast Reconstr Surg 2001;107(6):1419–1429
9. Rinker B, Liau JY. A prospective randomized study comparing woven polyglycolic acid and autogenous vein conduits for reconstruction of digital nerve gaps. J Hand Surg Am 2011;36(5):775-781
10. Strauch B. Use of nerve conduits in peripheral nerve repair. Hand Clin 2000;16:123–130
11. Rappaport WD, et al. Clinical utilization and complications of sural nerve biopsy.Am J Surg1993;166(3):252–56
12. Staniforth P, Fisher TR. The effects of sural nerve excision in autogenous nerve grafting. Hand 1978;10(2):187–190
13. Taras JS, Jacoby SM, Lincoski CJ. Reconstruction of digital nerves with collagen conduits. J Hand Surg Am 2011;36(9):1441-1446
14. Neubauer D, Graham JB, Muir D. Chondroitinase treatment increases the effective length of acellular nerve grafts. Exp Neurol 2007;207:163–170
15. Meek MF, Coert JH. Clinical use of nerve conduits in peripheral nerve repair: review of the literature. J Reconstr Microsurg 2002;18:97–109
16. Noble J, Munro CA, Prasad VS, Midha R. Analysis of upper and lower extremity peripheral nerve injuries in a population of patients with multiple injuries. J Trauma 1998; 45:116-122
17. Karabekmez FE, Duymaz A, Moran SL. Early clinical outcomes with the use of decellularized nerve allograft for repair of sensory defects within the hand. Hand 2009;4(3):245-249
18. Lundborg G. A 25-year perspective of peripheral nerve surgery: evolving neuro scientific concepts and clinical significance. J Hand Surg Am 2000;25(3):391–414
19. Cho MS, et al. Functional outcomes following nerve repair in the upper extremity using processed nerve allograft. J Hand Surg 2012;37A:2340-2349
20. Johnson PJ, Newton P, Hunter DA, Mackinnon SE. Nerve endoneurial microstructure facilitates uniform distribution of regenerative fibers: a post hoc comparison of midgraft nerve fiber densities. J Reconstr Microsurg 2011;27(2):83-90
21. Hudak PL, Amadio PC, Bombardier C. Development of an upper arm extremity outcome measure: the DASH (disabilities of the arm, shoulder, and hand) [corrected] The Upper Extremity Collaborative Group (UECG) Am J Ind Med 1996;29(6):602-608. Erratum in: Am J Ind Med 1996;30(3):372

Table 1. Nerve Repair Patient Data

Patient	Age	Gender	Dom Side	Affected Side	Mech of Inj	Digit	Nerve Injured	Gap Length (mm)	Final Follow-Up (mos)	S2PD	M2PD	SW	Initial Pain	Final Pain	Initial QuickDASH Score	Final QuickDASH Score	Difference in QuickDASH	Graded Results
1	25	Female	R	R	Ceramic	Thumb	RDN	10	25.3	8	4	3.22	5	1	27.3	2.3	25.0	E
2	25	Female	R	R	Ceramic	Thumb	UDN	10	25.3	8	4	3.22	5	1	27.3	2.3	25.0	E
3	31	Female	R	L	Knife	Small	UDN	10	14.8	5	4	2.63	1	1	2.3	2.3	0.0	E
4	19	Male	R	L	Circular	Thumb	UDN	10	12.0	7	5	3.22	2	1	65.9	43.2	22.7	G
5	69	Male	R	R	Circular	Small	UDN	6	12.4	6	4	2.36	3	1	29.5	22.7	6.8	E
6	39	Male	R	R	Saw	Long	UDN	17	23.4	8	8	6.65	5	8	79.5	50.0	29.5	F
7	29	Male	R	L	Circular	Small	UDN	28	12.6	8	8	3.84	7	1	34.1	11.4	22.7	F
8	54	Male	R	R	Sheet	Index	RDN	10	16.2	8	6	3.84	3	3	56.8	43.2	13.6	G
9	57	Male	R	L	Table	Thumb	RDN	10	12.0	6	4	3.22	3	1	43.2	36.4	6.8	E
10	57	Male	R	L	Table	Thumb	UDN	10	12.0	6	4	3.22	3	1	43.2	36.4	6.8	E
11	22	Female	R	R	Glass	Thumb	RDN	10	11.9	8	5	3.61	4	2	61.4	38.6	22.8	G
12	22	Female	R	R	Glass	Thumb	UDN	10	11.9	6	5	3.61	4	2	61.4	38.6	22.8	G
13	29	Male	R	L	Knife	Index	RDN	5	11.7	5	2	2.83	8	1	11.4	6.8	4.6	E
14	18	Male	R	L	Glass	Ring	RDN	12	15.0	7	8	6.65	10	1	65.9	18.2	47.7	F
15	57	Male	R	R	Tape	Index	RDN	7	17.4	8	7	3.22	5	2	61.4	34.1	27.3	G
16	57	Male	R	R	Tape	Long	RDN	5	17.4	8	7	3.22	5	2	61.4	34.1	27.3	G
17	24	Male	R	R	Steel	Index	UDN	30	11.5	8	8	6.65	4	4	29.5	27.3	2.2	F
18	76	Female	R	R	Glass	Small	UDN	5	11.6	8	5	2.83	5	1	44.5	26	18.5	G
Avg	**39**							**11**	**15.3**	**7**	**5**	**3.78**	**5**	**2**	**44.8**	**26.3**	**18.4**	

Figure 1. Measurement of gapped ends

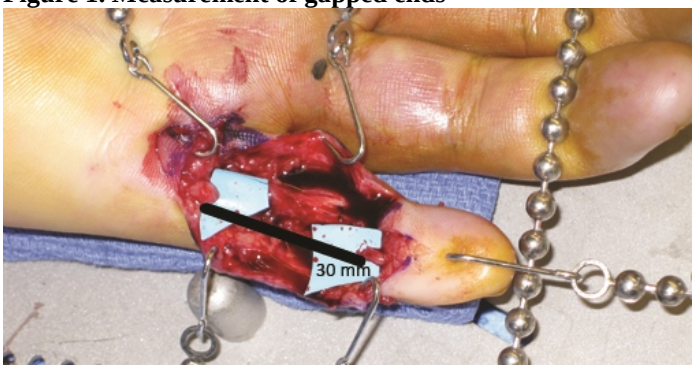
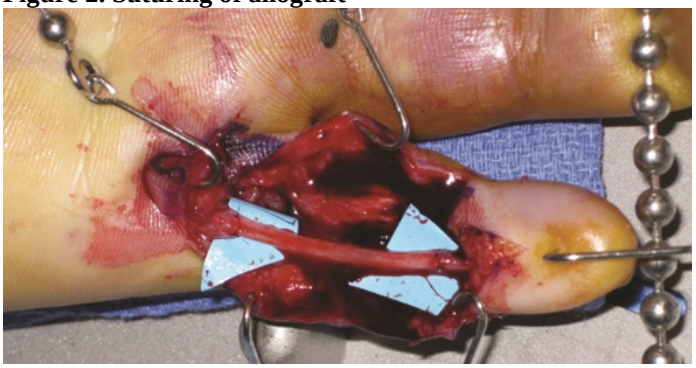


Figure 2. Suturing of allograft



Article: Extraction of a Retained Rectal Foreign Body: Case Series and Literature Review

Alexey Markelov, MD*, Omkar Baxi**, Robert Bloch, MD*, Eric Liedtke, DO*
*Easton Hospital: Department of Surgery
**Drexel College of Medicine: Department of Surgery

ABSTRACT
Rectal foreign bodies present a difficult diagnostic and management dilemma because of delayed presentation, a variety of objects, and a wide spectrum of injuries. The first goal of the therapy is extraction of the foreign body

using the simplest method possible while protecting the integrity of the colon.[1] Many instruments and methods have been suggested for this purpose.[2] We present three cases of trans-anal extraction of a foreign body from the rectum using unconventional techniques along with a review of literature.

CASE REPORT

Case 1: A 46 year-old man presented to the emergency department six hours after insertion of a vibrator device into his rectum. A plain abdominal radiograph (Fig 1) showed the container just above the rectosigmoid region. There were no signs of perforation. The vibrator could not be extracted by bimanual manipulation. A subsequent attempt to remove the object with conventional endoscopic instruments such as polypectomy snares was unsuccessful. As a result, the patient was given general anesthesia with muscle relaxation and retractors were placed in the rectum. The foreign body was visualized and evacuated using sponge forceps.

Case 2: A 15 year-old male presented to the emergency department after being unable to retrieve a device he had inserted into his rectum. He complained of lower abdominal pain and slight bleeding per rectum but signs and symptoms of mechanical intestinal obstruction and colorectal perforation were absent. The patient underwent a flat plate KUB of his abdomen, which demonstrated a distinct foreign body within his rectosigmoid area (Fig 2). Digital rectal exam was performed and the rectal foreign body was palpated. The foreign body was exposed with Sims retractors, grasped with ring forceps, retracted into the rectum and then grasped firmly with Kocher forceps and removed from the rectum.

Case 3: A 45 year-old man presented to the hospital with a five day history of a foreign object in the rectum. There was no perineal trauma on inspection. Xray is shown in Figure 3. Attempts to retrieve the device without anesthetic use failed, so the patient was transferred to the operating room and placed under general anesthesia. Flexible sigmoidoscopy was performed and the object was grasped using polypectomy snares. After the object was pulled down with the snares, it was grasped using Rochester forceps which were passed alongside the sigmoidoscope and cautiously withdrawn from patient's rectum.

DISCUSSION

Rectal foreign bodies often pose a challenging diagnostic and management dilemma that begins with the initial evaluation in the emergency department and continues through the post-extraction period. Numerous objects ranging from billy clubs, varied fruits and vegetables, nails, light bulbs, and turkey basters to a propane tank have been described as retained rectal foreign bodies. Because of the wide variety of objects and the variable trauma that can be caused to the local tissues of the rectum and distal colon, a systematic approach to the diagnosis and management of the retained rectal foreign body is essential (Fig 4). A large number of surgical and non-surgical techniques have been described to remove rectal foreign bodies. Techniques for the safe extraction of a rectal foreign body frequently require ingenuity.[3,4] The technique chosen usually depends on the size, shape, and contour of the foreign body. Our case series illustrates that one of the most important factors for successful extraction of foreign bodies retained in the rectosigmoid junction is patient relaxation. This can be achieved with a perianal nerve block, a spinal anesthetic, or either of these in combination with intravenous conscious sedation. All of these techniques allow the patient to relax, decrease anal tone, sphincter spasm, and improve visualization and exposure.

REFERENCES

1. Volpi A, et al. Colorectal retained foreign bodies per anum introduced. Three years retrospective study at Emergency Surgery Unit. G Chir. 2012;33(11-12):411-4
2. Ayantunde AA. Approach to the diagnosis and management of retained rectal foreign bodies: clinical update. Tech Coloproctol. 2012. epub ahead of print
3. Codd RJ, Havard TJ. A novel technique for the per-anal extraction of spherical rectal foreign bodies. Ann R Coll Surg Engl. 2012;94(6):439
4. Koornstra J, Weersma R. Management of rectal foreign bodies: Description of a new technique and clinical practice guidelines. World J Gastroeneterol. 2008;14(27):4403-6

Figure 1. Case 1 X-ray

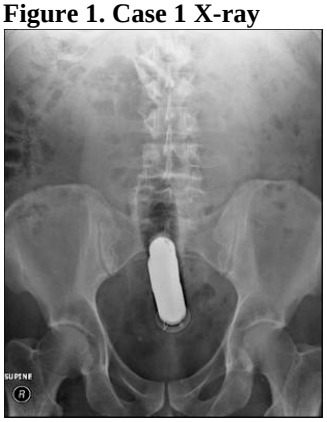


Figure 2. Case 2 X-ray

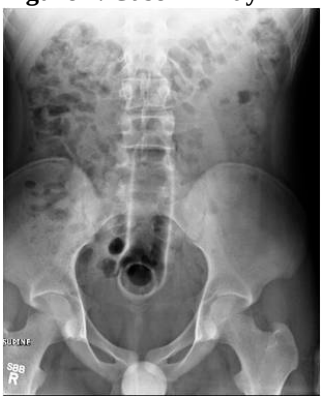


Figure 3. Case 3 X-ray

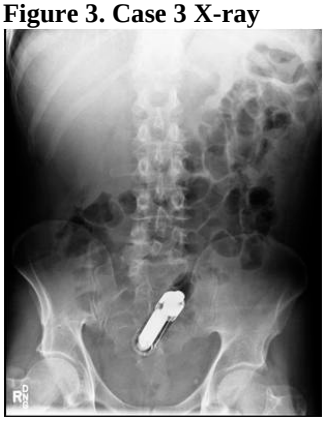
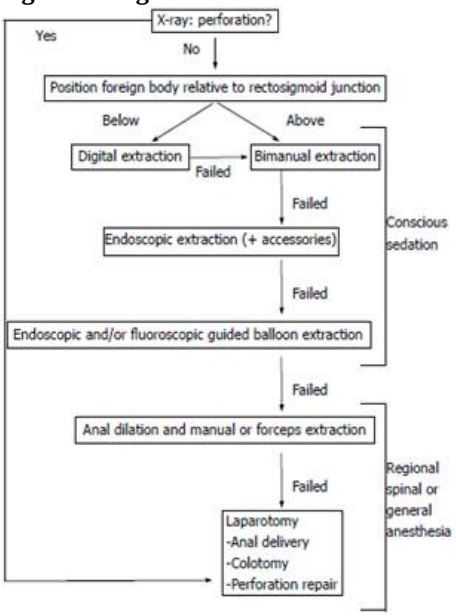


Figure 4: Algorithm for removal of rectal foreign body as suggested by Koornstra and Weersma[4]



Article: Factors Influencing Readmissions to Medicine Services at Monmouth Medical Center in 2011

Pawan Arora, MD, Harshil Bhatt, MD, Syed Sameeruddin, MD, Sreedevi Sodanapalli, MD, Sheeba Mushtaq, MD, Golda Fernandez, MD, Sara Wallach, MD
Monmouth Medical Center: Department of Medicine

INTRODUCTION

Healthcare costs are imposing an increasing burden on the United States' federal budget. Medicare spending has increased exponentially over the years and is expected to increase to $234.9 billion by 2019.[1] In April 2009, a report published in New England Journal of Medicine stated that 19.6% of Medicare fee-for-service beneficiaries were readmitted to the hospital within 30 days of discharge, 34% within 90 days and more than 51% within one year.[2] The Medicare Payment Advisory Commission asserted that readmission accounted for $15 billion of Medicare spending and found that 17.6% of hospital admissions resulted in readmissions within 30 days of discharge, 11.3% within 15 days, and 6.2% within 7 days.[3] There has been much debate concerning the etiology of these readmissions. In October 2012, Medicare will decrease the payments to the hospitals for certain diagnoses with large numbers of readmissions, regardless of the cause of the readmission.

The same report published in NEJM in 2009 also pointed out that readmission varies substantially by hospitals and by geographic areas, even after considering the disease type and severity of illness. The study also found higher readmission rates for some states such as New Jersey (21.9%) and Illinois (21.7%), and lower readmission rates for other states like Utah (14.2%).[2] Relatively high readmission rates are found for Medicare beneficiaries with multiple chronic illnesses. In a meta-analysis of 44 studies, the mean readmission rate was 34% for patients with chronic illnesses.[4] According to another study, those patients with five or more medically comorbid conditions had more than twice the likelihood of an unplanned readmission within 30 days than patients without those conditions.[5] Since then, many hospitals have implemented performance improvement projects to reduce readmission rates for various diseases such as congestive heart failure and chronic obstructive lung disease. The purpose of these performance improvement efforts is to prevent unnecessary patient readmissions that may lead to increased length of stay and in hospital complications. A corollary of this is that hospitals can avoid Medicare cuts by implementing these projects.

The purpose of this study is to retrospectively examine and categorize readmissions to the medicine service at Monmouth Medical Center. Examining the factors that lead to readmissions will enable our performance improvement multidisciplinary teams to develop projects to reduce our readmission rate.

METHODS

Our study is a retrospective descriptive study which will quantify and examine readmission rates to medicine services at Monmouth Medical Center, and determine the factors leading to patient readmissions. We examined 365 of 866 medical records of medicine patients who were admitted to the medical service from January 2011 through December 2011. Medical records were deemed suitable for review if the patients were over 18 years of age and readmitted within 28 days from a primary admission. The records were reviewed for the following factors: age, gender, ethnicity, diagnosis on both the primary admission and readmission, primary reason for admission addressed, disposition of patient, living status and functionality of the patient, mental status of the patient, medical errors committed during the readmission, chronic illnesses, and outpatient follow-up. As this is a chart review under the auspices of our medicine performance improvement committee, an institutional review board waiver was granted for this project.

RESULTS

Out of 866 charts, 365 charts were reviewed in total comparing above-mentioned risk factors. 41% of patients who were admitted within 28 days of discharge were more than 75 years of age. Also 54 % of patients were females who were readmitted. 50% of patients were discharged to a care facility from the first admission. See Tables.

DISCUSSION

In Monmouth Medical center as per information available from medical records in 2011 out of 7000 admissions we have 865 patients readmitted which gives a readmission rate of 12.3 % within 28 days of discharge. This appears to be a lower rate of readmission compared to data released from MedPACs 2008 report of 2005 data which was 17.6% at 30 days. Out of these the readmission rate was 33% within 7 days. From the data we analyzed 25% of these readmissions were preventable and 22.7 % were readmitted with same clinical diagnosis. When we do an organ-system based analysis of these readmissions in which clinical diagnosis was the same, we found that patients with nephrologic and gastroenterological diagnosis had the highest readmission rate at 57% and 54% respectively. The data also revealed population above 75 years of age had a higher readmission rate within 28 days of discharge at 41% when compared to 12.3 % for the general population in Monmouth Medical center. The gender distribution in readmission was almost even with females accounting for 54% of readmissions.

We had few limitations to our study which include inability to access data if these patients were hospitalized in other hospitals. Only holders of all hospital discharge data such as Medicare can track patients across providers and systems and can help us better identify preventable reasons for readmissions. In addition, the risk factors analyzed for readmissions need to be compared with the control group of non-readmitted patients which we didn't have at the time we accumulated the data. We also think the co-morbidities affecting readmission need to be further analyzed on the basis of direct impact on readmissions.

Re-hospitalizations are a costly and sometimes life-threatening event and are used as a measure of performance of hospitals. The purpose of our study was to analyze patients being readmitted to our hospital and find risk factors that might have contributed towards recurrent admissions.

REFERENCES
1. Congressional Budget Office, "CBO's August 2010 Baseline: Medicare."
2. Jencks SF, Williams MV, Coleman EA. Rehospitalizations among Patients in the Medicare Fee-for-Service Program. N Engl J Med 2009; 360: 1418-1428
3. Medicare Payment Advisory Commission (MedPAC), Report to Congress: Promoting Greater Efficiency in Medicare, June 2007, Chapter 5
4. Karen L, et al. Predictors of Hospital Readmission: A Meta-Analysis. Evaluation & the Health Professions. 1991;14(3):262-281
5. Marcantonio ER, et al. Factors Associated with Unplanned Hospital Readmission Among Patients 65 years of Age and Older in a Medicare Managed Care Plan. Am Journal of Medicine. 1999;107(1):13-17

Table 1. Readmission Patient Data

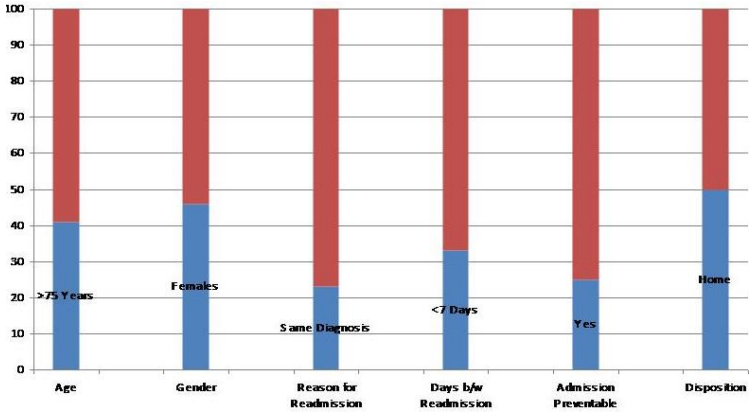


Table 2. Readmissions Involving Same Admitting Diagnosis with Organ System Distribution

Organ System	Total Cases	Preventable Readmission	Percentage of Preventable Readmission(%)
Pulmonary	15	4	26
Cardiovascular	15	7	47
Nephrology	7	4	57
Infectious Diseases	10	3	30
Gastroenterology	13	7	54
Endocrinology	2	0	0
Hematology/Oncology	7	3	43
Neurology	4	1	25
Musculoskeletal	2	0	0
Substance Abuse	8	4	50
Total	**83**	**33**	**39**

Article: Testosterone Replacement Therapy in Hypogonadal Men after Successful Treatment of Prostate Cancer

Michael Kraft, MD*, Bruce Sloane, MD**
*Drexel University College of Medicine: Department of Family, Community, and Preventive Medicine
**Philadelphia Urology Associates

INTRODUCTION
There has been a significant increase in the treatment of low testosterone (T) over the past decade. In fact there is a large body of evidence supporting the use of testosterone replacement therapy (TRT) and the benefits of restoring normal testosterone levels in men with T deficiency. T has been known to have cardiovascular benefits as well as benefits on cardiovascular risk factors. T has been shown to improve blood flow in men with CAD, lower

triglycerides, total cholesterol, low-density lipoprotein (LDL), fibrinogen, and plasminogen activator–1. T has also been shown to positively affect the factors making up metabolic syndrome including decrease body mass index (BMI), waist circumference, waist/hip ratio, amount of visceral fat, serum leptin levels, serum insulin levels, and serum free fatty acid concentrations.[1,2]

In spite of these benefits, however, physicians still have significant concerns about the safety of TRT and its possible adverse effects. This is especially true with regard to the androgen sensitive prostate gland where there has been, up until recently, an unquestioned belief that increasing T levels would cause the rapid growth of prostate cancer. This belief came about by the work of Charles Huggins, MD, et al.[3,4] In 1941, Dr. Huggins and colleagues observed that in prostate cancer there is an androgen dependent nature to the process and how reducing levels of T to castrate levels will regress the prostate cancer. However, this observation was in a single patient and has subsequently become the standard of treatment in prostate cancer, even to this day. Since 1941, several studies have proposed a different model for interaction between T and prostate cancer. These studies and hypothesis suggest a saturation model for T within the prostate.

There is good evidence for this saturation model, two studies that come to mind. The first being Behre and colleagues, which showed T levels greater than this saturation point within the prostate did not show additional growth. Administering TRT in hypogonadal men completed this, this treatment caused the hypogonadal men to have a volume increase in the prostate but only up to the size of age-matched eugonadal men controls.[5] The other study is by Bhasin and colleagues showing that within a group of healthy men there was no change in PSA or prostate volume with supraphysiologic doses of T.[6] The saturation model is further supported by Marks in a landmark article published in JAMA. This article showed that levels of T and DHT, as well as markers of cellular proliferation, did not change within the prostate with TRT. This was accomplished by determining intraprostatic levels of T and DHT measured before and after 6 months of TRT. Within the treatment arm of the study serum T levels rose substantially, however, intraprostatic levels of T and DHT did not change much as compared to the placebo arm of treatment. This was also seen with markers of cellular proliferation.[7] The results of this study are two fold; changes in serum androgen levels in hypogonadal men are not reflected within the prostate and that the prostate seems to be able to create a homeostatic environment in regards to androgens.[8]

So the question becomes is it safe to treat hypogonadal men with a history of successfully treated prostate cancer with TRT. Several published reports have shown that this treatment is safe and no ill effects were experienced with TRT in hypogonadal men after being treated for prostate cancer. Two small series have shown that in men with an undetectable PSA level after radical prostatectomy there were no recurrences of prostate cancer for up to 12 years.[9,10] This has also been shown in a more recent study involving men who had prostate cancer and received brachytherapy. In this study 31 hypogonadal men successfully treated with brachytherapy for prostate cancer received TRT and were followed for a mean 4.5 years. It showed that 100% of the subjects maintained a PSA level <1.0ng/mL.[11]

METHODS

With these reports in mind we began instituting T replacement in our patients with prostate cancer beginning in 2005. Patients had to meet the following criteria to be started on T replacement: Total T <350ng/L or Free T< 9ng/dL . In men treated with radical prostatectomy, serum PSA had to be undetectable for at least one year and no adjuvant therapy for positive margins or recurrent disease was given. Men who underwent radiation therapy (any form) with or without androgen deprivation therapy had to evidence stable PSA values for at least 2 years with a nadir value <1.0 ng/ml and have completed their androgen deprivation therapy for at least 6 months.

RESULTS

We have similar results within patients that have been successfully treated for prostate cancer, using various modalities, and treated with TRT. In a chart review of several patients 8 were selected meeting the following criteria: known Gleason score prior to treatment, known type of prostate cancer treatment, PSA prior to treatment, PSA post treatment, history of or current TRT, type of TRT, PSA prior to TRT, PSA while on or post TRT. Within the selected patients they had a Gleason score ranging from 6-9. The types of treatment for prostate cancer included several modalities, radical prostatectomy only, XRT only, XRT with hormone therapy, and brachytherapy with hormone therapy. PSA prior to prostate cancer treatment ranged from 1.2ng/mL to 34.1ng/mL and post treatment PSA ranged from <0.1ng/mL to 0.2ng/mL. The type of TRT used was both topical and injection. In all patients PSA

while on or post TRT did not rise above 1 ng/mL. In one patient there were fluctuations within his PSA, however with continued monitoring he did return to his baseline of 0.2 ng/mL.

DISCUSSION

In conclusion, through observations in our patients and the aforementioned studies, it does appears safe to institute TRT in hypogonadal men who exhibit evidence of successfully treated prostate cancer. However, larger studies and longer follow-up are needed to document the long-term safety of TRT in this patient population. Close follow of these patients with PSA and physical examination is warranted.

REFERENCES

1. Webb CM, McNeill JG, Hayward CS, deZeigler D, Collins P. Effects of testosterone on coronary vasomotor regulation in men with coronary heart disease. Circulation 1999;100:1690–1696
2. Shabsigh R, Katz M, Yan G, Makhsida N. Cardiovascular Issues in Hypogonadism and Testosterone Therapy, American Journal of Cardiology. 2005; 96(12), S2:Pages 67-72
3. Huggins C, Hodges CV. Studies on prostatic cancer. I. The effect of castration, of estrogen and of androgen injection on serum phosphatases in metastatic carcinoma of the prostate. Cancer Res 1941;1:293-297
4. Huggins C, Stevens RE, Hodges CV. Studies on prostatic cancer. II. The effects of castration on advanced carcinoma of the prostate gland. Arch Surg 1941;43:209-223
5. Behre HM, Bohmeyer J, Nieschlag E. Prostate volume in testosterone-treated and untreated hypogonadal men in comparison to age-matched normal controls. Clin Endocrinol 1994;40:341-349
6. Bhasin S, et al. Testosterone dose-response relationships in healthy young men. Am J Physiol Endocrinol Metab 2001;281:E1172-E1181
7. Marks LS, et al. Effect of testosterone replacement therapy on prostate tissue in men with late-onset hypogonadism: a randomized controlled trial. JAMA 2006;296:2351-2361
8) Morgentaler, A. Testosterone Replacement Therapy and Prostate Cancer. Urologic Clinics of North America 2007;34:555-563
9. Kaufman JM, Graydon RJ. Androgen replacement after curative radical prostatectomy for prostate cancer in hypogonadal men. J Urol 2004;172:920-922
10. Agarwal PK, Oefelein MG. Testosterone replacement therapy after primary treatment for prostate cancer. J Urol 2005;173:533-536
11. Sarosdy MF. Testosterone replacement for hypogonadism after treatment of early prostate cancer with brachytherapy. Cancer 2007;109:536-541

Article: The Salto Talaris Total Ankle Joint Replacement: A Review of 7 Cases at Two-Year Follow-up

Robert Rajczy, DPM, Steven Boc, DPM, Howard Shapiro, DPM
Drexel University College of Medicine: Podiatric Medicine and Surgery

INTRODUCTION

Treatment of ankle arthritis was transformed with the advent of the total ankle replacement in the 1970s.[3] Surgical treatment that preserves function and motion at the ankle joint presented an alternative to more traditional procedures, namely arthrodesis. Total ankle joint replacements themselves are now surpassing their infancy and have seen more extensive use in recent years. However, long-term effects and complications are still under review. Studies including Nunley and Bonnin have presented long term outcomes of a mean of 61 and 106 months respectively, with an implant survival rate of 65-93%.[3,9] The Salto Talaris (Tornier, Inc.) ankle prosthesis has an anatomic design composed of cobalt chromium alloy with an outer titanium coating with a polyethylene insert (Fig 1). The prosthesis consists of talar and tibial components. The geometry of the tibial component allows for maximized contact area with the talar component. With a specialized medial and lateral curvature, the talar component reproduces normal ankle kinematics and prevents overstressing of the deltoids.[12] The implant was developed for use in 1997 and is a treatment option for both osteoarthritis and post-traumatic arthritis in a range of ages from mid-twenties to early eighties.[4] This study investigates the outcome of the Salto Talaris implant at a minimum of two years in an attempt to evaluate patient satisfaction and function. Additionally, the study assesses radiographs for correction of deformities and implant stability.

METHODS

We retrospectively reviewed 16 patients who underwent ankle arthroplasty with Salto Talaris implant between November 2008 and October 2010. Each participating patient was verbally surveyed and a physical exam of the affected ankle was conducted. Indications for the procedure were osteoarthritis or post-traumatic arthritis. No patient had a diagnosis of rheumatoid arthritis. 16 patients were initially included however 8 were lost to follow-up and one had revisional arthrodesis. The remaining participants had a mean age of 48 (27 to 64 years old), with 6 females and one male. Past medical history included DM, HTN, hyperlipidemia, and one patient had hepatitis C. Pre-operative BMI was obtained, with a mean of 30.55 +/- 5.45 (Table 1). All patients underwent prior conservative treatments including bracing, therapy, and cortisone injection. Patients also underwent prior surgery including ankle open reduction internal fixation following trauma and surgical arthroscopy with debridement (Table 2). One patient underwent subtalar joint arthrodesis after ankle arthroplasty surgery.

For the surgical procedure an anterior extensile incision is made between the tibialis anterior and extensor hallucis longus tendons followed by adequate soft tissue dissection with exposure of the ankle joint. Exostectomy of the talus and tibia are performed prior to alignment guide positioning. The alignment guide is prepped to neutral position followed by positioning the guide along the long axis of the tibial shaft by drilling pins in the tibial tuberosity and distal metaphysis. Adjustments can be made at this time to the frontal or sagital planes as well as height of the tibial component. The cutting guide is applied to the tibial component and an oscillating saw is utilized for the tibial resection with the ankle at 90 degrees with no rotational malalignment (Fig 2). The talus is prepped and resected with the same oscillating saw and the aid of the cutting guide and two inferior guide pins. With the aid of a talar chamfer, the bone surface is reemed and further lateral resection is performed. A trial implant is applied followed by the permanent implant once appropriately sized. The tibial trial component is then fitted and a tibial plug is drilled to fit the anterior tibial keel followed by the final implant (Fig 3). The patients are kept in a non-weight bearing below-knee cast for approximately 4 weeks until the skin incision heals. Active ROM is initiated at 4 weeks with transition to a cast boot followed by gradual weight bearing at 6 weeks. Potential complications of this procedure include wound dehiscence, chronic wound, infection, amputation, loosening of components, instability, bone fracture and neuritis/neuropathy.

The patients presenting for follow-up were both surveyed and clinically evaluated on the side of the ankle implant. Questions and physical exam were based on the American Orthopedic Foot and Ankle Society Ankle-Hindfoot Scale. The survey was conducted as an anonymous verbal questionnaire. Physical exam evaluated for ankle range of motion with dorsiflexion, plantar flexion and subtalar joint eversion and inversion. Patients were evaluated while weight bearing to assess foot and ankle alignment. Finally, patients were asked to rate their overall satisfaction compared to their condition prior to the surgery as “much better,” “better,” “no improvement” or “worse” and asked to focus on pain and activity level.

Pre-operative radiographs were evaluated utilizing the Kellgren-Lawrence score for evaluating osteoarthritis (Mean 2.5 with median of 3). The grading scale of 2 represents a joint with definitive narrowing and osteophytes. A grade of 3 includes a deforming bone contour and sclerosis, while 4 represents severe deformity of bone contour with large osteophytes (Fig 4). The anterior angle of the tibia was evaluated for deformities (Fig 5). The angle measured the long axis of the tibia with the transverse axis of the talar dome. Likewise the talo-calcaneal angle was measured to evaluate the talar component and any subsequent migration. These measurements were implemented by Bonnin in their evaluation of ankle implants.[3] A normal tibial angle is 90 degrees in AP and lateral radiographs while a talo-calcaneal angle greater than 5 degrees is significant in the previously mentioned study (Fig 6). Post-operatively, these same angles were measured again to evaluate for corrections of planar deformities. In addition, radiographs were assessed for loosening of the three implant components in addition to migration and osteolysis of the surrounding bone. Radiographic evaluation for recurrent spurring or bony overgrowth was also evaluated which could potentially result in jamming at the level of the joint.

Due to a small sample size of this study, statistical analysis would not facilitate meaningful statistical results. For this sample, percentages were utilized for categorical data while mean, standard deviation was utilized for numerical data (radiographic analysis). The results are an exploratory view of the clinical outcome.

RESULTS

The results of this study were separated into two categories: patient evaluation (subjective and objective findings) and radiographic findings. Patient evaluation via the AOFAS pain scale averaged 62.71. Average pain per patient

was 14.28/40. Upon further evaluation for patient satisfaction, only one patient had a response of "worse." Two patients required revisional surgery and one underwent subtalar joint arthrodesis resulting in a reduced AOFAS score upon the physical exam. The additional procedures for these individuals were exostectomy of bony osteophytes of the tibia and talus (Table 3). Surgical site dehiscence did not require further surgery or hospitalization for infection. Dehiscence was treated with local wound care until resolution, and soft tissue cellulitis was treated with oral antibiotics. Radiographically, the anterior tibial angle was evaluated before surgery, immediately post-op and on the last follow-up visit (Table 4). The mean angle on the most recent visit was 87.43 degrees with a median of 90 degrees and standard deviation of 5.26. This is compared with a mean angle of 89.48 and 88.74 degrees post-op and prior to surgery respectively. Lateral tibial angle was performed to assess migration of the tibial component post-operatively had a mean angle of 85.57 compared to most recent follow up of 80.87 degrees. The talo-calcaneal angle was 11.86 degrees at last follow-up and 14.43 degrees post-op. The talo-calcaneal angle mean differed only by 2.57 degrees on all ankles. All but one patient had a change less than or equal to 5 degrees which is significant for movement or loosening of the talar component. Osteolysis was noted in two (29%) patients, most notably around the tibial component. Osteophyte formation was also noted in three (43%) patients at most recent follow-up (Table 5).

DISCUSSION

The purpose of this study was to evaluate patients with a SaltoTalaris total ankle joint replacement with a minimum of two year follow-up. The small sample size will not reveal a statistically significant result but can be used as a window to portray patients who underwent this procedure and how they have progressed subjectively, clinically and radiographically. In terms of pain scale, no patient stated they were pain-free or experience infrequent discomfort. Two patients experience chronic pain while 4 others complain of pain on a daily basis. The AOFAS score of the two individuals who experience the most discomfort is the lowest while the other four patients are 15-20 points improved. In terms of satisfaction, only one patient stated they feel worse, while the other patients felt the implant has improved their ankle range of motion, activity level, and quality of life compared to before the surgery. One patient who had an AOFAS pain scale of 0/40 still felt the implant allowed them to have a better level of function than before the surgery. Therefore, from this sample one can deduce even though pain is a sequelae, this procedure can improve function to regain and enhance regular daily activities. Bonnin et al. followed-up 140 at mean 53.8 months found an average AOFAS score of 87.7.[4] Utilizing different implants, Anderson et al. followed-up 39 patients with a mean score of 74 points.[1] Morgan et al. followed up 108 patients at 61 months with a score of 85.9.[9] These studies demonstrate satisfactory outcomes with the use of these TARs with a greater population over a longer period of time. Serial radiographs during the post-operative course are crucial to evaluate the state of the implant in addition to the condition of the surrounding bone. Analyzing angles within the ankle joint and surrounding bone have been evaluated by the likes of Bonnin and Bestic.[2,3] In our study, radiographic angles were evaluated for ankle alignment as well as any potential migration or loosening of the ankle joint components. The AP tibial angle with a mean and median of 87 and 90 degrees respectively, demonstrate little to zero malalignment in the transverse or frontal planes. Lateral radiographs utilized to evaluate loosening of the components showed a small amount of migration, an average of 3.5 degree deviation by the most recent follow-up visit. Osteolysis around the components was also evaluated, which found two of the six as having mild degree of loosening around the implants. Despite these findings, there has been no need for revision or further surgery within this population. In fact, the implants with any evidence of loosening (Case 1 & 2) had a higher AOFAS score with favorable outcomes. Osteophyte formations around the implants were also observed in 3 of the seven patients. This observation was not mentioned in our literary review and clinically does not correlate to a decreased range of motion of jamming typically experienced with arthritic changes. Instead, it may coincide with an increase in pain and discomfort around the joint which can be demonstrated by the outcome of Case 3. Two patients (Case 4 & 7) who likewise complained of increasing pain, did elect to undergo revisional exostectomy of the tibia and talus. Each procedure was greater than one year after the initial surgery. It is merely hypothetical that these radiographic findings may be indicative of implant complication resulting in less patient satisfaction and this may be an area of further review, especially if additional surgery is warranted.

CONCLUSION

The total ankle arthroplasty with implant is a modern treatment for ankle arthrosis. While the literature produced favorable outcomes over several years, the long-term survival rate (over ten years) and integrity of the implant itself remains to be seen. Over the last decade, physician training as well as evolution of the implant have pushed the procedure to the forefront of treatment for severe ankle arthritis along with more conventional procedures including arthrodesis. Recent literature reveals improved quality, function and gait with the total ankle arthroplasty over traditional arthrodesis.[6,7,11] In this study, we reviewed six patients who underwent total ankle arthroplasty with a

Salto Talaris implant and found mixed positive and negative results. A majority of the patients, however, found the implant beneficial and have improved function with their daily activities compared to their condition prior to surgery. From this study, one can conclude the total ankle arthroplasty is not immune to complications but with the proper patient selection and qualifications, can be an effective alternative to ankle arthrodesis.

REFERENCES

1. Anderson T, Montgomery F, Carlsson A Uncemented STAR total ankle prostheses. Three to eight-year follow-up of fifty-one consecutive ankles. J Bone Joint Surg Am. 2003;85-A(7):1321-9
2. Bestic JM, Bancroft LW, Peterson JJ, Kransdorf MJ. Postoperative imaging of the total ankle arthroplasty.Radiol Clin North Am. 2008;46(6):1003-15, v-vi
3. Bonnin M, et al. The Salto total ankle arthroplasty: survivorship and analysis of failures at 7 to 11 years. Clin Orthop Relat Res. 2011;469(1):225-36
4. Bonnin MP, Laurent JR, Casillas M. Ankle function and sports activity after total ankle arthroplasty. Foot Ankle Int. 2009;30(10):933-44
5. Criswell BJ, Douglas K, Naik R, Thomson AB. High revision and reoperation rates using the Agility™ Total Ankle System. Clin Orthop Relat Res. 2012;470(7):1980-6
6. Hahn ME, et al. Comparative gait analysis of ankle arthrodesis and arthroplasty: initial findings of a prospective study. Foot Ankle Int. 2012;33(4):282-9
7. Kwon DG, et al. Arthroplasty versus arthrodesis for end-stage ankle arthritis: decision analysis using Markov model. Int Orthop. 2011;35(11):1647-53
8. Morgan SS, Brook B, Harris NJ. Is There a Use for Total Ankle Replacement in Polio Patients? A Case Report and Review of the Literature. Foot Ankle Surg. 2012;18(1):74-6
9. Nunley JA, Caputo AM, Easley ME, Cook C. Intermediate to long-term outcomes of the STAR Total Ankle Replacement: the patient perspective. J Bone Joint Surg Am. 2012;94(1):43-8
10. Morgan SS, Brooke B, Harris NJ. Total ankle replacement by the Ankle Evolution System: medium-term outcome. J Bone Joint Surg Br. 2010;92(1):61-5
11. Piriou P, et al. Ankle replacement versus arthrodesis: a comparative gait analysis study. Foot Ankle Int. 2008; 29(1):3
12. Salto Talaris Instrumentation II, Tornier, Inc.

Figure 1. Salto Talaris Implant (Tornier, Inc.)

Figure 2. The alignment guide prior to tibial resection

Figure 3: The final application of the implant

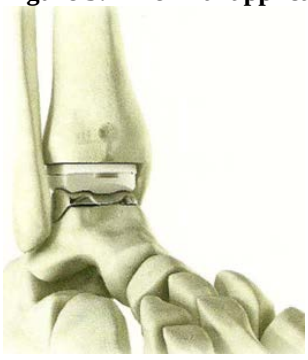


Figure 4: Pre-op X-ray of pt. with osteoarthritis

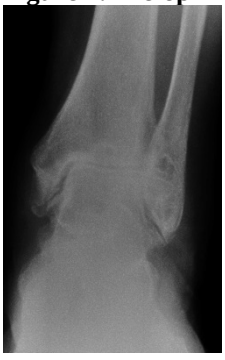


Figure 5. Post-op AP with anterior tibial angle measuring long axis of tibia with talar dome

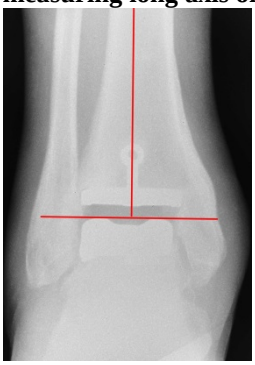


Figure 6. Lateral post-op X-ray lateral tibial and talo-calcaneal angles

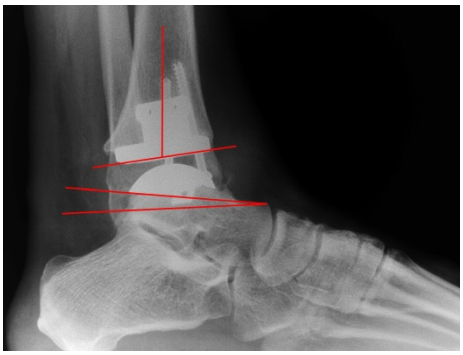


Figure 7: Noted osteolysis around the tibial component, both posteriorly and anteriorly

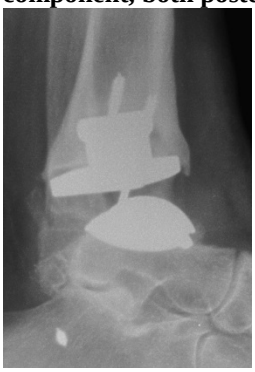


Figure 8: Osteophyte formation at post tibial and increased density at ant joint components

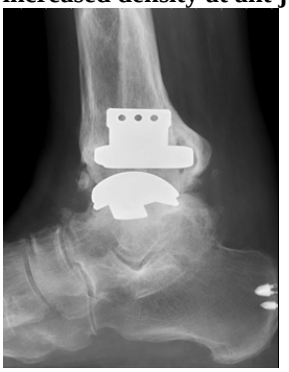


Table 1. Demographics at time of surgery

Case	Dx.	Age	M/F	Wt. (lbs.)	BMI	Kellgren-Lawrence Score
1	PTA	51	F	195	35.7	2
2	OA	58	F	207	35.6	4
3	PTA	47	M	208	30.7	3
4	PTA	64	F	193	34.2	3
5	PTA	27	F	145	26.2	2
6	OA	48	F	137	25.1	2
7	PTA	41	F	161	26	2

PTA- Post traumatic arthrosis
OA- Osteoarthritis

Table 2. Detailed history of patients

Case	Hx. & Presenting C.C.	Prior Surgery	Other Treatments	Special Studies
1	Talar OCD, PT tendonitis, chronic pain	Talar OCD repair with osteochondral graft	Injections, Bracing, Custom shoes, Immobilization	MRI
2	Ankle fracture	Ligament stabilization	Immobilization	CT Scan
3	Ankle fracture with ORIF, Ankle & Subtalar joint arthrosis	Hardware removal, Ankle arthroscopy	Injections, physical therapy, Immobilization	CT Scan
4	Ankle fracture with ORIF	Ankle arthroscopy	Injections, bracing	MRI
5	Ankle fracture with ORIF, equinus deformity	Hardware removal, Ankle arthroscopy, Tendo-achilles lengthening	Immobilization, Bracing, Physical therapy, Heel lifts	CT Scan, MRI
6	Ankle arthritis	Ankle arthroscopy	Injections, Bracing, Immobilization	MRI
7	Ankle fracture with ORIF	Hardware removal, Ankle arthroscopy	Injections, Bracing	CT Scan

OCD: Osteochondral Defect
ORIF: Open Reduction Internal Fixation

Table 3. Outcome of patients and final review

Pain Scale and Score is based on American Orthopedic Foot and Ankle Society

Case	Pain Scale	AOFAS Score	Revision	Satisfaction
1	20/40	69	N	Better
2	20/40	63	N	Better
3*	0/40	47*	N	Worse
4	20/40	70	Y	Better
5	0/40	45	N	Better
6	20/40	72	N	No Improvement
7	20/40	72	Y	Better

*This patient had a subtalar joint arthrodesis on the same side following TAR surgery.

Table 4. Evaluation of radiographic angles pre-operative, immediate post-op and at last follow-up

	TA Pre-TAR	TA Immediate Post-op	TA Min. 2 Years	TA Lat. Immediate Post-op	TA Lat. Min. 2 Years	TC Immediate Post-op	TC Min. 2 Years
Mean	88.74	89.48	87.43	85.57	80.87	14.43	11.86
Median	88.86	90	90	84	80.29	15	11.93
Standard Deviation	3.73	0.96	5.26	5.65	4.43	4	3.19

TA: Tibial Angle
TA Lat: Lateral Tibial Angle
TC: Talo-calcaneal Angle
Average follow-up 36.28 months
All values averaged to nearest hundredth

Table 5. Radiographic evaluation of osteolysis or osteophyte/spurring formation

Case	Osteolysis	Osteophyte/Spurring
1	Y	N
2	Y	N
3	N	Y
4	N	Y
5	N	N
6	N	N
7	N	Y
Population %	29	43

Article: Treatment of Distal Radius Fractures in the Athlete: A Novel Fixation Device Compared with Conventional Treatment Strategies

Kenneth Donohue, MD*, John Taras, MD**
*Drexel University College of Medicine: Department of Orthopaedic Surgery
** Philadelphia Hand Center

ABSTRACT

Background: Early rehabilitation has been shown to enhance short-term function following open reduction and internal fixation of distal radius fractures. This may be particularly important for athletes who wish to minimize stiffness and loss of dexterity during the early postoperative period. A strategy that combines rigid surgical fixation with appropriate rehabilitation may accelerate the athlete's ability to return to play and help minimize the potential for re-injury after distal radius fracture. This study evaluates the outcomes of early rehabilitation in athletes treated with an alternative fixation strategy, the purpose-designed threaded pin (T-Pin, UnionSurgical, LLC, Philadelphia, PA).

Methods: A prospective study was performed on 19 athletes with displaced, unstable, distal radius fractures treated surgically with the purpose-designed threaded pin over a 5-year period. All patients were enrolled in an early rehabilitation protocol, with formal therapy initiated on postoperative days 1 to 3. Range of motion and strength measurements were documented during the first 4 months of treatment. Postoperative complications and maintenance of reduction were recorded. The duration of follow-up extended at least 18 months.

Results: Fourteen athletes met the inclusion criteria for this study. All athletes treated with purpose-designed threaded pins and early rehabilitation demonstrated improved wrist range of motion and grip strength at average follow-up of 11 weeks. All fractures healed without redisplacement. Postoperative wrist range of motion was

comparable to published results for patients treated with volar plating and early rehabilitation at a similar time period. At 3-month follow-up, the average grip strength of patients treated with the purpose-designed threaded pin was higher than that of patients treated with the volar plate. All patients returned to their sport. The average return to competition occurred at 8 weeks postoperative.

Conclusions: Surgical fixation with the purpose-designed threaded pin is a useful alternative to volar plating for isolated radial styloid and extra-articular distal radius fractures. Both fixation techniques provide maintenance of reduction and allow for early rehabilitation. Several studies have demonstrated short-term benefits of early motion following surgical fixation. Athletes benefit from threaded pin fixation by preserving strength and dexterity and minimizing time lost before return to play.

INTRODUCTION

Distal radius fractures are the most frequent type of bony injury in the upper extremity, accounting for 8 to 15% of all skeletal injuries.[1,11] These injuries have become increasingly common because of the increased participation in sporting activities. Distal radius fractures most commonly occur in impact sports such as football, rugby, basketball, snowboarding, rollerblading, and soccer; however, they may also occur in racket and stick sports such as baseball, tennis, hockey, golf, and racquetball, or in apparatus or contact sports such as gymnastics, rock climbing and weightlifting.[5] Successful management of these fractures in the athlete requires balancing the short-term goal of allowing safe return to play with the long-term goal of minimizing radiocarpal deformity and instability. Distal radius fractures in athletes require careful consideration of pre-injury function and sport-specific treatment goals. Kinematic studies have demonstrated that these fractures can severely impair an athlete's ability to throw, catch, grasp, shoot, strike, or push-off.[12] Deformity after a fracture can reduce wrist range of motion and result in post-traumatic arthritis; thus, maintenance of anatomic reduction should be the treating surgeon's foremost concern. Studies identified a 60% incidence of fracture displacement following nonoperative management in a cast or splint.[2] In order to stabilize these fractures, a variety of surgical techniques have been developed.[3,13] Early surgical treatment strategies such as pins and plaster required prolonged immobilization, and loss of motion was often observed. The recentdevelopment of more robust fixation devices such as volar plates and the purpose-designed threaded pin (T-Pin, Union Surgical, LLC, Philadelphia, PA) has allowed for earlier rehabilitation, which may benefit the athlete considerably.

Successful rehabilitation after a distal radius fracture is critical before allowing an athlete to return to play. The primary goals of rehabilitation are to prevent re-injury, restore range of motion, increase strength, and improve dexterity. Studies have shown that hand therapy can influence short-term outcome following distal radius fracture.[6,9,14,15] Rozental et al. reported superior short-term results for distal radius fractures treated with volar plating and early range of motion when compared to fractures treated with percutaneous pinning and cast immobilization. The current series evaluates similar outcome measures in athletes treated with the purpose-designed threaded pin and early rehabilitation.

METHODS

Institutional review board approval was obtained to undertake this study. A series of 19 athletes with distal radius fractures treated with the purpose-designed threaded pin was evaluated. The study cohort included only high-level amateur athletes whose ability to participate in his or her sport was a vital part of their lives. The fracture patterns on preoperative radiographs were classified according to the AO fracture classification system (Fig 1). There were 4 type A2 fractures, 4 type A3 fractures, 4 type B1 fractures and 2 type C1 fractures. To be included in the study, athletes were required to have a primary isolated fracture of the distal radius treated with purpose-designed threaded pin fixation and initiation of an early rehabilitation protocol (Table 1). All surgeries were performed under the supervision of 1 fellowship-trained orthopaedic hand surgeon at an academic tertiary-care medical center.

Demographic information included gender, age, hand dominance, sport played, and profession. Treatment-specific parameters included mechanism of injury, AO fracture pattern, operative indications, purpose-designed threaded pin configuration, postoperative flexion, extension, pronation, supination, postoperative complications, and the number of weeks before return to sport. When available, grip strength was recorded for both the injured and contralateral wrists. Range of motion and strength data were logged from the last postoperative note having complete data during the 4-month follow-up period.

The mechanism of injury was a fall from standing height in 8 cases, a fall from a bicycle in 2 cases, and a direct blow in 4 cases. The operative criteria included dorsal angulation in 2 patients, dorsal angulation and radial shortening in 6 patients, and unstable fracture pattern in 6 cases. Fractures were assessed with standard radiographs.

All procedures were performed under conscious sedation with a local field block using bupivacaine 0.5% without epinephrine. Closed reduction was performed under fluoroscopic guidance. In cases in which closed reduction failed to recreate anatomic alignment, a 0.062-inch Kirschner wire was used to manipulate the distal fragment percutaneously. After satisfactory reduction, a 1 to 2 cm longitudinal incision was made over the radial styloid. Care was taken to protect the dorsal sensory branches of the radial nerve, and the dissection was carried down to bone between the first and second dorsal compartments. Next, a 1 mm guide wire was advanced across the fracture site from a starting point at the radial styloid. The guide wire was positioned at a 20 to 45 degree angle relative to the longitudinal axis of the radial shaft and advanced to the opposite cortex. The guide wire was measured, and the appropriately sized purpose-designed threaded pin was inserted. Fracture stability was assessed by flexing and extending the wrist under fluoroscopy. For extra-articular fractures, a second purpose-designed threaded pin was inserted either adjacent to the first in a divergent fashion or between the 4th and 5th extensor compartments in a crossed-pin configuration. The fracture pattern and availability of distal bone stock determined which pin configuration was chosen. Care was taken to ensure that all purpose-designed threaded pins were flush with bone in order to prevent soft tissue irritation. Incisions were then closed with nylon suture. Postoperative dressings consisted of sterile gauze and a volar slab splint. Typical tourniquet time was approximately 20 minutes (Figs 2-4).

All patients were seen for their initial postoperative therapy visit between postoperative days 1 and 3. At this visit, the plaster splint and dressings were removed, and the patient was fitted with a custom thermoplastic volar wrist splint. The splint was molded to position the wrist in slight extension. Patients also received instruction in a home exercise program. This program comprised tendon gliding exercises, active wrist range of motion through a 60-degree arc-of-motion, gentle radial and ulnar deviation, and gentle forearm range of motion. All exercises were performed in sets of 10 repetitions, 4 times per day. At the patients' 2-week postoperative visit, they were allowed to advance to active wrist flexion and extension as tolerated. Patients began to wean from their splint by 4 weeks. At week 6, splinting was discontinued and patients were allowed to begin strengthening exercises of the forearm, wrist, and hand. Resistance was increased according to the patient's tolerance.

RESULTS

Fourteen patients (7 males and 7 females) met the criteria for inclusion in this study. Three patients with supplemental hardware and 2 patients with previous fracture malunion, were excluded from the study. The average patient age was 35 years. Range of motion and strength evaluations were performed at an average of 11 weeks after surgery (range 5-16 weeks). All fractures healed without loss of alignment within the 4-month follow-up period. The purpose-designed threaded pin configuration included a single radial styloid pin in 7 patients, 2 divergent radial styloid purpose-designed threaded pins in 4 patients, and 2 crossed purpose-designed threaded pins in 3 patients (Table 2). Average postoperative flexion was 62 degrees, and average extension was 59 degrees. Average pronation was 83 degrees, and average supination was 80 degrees. Average postoperative grip strength measured 57 lbs. (70% of the uninjured side) at the time of release to practice play with immobilization. All patients returned to sport within 12 weeks of injury. The average time span between surgery and return to play was 8 weeks (Table 3). Postoperative complications included 1 patient with irritation from slightly prominent hardware which required removal. Two additional patients requested pin removal in the off-season, however there were no issues related to the hardware. Finally, two patients reported postoperative wrist aching that resolved completely by 6 months. There were no cases of infection, delayed union, nonunion, or significant fracture displacement.

DISCUSSION

Distal radius fractures are common injuries in athletes. When treating this population, emphasis should be placed on restoring anatomic alignment, achieving fracture union, and effectively rehabilitating the injured extremity before allowing return to play. In this study, the criteria for return to play included painless range of motion and grip strength of at least 50% of the uninjured side. In light of the high redisplacement rates following non-operative treatment, there is a current trend towards surgical stabilization of these fractures. Debate continues over the necessity of operative treatment, consequences of prolonged immobilization, and effectiveness of early rehabilitation. Despite a lack of consensus, athletes present a unique patient population which may benefit from operative stabilization and early rehabilitation after distal radius fracture.

Many sporting activities place considerable demand on the wrist and require precise regulation of the joint through its complete arc-of-motion. Biomechanical studies have shown that baseball pitching requires the wrist to move through a 94-degree-arc over 105 msec. Golf swings require a 103-degree arc in the dominant wrist and a 71-degree-arc in the nondominant wrist.[16] Basketball free throws require motion through a 120-degree-arc from 50 degrees of extension to 70 degrees of flexion.[10] In addition, athletes tend to place greater loads across the wrist during sporting activities and poorly tolerate any instability or malalignment after distal radius fracture. These facts support a more assertive approach to managing the distal radius fracture in the athlete.

Rozental et al. showed that distal radius fractures treated with open reduction and internal fixation in conjunction with early rehabilitation had superior short-term results when compared with fractures treated with percutaneous pinning and cast immobilization. They identified better DASH scores, range of motion, strength, and patient satisfaction values at postoperative weeks 6 and 9 in patients treated with volar plating and early rehabilitation. At week 9, the average range of motion in these patients measured 56 degrees of flexion, 54 degrees of extension, 81 degrees of supination, and 86 degrees of pronation.[14] This series' range of motion measurements are very similar to those reported in Rozental's study, but grip strength is higher after threaded-pin fixation. Average grip strength in the current study measured 57 lbs (70% of the uninjured side) at 11 weeks. In contrast, Rozental reported average grip strength of 29 lbs (19% of the uninjured side) at similar time period.[14] The authors of the current study hypothesize that fixation with the threaded-pin technique results in better grip strength by minimizing soft tissue trauma around the extrinsic flexor tendons of the wrist. Volar surgical approaches such as the trans-flexor carpi radialis approach used by Rozental produces more soft tissue trauma, which may delay recovery of grip strength. Previous studies have established that the advantages of surgical treatment and early rehabilitation decrease over time.[4] Lozano-Calderon et al. reported no difference between distal radius fractures that were mobilized within 2 weeks or after 6 weeks following volar plating. In that study, all patients were placed in a removable thermoplastic splint during the first postoperative week. Follow-up was conducted at 3- and 6-months after surgery and included assessment of range of motion, grip strength, Gartland and Werley score, and Mayo wrist score.[8] It is important to note that patients in that study did not receive a directed therapy program, and the authors made no attempt to confirm patient adherence to splint wear. In Rozental's study, differences in range of motion and strength lost significance after 12 weeks; yet, patients with internally fixed fractures maintained superior DASH scores, patient satisfaction, and patient-perceived wrist function until follow-up at 1 year. The authors concluded that although the advantages of internal fixation and early rehabilitation diminish over time, this treatment should be considered for patients requiring faster return to function after injury.[14]

Several studies support the use of directed therapy after distal radius fracture. Valdes investigated the effect of early rehabilitation after internal fixation in 23 patients. She reported that patients who began therapy during the first postoperative week required significantly fewer therapy sessions (6.5 versus 17 sessions) and more quickly regained functional wrist range of motion (35 versus 72 days) than patients who began therapy after 6 weeks of immobilization.[15] Kay et al. performed a randomized controlled trial to investigate the effect of therapy in 56 patients following cast immobilization. They reported improved QuikDASH score at 3 weeks and improved Patient-Rated Wrist Evaluation pain score at 3 and 6 weeks in patients who received a directed program of advice and exercises.[6] Lyngcoln et al. prospectively evaluated the effect of patient compliance in 15 patients receiving therapy following immobilization of distal radius fractures. He found that adherence to therapy predicted over 50% variance in wrist extension change, Levine questionnaire score change, and change in the simulated feeding item of the Jebsen Test of Hand Function at 6 weeks.[9]

A limitation of this study was the absence of a direct control group, although Rozental's study provides a good model for comparison. The patients in the current study were significantly younger and may have been stronger given their participation in athletics. These variables should be considered when interpreting this study's range of motion and grip strength results. There were also proportionally more AO type C fracture patterns in the Rozental study. Despite these limitations, the current study provides insight into the clinical outcomes following treatment of distal radius fractures with a new fixation device. Improvements in grip strength make intuitive sense in light of the minimally-invasive dissection required to implant the threaded pin. Finally, the benefits of rehabilitation are well established in the early postoperative period.

CONCLUSION

Surgical fixation with the purpose-designed threaded pin is an effective alternative to volar plating for isolated radial styloid and extra-articular distal radius fractures. Both techniques maintain reduction and allow for early

rehabilitation. By minimizing soft tissue dissection, the purpose-designed threaded pin may result in improved grip strength during the early postoperative period. Additionally, the use of purpose-designed threaded pins may reduce the risk of flexor tendon irritation and rupture that can occur following volar plating. In both techniques, early rehabilitation protocols likely benefit the athlete by reducing disuse atrophy, stiffness, and loss of dexterity following distal radius fracture. These benefits help to promote a safer and earlier return to play.

REFERENCES

1. Fernandez DL, Jupiter JB. Fractures of the Distal Radius. New York, Springer-Verlag, 1996
2. Gartland JJ, Werley CW. Evaluation of healed Colles' fractures. J Bone Joint Surg Am. 1951;33:895-907
3. Gupta R, Raheja A, Modi U. Colles' fracture: management by percutaneous crossed-pin fixation versus plaster of Paris cast immobilization. Orthopedics. 1999;22(7):680–682
4. Handoll HH, Madhok R. Surgical interventions for treating distal radial fractures in adults. Cochrane Database Syst. Rev 2003;3:CD003209
5. Hanker, GJ. Radius fractures in the athlete. Clinics in sports medicine. 2001;20(1):189–201
6. Kay S, McMahon M, Stiller K. An advice and exercise program has some benefits over natural recovery after distal radius fracture: a randomized trial. Australian J of Physiotherapy. 2008;54:253-259
7. Kreder HJ, et al. Indirect reduction and percutaneous fixation versus open reduction and internal fixation for displaced intraarticular fractures of the distal radius. J Bone Joint Surgery Br. 2005;87:829-836
8. Lozano-Calderon SA, Souer S, Mudgal C, Jupiter JB, Ring D. Wrist mobilization following volar plate fixation of the distal part of the radius. J Bone Joint Surgery Am. 2008;90:1297-304
9. Lyngcoln A, Taylor N, Pizzari T, Baskus K. The relationship between adherence to hand therapy and short-term outcome after distal radius fracture. J Hand Therapy. 2005;18:2-8
10. Ohnishi N, et al. Analysis of wrist motion during basketball shooting, in Nakamura RL, Linscheid RL, Miura T (eds): Wrist Disorders: Current Concepts and Challenges. New York, Springer-Verlag, 1992, 49-55
11. Palmer AK, Fernandez DL. Fractures of the distal radius. In Green DP, Hotchkiss RN, Pederson WC (eds): Green's Operative Hand Surgery, ed 4. New York, Churchill Livingstone, 1999
12. Pappas AM, Morgan WJ, Schulz LA, Diana R: Wrist kinematics during pitching a preliminary report. Am J Sports Med. 1995;23(3):312-5
13. Rodríguez-Merchán, EC. Plaster cast versus percutaneous pin fixation for comminuted fractures of the distal radius in patients between 46 and 65 years of age. Journal of orthopaedic trauma. 1997;11(3):212–217
14. Rozental, et al. Functional outcomes for unstable distal radius fractures treated with open reduction and internal fixation or closed reduction and percutaneous pinning. J Bone Joint Surg Am. 2009;91:1836-46
15. Valdes, K. A retrospective pilot study comparing the number of therapy visits required to regain functional wrist and forearm range of motion following volar plating of a distal radius fracture. J Hand Therapy. 2009;22:312-9
16. Werner SL, Plancher KD. Biomechanics of wrist injuries in sports. Clin Sports Med. 1998;17(3):407-20

Figure 1. Fracture classification

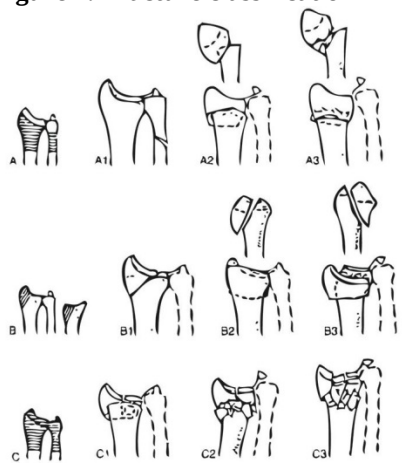


Figure 2. Pre-operative radiograph

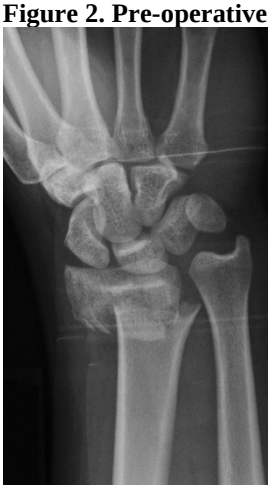


Figure 2. Pre-operative radiograph - lateral

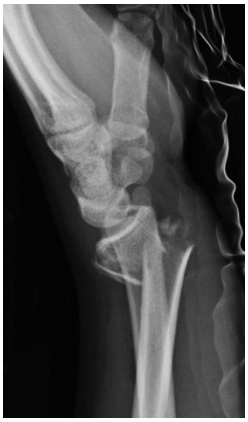


Figure 3. Post-pinning radiograph

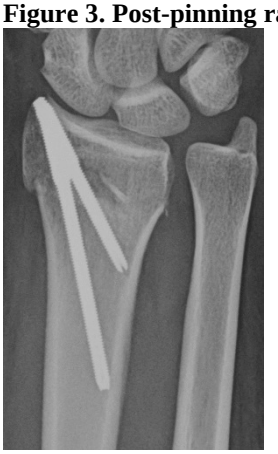


Table 1. Demographics

# Athletes	Sport
3	Cycling
3	Skating
2	Hockey
1	Skiing
1	Tennis
1	Softball
1	Football
1	Weightlifting
1	Track

Table 2. Surgical indication and fixation type

AO pattern	Operative Indication	Fixation
Type A2	Dorsal angulation + shortening	1 Radial + 1 ulnar Purpose-designed threaded pin
Type B1	Unstable pattern	1 Radial styloid Purpose-designed threaded pin
Type A3	Dorsal angulation + shortening	1 Radial + 1 ulnar Purpose-designed threaded pin
Type B1	Unstable pattern	1 Radial styloid Purpose-designed threaded pin
Type A2	Dorsal angulation	2 Radial styloid Purpose-designed threaded pins
Type A3	Unstable pattern	1 Radial styloid Purpose-designed threaded pin
Type A3	Dorsal angulation + shortening	2 Radial styloid Purpose-designed threaded pins
Type A3	Dorsal angulation + shortening	2 Radial styloid Purpose-designed threaded pins
Type C1	Unstable pattern	1 Radial + 1 ulnar Purpose-designed threaded pin
Type A2	Dorsal angulation + shortening	1 Radial styloid Purpose-designed threaded pin
Type B1	Unstable pattern	1 Radial styloid Purpose-designed threaded pin
Type A2	Dorsal angulation	1 Radial styloid Purpose-designed threaded pin
Type B1	Unstable pattern	1 Radial styloid Purpose-designed threaded pin
Type C1	Dorsal angulation + shortening	2 Radial styloid Purpose-designed threaded pins

Table 3. Post-op course and return to play data

Postop Day	Flexion	Extension	Pronation	Supination	Injured Grip Strength	Opposite Grip Strength	Weeks Return to Sport
98	45	40	90	45	34	70	4
68	65	55	90	90	65	115	8
72	55	60	80	85	25	58	8
51	75	55	80	80	71	91	6
101	50	40	80	80	75	90	12
118	60	70	80	80	56	55	8
83	60	42	60	70	30	70	10
70	62	65	70	75	50	75	10
103	80	60	90	70	31	55	2
37	50	60	80	80	70	85	4
61	65	70	90	90	38	40	8
116	90	75	90	90	66	86	10
69	40	60	90	90	95	140	10
71	70	75	85	88	100	110	8

Poem: Poem on Heart

Swarnalatha Kanneganti, MD
Easton Hospital: Department of Medicine

Heart, you are the music of life.
Your thought makes life beautiful.
Heart, you are the symbol of love.
Your rhythm plays a clairtone in my heart.
“Lub Dub” sound is the beat of heart,
Starts at birth and ends with the death.
Though we have brains, most times we think with our hearts.
Our emotions are real when they come from our hearts.

The interior décor of your chambers is amazing;
No lesser than the nature scapes.
The red blood cells, the white blood cells and the platelets;
Mixed in the flowing plasma, make a thunder blood.
You are the warrior pumping it hard.

Your circuit is fabulous, but why do you depend on a pacer?
Do you need a stimulus for every beat?
Can’t you function by yourself?
Your murmurs are rattling.
We panic if you race.
Your paradox is a functional antonym.

“Disease of the heart”, hard to accept it.
Your arrest is life threatening.
We do our best to restart you.
We do feel sorry to shock you.
Hold your “chords” and wake up;
Please do not fall asleep.
There is no life without you.

Heart, you are eternal.
Heart, you are the existence.

Review Article: Acute Toluene Toxicity from Inhalant Abuse: Case Presentation and Concise Review

Josebelo Chong, MD, Mark Lega, MD
Allegheny General Hospital: Department of Medicine, Division of Pulmonary Disease/Critical Care Medicine

CASE PRESENTATION

A 36 year-old Caucasian male presented to our facility complaining of epigastric pain and generalized muscle weakness. His history was positive for polysubstance abuse, and on further inquiry he admitted to using crack cocaine and "huffing" paint thinner for approximately 4 hours the night before. He was hyporeflexic with a generalized decrease in muscle strength. Although his vital signs were stable, his laboratory results were significantly abnormal. His potassium level was <1.5mmol/L (normal 3.5-5mmol/L), his phosphorus level was 1.3mg/dL (2.5-4.5mg/dL) his creatinine level was 1.34mg/dL (0.7-1.5mg/dL), and his lipase level was 1636U/L (6-75U/L). His arterial blood gas exhibited a metabolic acidosis with a mildly elevated anion gap of 15. Due to these grossly deranged values, he was admitted into the medical intensive care unit. He was given intravenous hydration and his electrolytes were aggressively repleted. Over the next three days, he received a total of 1180 meqs of potassium and 165 mmol of sodium phosphate, which eventually succeeded in bringing his levels to within normal range. His abdominal pain improved with bowel rest, and his lipase and creatinine levels normalized as well. He was ultimately discharged home with profuse counseling regarding substance abuse.

DISCUSSION

This patient presented with the effects of acute toluene toxicity from inhalant abuse. Toluene is a clear, colorless, volatile, water-insoluble liquid that is used as a solvent. It is one of the most abundantly-produced chemicals in the United States and is extensively used in the rubber, paint, dye, glue, printing, and pharmaceutical industries.[1] Inhalant abuse is an important yet under-recognized form of substance abuse. Due to their widespread availability, low cost, and perceived low risk of adverse effects, volatile substances that produce a transient "high" are easily abused. This includes, but is not limited to, solvents (e.g. paint and polish removers), correction and felt-tip marker fluid, cleansing agents, personal care products, and glues. Although initially thought to be mostly a problem in teens and adolescents, the incidence of inhalant abuse in adults has been rising, while the trend in the former has actually been decreasing.[2,3] In the acute setting, exposed persons can develop CNS symptoms ranging from mild headaches to hallucinations and coma. The exact mechanism of neurologic toxicity is unknown, but has been suggested to be related to its liposolubility and subsequent interaction with neuronal proteins. In the cardiovascular system, the heart becomes more sensitive to epinephrine, which can lead to arrhythmias; hence, sympathomimetic agents should be used with caution.[1] Renal effects initially include an initial high anion gap metabolic acidosis with later development of distal renal tubular acidosis, hypokalemia, and hypophosphatemia.[4-6] Rhabdomyolysis may develop, leading to acute kidney injury from acute tubular necrosis.[7] Gastrointestinal effects such as nausea, vomiting, and hepatotoxicity can also occur. Skin exposure can cause local irritation while inhalation can cause airway irritation, bronchospasm, and pneumonitis.[1] The diagnosis of toluene toxicity can be revealed by taking a detailed history, or if unavailable, recognizing the appropriate pattern of laboratory abnormalities (metabolic acidosis with profound hypokalemia, hypophosphatemia, and hypocalcemia). If the test is available, hippuric acid (an end-product of toluene metabolism) should be checked in the urine as well.[8] The key to diagnosis is a high index of suspicion; the astute clinician needs to be alert to the possibility of inhalant abuse even in adults, and should always maintain a high level suspicion when a patient presents with otherwise unexplainable metabolic derangements. Unfortunately, there is no antidote for toluene, and hemodialysis is not effective for drug removal. Treatment is supportive; appropriate decontamination procedures should be followed and any electrolyte abnormalities should be aggressively replaced.[1] Abusers should receive appropriate counseling.

REFERENCES

1. Medical Management Guidelines for Toluene. Atsdr.cdc.gov. Centers for Disease Control and Prevention, 3 Mar. 2011. Web. <http://www.atsdr.cdc.gov/mmg/mmg.asp?id=157&tid=29>
2. Center for Behavioral Health Statistics and Quality. Adults Represent Majority of Inhalant Treatment Admissions. Data Spotlight: Treatment Episode Data Set, March 17, 2011
3. Williams, et al. Inhalant Abuse. Pediatrics 2007; 119(5):1009-1017
4. Baskerville J, Tichenor G, Rosen P. Toluene Induced Hypokalaemia: Case Report and Literature Review. Emerg Med J 2001;18(6):514-16

5. Schrier, Robert W. Chapter 46: Nephrotoxicity Secondary to Drug Abuse and Lithium Use in Diseases of the Kidney and Urinary Tract. 8th ed. Vol. 1. Philadelphia, PA: Lippincott Williams & Wilkins, 2006
6. Carlisle, et al. Glue-sniffing and distal renal tubular acidosis: sticking to the facts. J Am Soc Nephrol. 1991;1(8): 1019-27
7. Reisin, et al. Myoglobinuria and renal failure in toluene poisoning. Br J Ind Med. 1975;32(2):163–164
8. Thiesen, et al. Laboratory diagnosis of toluene-based inhalants abuse. Clin Toxicol (Phila). 2007;45(5):557-62

Review Article: Catatonia in Autism Spectrum Disorder (ASD) vs. Psychotic Disorder: a potential source of confusion that may lead to overmedication and subsequent overt catatonia

KC Niranjan, MD, Lisa Jamnback, MD, Gary Swanson, MD
Allegheny General Hospital: Department of Psychiatry - Division of Child and Adolescent Psychiatry

INTRODUCTION

Catatonia, first described in 1874 by German psychiatrist Kurt Kahlbaum, is a clinical syndrome characterized by a cluster of signs and symptoms including mutism, stupor/immobility, staring, posturing, negativism, withdrawal, rigidity, and autonomic abnormalities. In the current DSM-IV-TR it is not recognized as a separate disorder, but is associated with psychiatric conditions such as schizophrenia (catatonic type), bipolar disorder, major depressive disorder, and other mood disorders, as well as drug abuse or overdose (or both). It may also be seen in many medical disorders including infections (such as encephalitis), autoimmune disorders, focal neurological lesions (including strokes), metabolic disturbances, and abrupt or overly rapid benzodiazepine withdrawal. According to the DSM-IV, the "with catatonic features" specifier can be applied if the clinical picture is dominated by at least two of the following: 1) motor immobility as evidenced by catalepsy (including waxy flexibility) or stupor, 2) excessive motor activity (purposeless, not influenced by external stimuli), 3) extreme negativism (motiveless resistance to all instructions or maintenance of a rigid posture against attempts to be moved) or mutism, 4) peculiarities of voluntary movement as evidenced by posturing, stereotyped movements, prominent mannerisms, or prominent grimacing, or 5) echolalia or echopraxia. Recently, catatonia has been increasingly diagnosed in patients with autism spectrum disorder (ASD) and has been noted in multiple clinical reports. The tem ASD itself is being increasingly used to describe group of syndromes that include autism, pervasive developmental disorder (PDD), Asperger syndrome, Rett syndrome, and childhood disintegrative disorder.

CASE REPORT

The patient is a 13 year-old female with a history of ASD and borderline MR, who was hospitalized twice within a month. Both admissions were prompted secondary to her disorganized behavior, mutism, withdrawn state and increased staring spells. A diagnosis of psychosis NOS was considered in both admissions. According to her family, at baseline the patient was able to do all her activities of daily living. Around two weeks prior to her first admission, the patient started complaining of being bullied at school. The parents lodged a complaint, but it was not taken seriously by the school authorities. The patient then started regressing and was admitted to a psychiatric unit for further evaluation and management. An aripiprazole trial was started to address the diagnosis of psychosis. There were concerns from family regarding lethargy, but she was ultimately discharged on the same medication as her symptoms seem to improve. Shortly after her discharge, she again presented to the hospital for worsening of her symptoms. This time a diagnosis of catatonic episode was also considered. Lorazepam 1mg PO TID and risperidone 1mg PO QAM and 3mg PO QHS were started, as her symptoms were considered secondary to psychosis, provisionally diagnosed as schizophrenia. Lorazepam was tapered off after five days, but risperidone was continued. The patient remained non-verbal and followed commands with significant latency. She then developed cogwheeling. Risperidone was decreased and substituted with olanzapine (5mg PO QAM and 10mg PO QHS at first, and ultimately 10mg PO BID). Symptoms further worsened and family requested discharge AMA. Once at home, the patient’s mother tapered off all her medications except lorazepam “for sleep.” Marked improvement was seen soon after.

LITERATURE REVIEWS

Catatonia in Autism Spectrum Disorder

The literature highlights the association between catatonia and autism, although they do not clarify the nature of the association. Explanations vary from autism as a manifestation of the catatonia syndrome, catatonia as a complication of autism, and catatonia as a cause of deterioration in patients with autism. Catatonia is well defined in adults, and

diagnostic criteria are detailed in Table 1.[9] Yet specific criteria for catatonia in children or adolescents are lacking. The studies that describe catatonic features in young patients, however, suggest that the criteria applied to adults are applicable to children and teenagers, and the presentation of catatonia is consistent across all age groups.[7,14,16] Several published reports suggest an association between catatonia and autism, with dramatic improvements when autistic patients with catatonia are treated for the syndrome of catatonia. A description of three autistic patients reported that they shared the symptoms of mutism, echopraxia or echolalia, and stereotypy with catatonia.[13] The authors concluded that catatonia in autism may be a variant of the autistic condition. The clinical presentation, course, and treatment response of a 14-year-old boy with catatonic stupor and with a preexisting diagnosis of autism reported that the patient displayed mutism, akinesia, motor rigidity, waxy flexibility, posturing (including psychological pillow), facial grimacing, and other involuntary movements of his upper extremities.[15] Intravenous injection of sodium amobarbital failed to resolve the motor symptoms. A course of ECT, however, elicited sustained relief of catatonic stupor without a change in the symptoms of autism. The largest study of the association of catatonia and autism was reported by Wing and Shah in 2000. Using a semi-structured diagnostic schedule to gather information from parents and caregivers of 504 patients referred to a clinic for ASDs, they reported that 17% of patients aged 15 or older had features of catatonia. These patients were more likely than the comparison group to have had, before the onset of the change in behavior, impaired language and passivity in social interaction of skill and behavior. An earlier study reported 6.5% of autistic children developing catatonia by their late teens.[17] Dhossche in 2004 described catatonic symptoms (Table 2) to be common in autism and implicated abnormal gamma-aminobutyric acid (GABA) function in both disorders.

Evaluation, Diagnosis and Treatment of Catatonia in Child and Adolescent

Possible catatonia-like deterioration in patients with ASDs should prompt a thorough clinical assessment. Physical examination and laboratory investigations (including a pregnancy test for all female patients) are dictated by clinical assessment. Infectious, metabolic, endocrinological, neurological, and autoimmune diseases have been associated with catatonia, and must therefore be ruled out.[2,9] See Table 3.

All prescribed medications should be evaluated for their potential to induce catatonic symptoms since many medical and psychiatric medications can cause catatonia or catatonia-like conditions.[9,18] Antipsychotic agents should be discontinued as they are contraindicated in patients with ASDs who exhibit the signs of catatonia because of the reported increased incidence of malignant catatonia or neuroleptic malignant syndrome (NMS) in patients with incipient signs of catatonia. When catatonia is resolved, antipsychotics may be useful for select indications, but reemergence of catatonic symptoms should prompt discontinuation. Illicit drugs (PCP, mescaline, psilocybin, cocaine, opiates, and opioids), disulfiram, steroids, antibiotic agents (ciprofloxacin), and bupropion have also been associated with the emergence of catatonia in case reports. Withdrawal of benzodiazepines, gabapentin, and dopaminergic drugs, especially if done rapidly, has precipitated catatonia in some patients.[9]

Once a diagnosis of catatonia is made, the severity of the condition should be determined by assessing the degree to which activities of daily living, occupational activities, and physiological necessities (eating, drinking, and excretion) are affected. The level of impairment should dictate the need for services and staffing levels accordingly.

Evaluation, diagnosis and treatment of catatonia are summarized in Tables 4 and 5.

DISCUSSION

This case report above describes the presentation of catatonia in a teenaged female diagnosed with ASD. While the diagnosis of catatonia is based on the DSM-IV criteria, differential diagnoses are to be considered and a thorough investigation is imperative. It is also worth looking into diagnostic criteria for catatonia in ASDs. More recently, catatonia is being increasingly diagnosed in patients with ASD. It should be considered in any patient with ASD, of any age, when there is an obvious and marked deterioration in movement, vocalizations, pattern of activities, self-care, and practical skills. It follows that ASD should also be considered as the underlying condition in patients presenting with catatonia, especially in those with histories of developmental disorders. Catatonia may be misdiagnosed as a feature of schizophrenia or other mood disorder, and any underlying diagnosis of ASD may be missed, leading to possible suboptimal treatment of both catatonia and ASD with the heavy use of antipsychotic medications or other medications triggering further deterioration as evidenced in our case. Researchers have shown varying outcomes with the use of antipsychotics in catatonia in general, with most being detrimental (except clozapine) and even leading to worsening of catatonic symptoms.[11] At the same time they have also shown increased incidence of malignant catatonia or NMS with the use of antipsychotics in catatonia in ASD. It is prudent that we

stop any antipsychotics that the patient may be prescribed until the patient comes out of the catatonic state. This case clearly shows the worsening of catatonia in ASD with the use of aripiprazole, risperidone and olanzapine. At the same time, this case also points to the beneficial effects of well-established treatment (i.e. benzodiazepine) for catatonia.

CONCLUSION

Thorough examination and consideration of differential diagnosis in ASD presenting with catatonic features is needed to prevent overt catatonia due to heavy antipsychotic use. Efficacy of benzodiazepine and ECT has been well established in the treatment of catatonia in ASD. One should not hesitate to initiate benzodiazepines in any case of catatonia. As given in the treatment algorithm, one should opt for a lorazepam challenge test in every case of catatonia. If the lorazepam challenge test fails, then ECT, another well-established treatment, may be considered. At the same time, as highlighted in various research and in our case above, antipsychotic agents should be discontinued as they are contraindicated in patients with ASDs who exhibit the signs of catatonia because of the reported increased incidence of malignant catatonia or neuroleptic malignant syndrome (NMS) in patients with incipient signs of catatonia. When catatonia is resolved, antipsychotics may be useful for select indications, but reemergence of catatonic symptoms should prompt discontinuation.

While the increasing evidence of catatonia in ASD has been supported by multiple clinical trials and case reports, further investigations are needed to establish the causal relationship between catatonia and ASD in terms of the biopsychosocial model. There have been varying outcomes with the use of antipsychotics in catatonia in general versus catatonia in ASD. Much remains to be investigated in regard to the role of individual antipsychotics in treatment of ASD with catatonia. Further, it is imperative that more research is done for robust catatonia rating scales and treatment guidelines in terms of severity in the patient population with ASD.

REFERENCES

1. Dhossche DM, Shah A, Wing L. Blueprints for the assessment, treatment,and future study of catatonia inautism spectrum disorders. Int Rev Neurobiol. 2006;72:267-84
2. Carroll B and Goforth H. Medical catatonia in Catatonia From Psychopathology to Neurobiology (S. CaroV, S. Mann, A. Francis, and G. L. Fricchione, Eds.), American Psychiatric Publishing, Inc. Washington, DC. 2004
3. Dhossche, D and Bouman, N. Catatonia in children and adolescents (letter). J. Am. Acad.Child Adolesc. Psychiatry. 1997;36:870–871
4. Dhossche, D, et al. Elevated plasma GABA levels in autistic youngsters: Stimulus for a GABA hypothesis of autism. Med. Sci. Monitor 2002;8:PR1–PR6
5. Dhossche, D. Autism as early expression of catatonia. Med. Sci. Monitor 2004;10:RA31–RA39
6. Dhossche, D and Stanfill, S. Could ECT be eVective in autism? Med. Hypotheses 2004;63:371–376
7. Wing L and Shah A. Catatonia in autistic spectrum disorders. Brit. J. Psychiatry 2000;176:357–362
8. Kakooza-Mwesige A, Wachtel LE, Dhossche DM. Catatonia in autism: implications across the life span. Eur Child Adolesc Psychiatry 2008;17:327–335.
9. Fink M and Taylor M. Catatonia: A Clinician's Guide to Diagnosis and Treatment. Cambridge University Press, Cambridge, United Kingdom. 2003
10. Fink M, Taylor MA, Ghaziuddin N. Catatonia in autistic spectrum disorders:a medical treatment algorithm. Int Rev Neurobiol. 2006;72:233-44
11. England ME, Öngür D, Konopaske GE, Karmacharya R. Catatonia in Psychotic Patients: Clinical Features and Treatment Response. J Neuropsychiatry and Clin Neurosci. 2011;23:223-226
12. Dhossche DM, Wilson C, Wachtel LE. MDl. Prmary Psychiatry 2012;17 (4):35-39
13. Realmuto G, August G. Catatonia in autistic disorder: a sign of comorbidity or variable expression. J Autism Dev Disord. 1991;21(4):517-528
14. Hare DJ and Malone C. Catatonia and autistic spectrum disorders. Autism 2004;8:183–195
15 .Zaw FK., Bates GD, Murali V, Bentham P. Catatonia, autism, and ECT. Dev. Med. Child Neurol. 1999;41:843–845
16. Takaoka K and Takata T. Catatonia in childhood and adolescence. Psychiatry Clin. Neurosci. 2003;57:129–137
17. Gillberg C and Steffenburg S. Outcomes and prognostic function in infantile autism and similar conditions. J. Autism Devel. Dis. 1987;17:273–287
18. Lopez-Canino A and Francis A. Drug-induced catatonia. In ''Catatonia. From Psychopathology to Neurobiology'' (S. CaroV, S. Mann, A. Francis, and G. L. Fricchione,Eds.), American Psychiatric Publishing, Inc. Washington, DC. 2004

Table 1. Diagnostic criteria for catatonia

A. Immobility, mutism or stupor of at least 1 hour duration, associated with at least one of the following: catalepsy, automatic obedience, or posturing, observed or elicited on two or more occasions

B. In the absence of immobility, mutism or stupor, at least two of the following, which can be observed or elicited on two or more occasions: stereotypy, echophenomena, catalepsy, automatic obedience, posturing or negativism, ambitendency

Table 2. Common catatonic symptoms

	Children (%)	*Adult (%)*
Mutism	87	78
Posturing/grimacing	52	66
Stupor	80	66
Starring	49	57
Waxy flexibility	62	35
Incontinence	45	--

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Table 3. Diff dx and psych disorders assoc w/catatonia

Table A. Differential diagnosis of catatonia	*Table B. Psychiatric disorders associated with catatonia*
Hyperkinetic disorders	Developmental disorders
Acute dystonia	Autistic Disorder
Tardive dyskinesia	Asperger Disorder
Akathisia	Pervasive Developmental Disorder Not
Withdrawal-emergent dyskinesias	Otherwise Specified (atypical autism)
Tics/ Tourette's syndrome	Possibly Childhood Disintegrative
Selective mutism	Disorder (late-onset autism)
Conversion disorder	Prader–Willi Syndrome
Compulsions (in Obsessive-	Mood disorders
Compulsive Disorder)	Psychotic Disorders including
Epilepsy	Schizophrenia
Delirium	Mental Disorders due to a General
Hypokinetic disorders	Medical Condition
Parkinsonism and Morbus	Substance-Induced Disorders
Parkinson	
Malignant hyperthermia	
Neuroleptic Malignant Syndrome	
Serotonergic syndrome	
Epilepsy	
Status epilepticus	
Delirium	
Coma	

Table 4. Pattern of treatment response

Treatment	*Cases, N*	*Observations*	*Response patterns*
Benzodiazepines	25	High doses (16mg/day) tolerated without sedation Response in days to weeks	7 definitely, 15 likely beneficial, 3 neutral
Electroconvulsive therapy (ECT)	12	Rapid response within 1-5 treatments Bilateral electrode placement Memory impairment that resolved in 50%	10 definitely, 2 likely beneficial
Antipsychotics			
Clozapine	7	Good overall improvement Requires slow titration	6 definitely, 1 likely beneficial
Olanzapine	9	Some benefit, but not consistent. Restlessness in patient. Some worsening of catatonia symptoms	4 definitely beneficial, 2 neutral; 3 likely detrimental
Quetiapine	5	Helpful with anxiety and insomnia Some confusion in patients. Some worsening of catatonia symptoms	3 definitely beneficial, 2likely detrimental
Risperidone	6	Restless and extrapyramidal symptoms observed	3 neutral, 3 likely detrimental
Aripiprazole	3	Some worsening of catatonia symptoms	2 neutral, 1 likely detrimental
Typical	5	Worsening of catatonia symptoms. Some rigidity and restlessness	2 neutral, 3 likely detrimental
Mood stabilizers			
Lithium	4	Overall helpful	4 likely beneficial
Valproate	5	Overall helpful	4 likely beneficial, 1 likely detrimental
Lamotrigine	3	Overall helpful	2 likely beneficial, 1 neutral
Antidepressant	4		3 neutral, 1 likely detrimental

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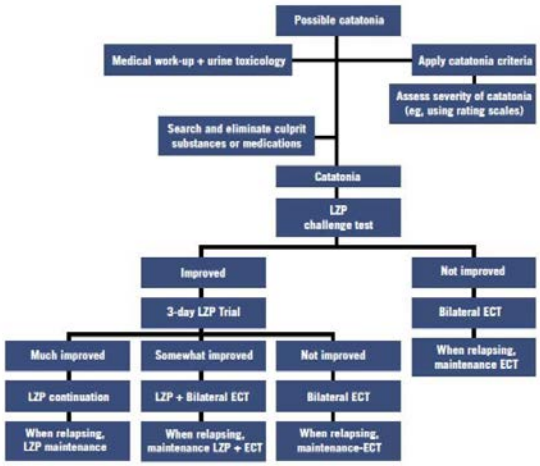
Table 5. Evaluation, diagnosis, and treatment algorithm



LZP=lorazepam; ECT=electroconvulsive therapy.

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Review Article: Commotio Cordis

Swarnalatha Kanneganti, MD, Koroush Khalighi, MD
Easton Hospital: Department of Medicine

INTRODUCTION

Commotio cordis is defined as sudden cardiac death from a life-threatening disruption of the heart rhythm due to a direct blow to the chest wall overlying the precordium. The term commotio cordis is derived from latin in which it means "agitation of the heart". In recent years, commotio cordis has gained public and media attention as it is one of

most common causes of sudden death in young athletes. This article reviews the epidemiology, potential mechanisms, treatment, and prevention of commotio cordis.

History

The occurrence of this phenomenon was described in the ancient Chinese martial art of Dim Mak (or touch of death) in which blows to the left of sternum caused sudden death in opponents.[1] But the term commotio cordis was first used in the 19th century.[2] From the mid-1700s, sporadic accounts of commotio cordis appeared in the medical literature as work place accidents. Through the mid-1990s, it was noted only on occasional case reports and went mostly unrecognized except by the forensic pathology community and Consumer Product Safety Commission.[3] In the mid-1990s, the National Commotio Cordis Registry was established in the United States for systematic collection of commotio cordis cases. Since then, the general population and the medical community have become increasingly aware of commotio cordis as an important cause of sudden cardiac death.

Epidemiology

Commotio cordis is a very rare event and is often considered when an athlete presents with sudden cardiac death. It occurs primarily in children, adolescents, and young adults involved in certain recreational or competitive sports.[4] Baseball, football, ice hockey, rugby, cricket, polo, softball, pelota, fencing, lacrosse, boxing, karate, kung fu and other martial arts are some of the sports which have high risk for this event. It can also occur in other situations like cases of torture and motor vehicle accidents in which the steering wheel can hit the chest wall. This has reduced substantially with the use of seatbelts and airbags. Sometimes the "precordial thump" given as part of CPR can cause commotio cordis. Generally, the energy delivered in precordial thumps is 5-10 times less than the threshold which can induce commotio cordis,[5] but in conditions of severe hypoxia, the minimal energy from a precordial thump can cause commotio cordis.

Young people are at higher risk which may be due to a relatively thin, underdeveloped, compliant chest cage with immature intercostal musculature. These physical characteristics of the thorax make it less capable of blunting the arrhythmogenic consequences of precordial blows.[4,6]

Since the US National Commotio Cordis Registry was established, 200 cases of confirmed commotio cordis were registered.[7,8] 95 percent of the victims were male, mean age being 15 years, and only 9 percent of reported cases have age more than 25 years. 75 percent of cases have occurred during athletics (50 percent during competitive sports and 25 percent during recreational sports). While the National Commotio Cordis Registry receives reports primarily from the United States, 60 cases from outside the US have been reported to the registry.[9] Patient demographics and survival between victims in the US and outside the US were fairly similar, though non-US victims were somewhat older (mean 19 versus 15 years of age), and a significantly greater number of non-US cases occurred during soccer (20 percent versus 3 percent of US cases).

MECHANISM

Commotio cordis is a lethal arrhythmic event which occurs when there is a direct impact on precordium in blunt chest wall injuries.[4] The mechanical energy generated by the impact profoundly alters the electrical stability of the heart, stunning the myocardium, resulting in ventricular fibrillation (VF) and sudden cardiac death. Several biologic and biochemical experimental models of commotio cordis were developed to elucidate the mechanisms by which rapid mechanical stimulus to the chest triggers VF.[10,11,12] In the late 19th century, relatively crude methods, like use of hammers and other blunt instruments, were used to induce commotio cordis in experimental animals.[13] More recent studies conducted under controlled conditions in pigs, dogs, and rabbits have provided insights into the underlying mechanisms of commotio cordis.

The critical variables which appear to increase the occurrence of commotio cordis are the timing of impact, site of impact, and velocity of impact. Timing of impact during the cardiac cycle is the most important variable in the development of VF.[10] Only impacts occuring during a 20 to 40 millisecond window on the upslope of the T-wave (early ventricular repolarization) will cause VF.[10,14] This is an electrically vulnerable period, when inhomogenous dispersion of repolarization is greatest, thus provoking VF. In pigs when blows occurred outside this brief window, VF was not the consequence; instead, what followed was transient complete heart block, left bundle-branch block, or ST-segment elevation.[10] These effects are also reported in some human survivors (with the presumed timing of the blow coinciding with the QRS complex during ventricular depolarization).[15,16] Location of the impact is another important variable. The impacts that occur directly over the cardiac silhouette can only result in VF.[17] Likelihood of

commotio cordis increases if the impact occurs at the right time in the right spot. The velocity of impact also plays a crucial role. As the velocity of the projectile object at impact increases, the chances of commotio cordis increase. As the projectile velocity increases up to 40 miles per hour (mph), the incidence of VF increases to 70 percent.[18,19] However, at impact velocities greater than 40 mph, the probability of VF decreases, while the frequency of structural damage like myocardial rupture and cardiac contusion increases.

Other factors which also increase the risk of commotio cordis include the hardness of the object and its size and shape, with hard, small, sphere shaped projectiles most likely to do harm. The heart becomes more vulnerable when it is physically strained during sports. The increase in heart rate may double the probability due to shortening of cardiac cycle.

Cellular mechanism

Information derived from experiments with Langendorff preparations of perfused rabbit heart[20] and from animal models[18,21] has led to a hypothesis concerning specific mechanistic pathways. It is believed that the mechanical force generated by precordial blows during repolarization causes left ventricular intracavitary pressure to rise instantaneously to 250-450 mm of Hg; this rise in pressure is directly correlated with an increased probability of VF.[7,17,18,20] It has been hypothesized that this elevation in pressure causes cell membranes to stretch, activating ion channels and increasing transmembrane current flow by means of mechanical-electric coupling.[20,22] The resultant amplified dispersion of repolarization creates an inhomogenous and electrically vulnerable substrate that is susceptible to VF. The culprit ion channels are ATP-sensitive potassium channels, which contribute to the initiation of VF in commotio cordis, and in myocardial infarction and ischemia.[23] It is possible that same mechanism has something in common with the pathophysiological mechanisms that give rise to primary arrhythmogenic conditions such as channelopathies.[24,25]

CLINICAL MANIFESTATION

Commotio cordis is one of the most common causes of sudden cardiac death in young athletes along with hypertrophic cardiomyopathy, congenital anomalies of the coronary arteries, arrhythmogenic right ventricular dysplasia, and channelopathies. It should be suspected if sudden unexpected death occurs in young individuals while involved in sports. Some commotio cordis events can abort spontaneously when the blow causes nonsustained arrhythmias.

The diagnosis of commotio cordis is presumptively made based on the clinical scenario[4,26] (blunt chest trauma followed by collapse), available electrocardiographic data demonstrating VF, and the absence of structural heart damage on imaging studies (echocardiogram, computed tomography of chest, etc.) or autopsy. Evidence of VF at time of injury is often not available and is not necessary to make the presumptive diagnosis, but supports the diagnosis if documented. The absence of structural cardiac injury distinguishes this from cardiac contusion in which high-impact blows result in traumatic damage to myocardial tissue and the overlying thorax.

Outcome

Commotio cordis appears to be a primary electrical event, with VF being the initial documented arrhythmia in most victims. Survival remains poor, with a reported survival of only 25 percent in all patients in the National Commotio Cordis Registry.[7] The outcome mainly depends on the circumstances in which commotio cordis occurs. Deaths have often been associated with the failure of bystanders to appreciate the life-threatening nature of the collapse and to initiate aggressive and timely measures of resuscitation.[6,26] Registry data show that survival rates have increased over time, rising to 35% over the past decade as compared with 15% for the preceding 10 years. This might be due to increased public awareness, availability of automated external defibrillators, and earlier activation of resuscitation measures.

PREVENTION

Primary prevention can be done in several ways.[28] The most important method is public education about the causes and early identification and initiation of resuscitation measures. Young athletes should be taught the importance of avoiding precordial blows.[27] Even an unintentional, modest-seeming blow to the chest can trigger life-threatening ventricular tachyarrhythmias.[6] Organized sports present the greatest opportunity to prevent commotio cordis by means of improved coaching techniques such as teaching young batters in baseball and softball to turn away from the ball to avoid errant pitches. Also the defensive players in lacrosse and hockey can be coached to avoid using their chest to block the ball preventing the goal.[7]

Improved design of commercial sports equipment would also probably help to prevent commotio cordis, like the safety baseball, a softer ball intended for players younger than 13 years.[30] Chest protectors and vests intended to reduce trauma from blunt bodily injuries are available on the market. However, the registry data indicate that they do not provide protection against commotio cordis.[4,6,7,31] Indeed, currently available chest protectors may create a false sense of security, given that almost 20% of commotio cordis victims in competitive football, baseball, lacrosse, and hockey were wearing equipment marketed as providing protection against traumatic chest injury.[4,6,7,31,32]

Secondary prevention measures include increased distribution of automated external defibrillators at youth sporting events and recreational settings, availability of coaches trained in providing life support, and enlightening the public about the importance of early initiation of resuscitation.[29]

Legal Issues

Several people have been convicted of involuntary manslaughter in cases involving insufficient and slow medical help to the victims during sports events[33,34] as well as in cases of intentional delivery of contusive blows.[35]

CONCLUSION

Commotio cordis is a life-threatening condition in which sudden cardiac death occurs from lethal disruption of THE heart rhythm due to a direct blow to the chest wall overlying the precordium. Primarily it is an electrical event, with VF being the initial documented arrhythmia in most victims. Most often it occurs in young athletes during recreational and competitive sports. The timing, location, and velocity of impact appear to be critical determinants to trigger the event. Survival and outcome are poor. More efforts are needed to prevent this avoidable cause of death by increasing public awareness for early initiation of chain of survival, which is an important part of cardiopulmonary resuscitation.

REFERENCES

1. Kelly M. Death touch: the science behind the legend of Dim-Mak. Boulder, CO: Paladin Press, 2001
2. Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. Lancet 2001;357:1195-1197
3. Rutherford GW, Kennedy J, McGhee L. Baseball and softball related injuries to children 5-14 years of age. Washington, DC: Consumer Product Safety Commission, 1984
4. Maron BJ, Poliac L, Kaplan JA, Mueller FO. Blunt impact of the chest leading to sudden death from cardiac arrest during sports activities. N Engl J Med 1995;333:337-342
5. Kohl P, Sachs F & Franz M (eds): Cardiac Mechano-Electric Feedback and Arrhythmias: from Pipette to Patient. Elsevier (Saunders), Philadelphia 2005
6. Maron BJ, et al. Clinical profile and spectrum of commotio cordis. JAMA 2002;287:1142-1146
7. Maron BJ, Estes NA 3rd. Commotio cordis. N Engl J Med 2010; 362:917
8. Maron BJ, et al. Commotio cordis and the epidemiology of sudden death in competitive lacrosse. Pediatrics 2009;124:966-971
9. Maron BJ, et al. Global epidemiology and demographics of commotio cordis. Heart Rhythm 2011;8:1969
10. Link MS, et al. An experimental model of sudden death due to low energy chest-wall impact (commotio cordis). N Engl J Med 1998;338:1805-1811
11. Liedtke AJ, Allen RP, Nellis SH. Effects of blunt cardiac trauma on coronary vasomotion, perfusion, myocardial mechanics, and metabolism. J Trauma 1980;20:777-785
12. Liedtke AJ, Gault JH, Demuth WE. Electrocardiographic and hemodynamic changes following nonpenetrating chest trauma in the experimental animal. Am J Physiol 1974;226:377-382
13. Frazer M, Mirchandani H. Commotio cordis, revisited. Am J Forensic Med Pathol 1984;5:249-251
14. Madias C, et al. Commotio cordis-sudden cardiac death with chest wall impact. J Cardiovasc Electrophysiol 2007; 18:115
15. Link MS, et al. Commotio cordis: cardiovascular manifestation of a rare survivor. Chest 1998;114:326-328
16. Abrunzo TJ. Commotio cordis: the single, most common cause of traumatic death in youth baseball. Am J Dis Child 1991;145:1279-1282
17. Link MS, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. J Am Coll Cardiol 2001;37:649
18. Link MS, et al. Upper and lower limits of vulnerability to sudden arrhythmic death with chest wall impact (commotio cordis). J Am Coll Cardiol 2003;41:99-104
19. Viano DC, Andrzejak DV, Polley TZ, King AI. Mechanism of fatal chest injury by baseball impact: development of an experimental model. Clin J Sport Med 1992;2:166-171

20. Bode F, et al. Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotio cordis. J Cardiovasc Electrophysiol 2006;17:1011-1017
21. Stout CW, Maron BJ, VanderBrink BA, Estes NAM III, Link MS. Importance of the autonomic nervous system in an experimental model of commotio cordis. Med Sci Monit 2007;13:BR11-BR15
22. Madias C, Maron BJ, Supron S, Estes NAM III, Link MS. Cell membrane stretch and chest blow-induced ventricular fibrillation (commotio cordis). J Cardiovasc Electrophysiol 2008;19:1304-1309
23. Link MS, et al. Selective activation of the K+ATP channel is a mechanism by which sudden death is produced by low energy chest wall impact (commotio cordis). Circulation 1999;100:413-418
24. Haissaguerre M, et al. Mapping and ablation of ventricular fibrillation associated with long-QT and Brugada syndromes. Circulation 2003;108:925-928
25. Antzelevitch C, Oliva A. Amplification of spatial dispersion of repolarization underlies sudden cardiac death associated with catecholaminergic polymorphic VT, long QT, short QT and Brugada syndromes. J Intern Med 2006;259:48-58
26. Maron BJ. Sudden death in young athletes. N Engl J Med 2003;349:1064-1075
27. Link MS, et al. Protecting our children from the consequences of chest blows on the playing field: a time for science over marketing. Pediatrics 2008;122:437-439
28. Link MS. Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. Circ Arrhythm Electrophysiol 2012;5:425
29. Myerburg RJ, Estes NAM III, Fontaine JM, Link MS, Zipes DP. Task Force 10: automated external defibrillators. J Am Coll Cardiol 2005;45:1369-1371
30. Link MS, et al. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. Pediatrics 2002;109:873-877
31. Doerer JJ, Haas TS, Estes NAM III, Link MS, Maron BJ. Evaluation of chest barriers for protection against sudden death due to commotio cordis. Am J Cardiol 2007;99:857-859
32. Kaplan JA, Karofsky PS, Volturo GA. Commotio cordis in two amateur ice hockey players despite the use of commercial chest protectors: case reports. J Trauma 1993;34:151-153
33. Maron BJ, Mitten MJ, Greene Burnett C. Criminal consequences of commotio cordis. Am J Cardiol 2002;89:210-213
34. Froede RC, Lindsey D, Steinbronn K. Sudden unexpected death from cardiac concussion (commotio cordis) with unusual legal complications. J Forensic Sci 1979;24:752-756
35. Denton JS, Kalelkar MB. Homicidal commotio cordis in two children. J Forensic Sci 2000;45:734-735

Review Article: Gastric Pneumatosis: A Rare Clinical Entity with Benign to Life-Threatening Causes

Alexey Markelov, MD, Nikita Shah
Easton Hospital: Department of Surgery

INTRODUCTION

Pneumatosis intestinalis, or air within the wall of the gastrointestinal tract, has been documented from the esophagus to the rectum. Its presence can suggest gangrenous changes of the stomach or colon and represents a surgical emergency. However, gastric pneumatosis can also occur as a result of a benign, nongangrenous condition. In our case, the probable cause was direct transmural diffusion of gas from the air-distended stomach. We review the reported cases of gastric emphysema and emphasize clinical recognition of this rare entity, which might be caused by benign nongangrenous condition and an unnecessary laparotomy may be avoided in a patient who presents with similar findings and does not appear acutely ill.

CASE REPORT

A 43 year-old male without any significant past medical or past surgical history developed recurrent loose bowel movements 24 hours prior to presentation, associated with moderate epigastric pain and abdominal distention. CT scan of the abdomen and pelvis showed a curvilinear gas within the posterior nondependent wall of the stomach suggestive of gastric pneumatosis (Fig 1). There was no evidence of bowel obstruction or inflammatory changes or pneumoperitoneum. Patient's symptoms manifested after he tried new scuba diving equipment in the pool at his house. Laboratory work up was within normal limits and he did not show any signs of sepsis. Abdominal exam was

benign and decision for conservative management was made. On the next day the patient's symptoms resolved and he was able to tolerate regular diet.

DISCUSSION

Gastric pneumatosis is a rare clinical finding that has been well documented in medical literature. The spectrum of clinical presentation can be very broad, ranging from benign radiographic findings to septic shock. Gastric pneumatosis or gastric emphysema describes the presence of gas within the stomach wall. This is caused by a disruption in gastric mucosa leading to the dissection of air into the wall. There are various causes for this phenomenon: conditions causing raised intragastric pressure, i.e. gastric outlet obstruction, post-gastroscopy (air insufflation and other instrumentation may cause mucosal damage), severe vomiting, which causes mucosal damage, and dissection of air from the mediastinum (e.g. ruptured bullae or pneumothorax) (Table 1). In our case the most probable cause was increased intragastric pressure from pressurized air from scuba tank. Patients are usually asymptomatic or complain of abdominal discomfort. Plain radiographs characteristically demonstrate a linear lucency conforming to the contour of a thin-walled, often distended, stomach, and enveloping any intraluminal gas and fluid content. The clinical course is usually benign, with spontaneous resolution after the cause is removed. In these cases, gas is not produced within the stomach wall but enters it from elsewhere. This should be the main criterion in making the diagnosis of gastric pneumatosis/emphysema.

In comparison, emphysematous gastritis occurs when there is diffuse infiltration of the stomach wall by pathogenic gas-forming bacteria. Thus the gas is formed within the stomach wall. In these cases, an earlier gastric mucosal injury allows gas-forming organisms to gain access to deeper tissue layers. Causes of this condition include ingestion of toxic or corrosive substances, alcohol ingestion, trauma, gastric infarction, ulcer disease, and necrotising enterocolitis. The most common organisms involved include haemolytic Streptococci, Clostridia welchi, Escherichia coli, and Staphylococcus aureus. These patients present with severe, acute epigastric pain, fever, shock, toxemia, nausea, often hematemesis, and a leukocytosis. The mere finding of gastric pneumatosis and its resolution with nonoperative management make our case interesting. Within the context of each clinical situation, the physician must determine whether conservative management is appropriate or whether gastric pneumatosis is a manifestation of ischemia or infarction that necessitates surgical exploration.

REFERENCES

1. Lee S, Rutledge JN. Gastric emphysema. Amer J Gastroenterol 1984;79(11):899-904
2. Low VHS, Thompson RI. Gastric emphysema due to necrosis from massive gastric distention. Clin Imaging 1995;19(l):34-36
3. Chin WS. Interstitial gastric and duodenal emphysema. Can Assoc Radiol J 1979;29:136-37
4. Kussin SZ, Henry C, Navarro C, Stenson W, Clain DJ. Gas within the wall of the stomach: Report of a case and review of the literature. Dig Dis Sci 1982;27(10):949-54
5. Zenooz NA, Robbin MR, Perez V. Gastric pneumatosis following nasogastric tube placement: a case report with literature review. Emerg Radiol 2007;13(4):205-7

Figure 1. CT showing gas in posterior stomach wall

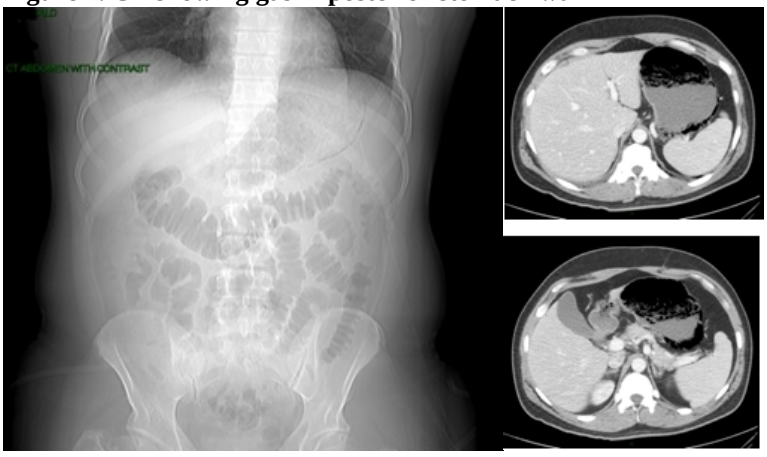

Table 1. Reported causes of gastric pneumatosis

Iatrogenic	Traumatic	Obstructive	Pulmonary
Endoscopic argon plasma coagulation Migration of biliary stent Placement of NG tube Post cardiac surgery Polychemotherapy	Mechanical Severe/prolonged vomiting Endoscopy with air insufflation Gastric surgery Gastrostomy Chemical causes: acids, alcohol, anti-inflammatories Mucosal ischemia Necrotizing enterocolitis GI infarction Gastric distention	Gastric outlet obstruction Peptic ulcer disease Antral cancer Pyloric Cancer Carcinoid tumor Hypetrophic pyloric stenosis Gastric volvulus Obstructing esophageal cancer Duodenal obstruction Gallstones Pancreatic cancer Duodenal stenosis Small bowel obstruction	Bullous pulmonary emphysema Asthmatic attack Positive pressure ventilation Obstructive airway disease Cardiopulmonary resuscitation

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Review Article: Imaging Appearance of Complications of Bacterial Meningitis in Pediatric Patients: A Review

Mary Mallon, MD, Eric Faerber, MD, Erica Poletto, MD, Jacqueline Urbine, MD
St. Christopher's Hospital for Children: Department of Radiology

ABSTRACT
A comprehensive review of pediatric patients with meningitis imaged at our institution between 2000 and the present was performed. The analysis yielded a wide variety of complications in patients diagnosed with meningitis. A multimodality review of the imaging appearance of complications associated with meningitis is offered here with emphasis on CT and MRI. These complications include cerebritis with abscess, hydrocephalus, ventriculitis, subdural effusion progressing to empyema, dural venous sinus thrombosis, and vasculopathy resulting in a territorial infarction. Since complicated meningitis can have longstanding and devastating repercussions, radiologists who read pediatric neuroimaging studies should be aware of the spectrum of abnormalities that may be encountered.

INTRODUCTION
Bacterial meningitis is relatively common in the pediatric population and may develop associated complications with potentially longstanding and devastating repercussions. These sequelae can include hearing loss, paresis, epilepsy, and psychomotor retardation.[1] Bacterial meningitis affects children more commonly than adults in approximately a 2:1 ratio.[2] Between 30-40% of children ultimately suffer long term sequelae of varying degrees of severity after an episode.[1,3]

Diagnosis of meningitis is mainly based on clinical findings, as imaging studies are often normal. Consequently, lumbar puncture remains the gold standard for diagnosis of meningitis. Laboratory evaluation of the cerebrospinal fluid in bacterial meningitis should reveal increased protein and decreased glucose levels.[3] Imaging has maximum utility in demonstrating complications related to meningitis.[3] Reasons to suspect complicated meningitis include an increasing head circumference, a worsening clinical condition, lack of response to appropriate treatment, neurologic deterioration, seizures or focal neurologic deficits, and/or signs or symptoms of increased intracranial pressure.[2-4] Multiple various complications of bacterial meningitis are described here as identified by MRI and CT imaging.

IMAGING FINDINGS
Intra-axial complications of bacterial meningitis include cerebritis with or without abscess, ventriculitis, and hydrocephalus. A cerebral abscess can be the cause or the result of meningitis with bacterial dissemination to the brain occurring via hematogenous spread.[3]

The most basic abnormal imaging finding resulting from uncomplicated meningitis is meningeal enhancement. Meningeal enhancement on cross-sectional imaging studies, while often seen in meningitis, is a non-specific finding; however, the pattern of enhancement might suggest a specific underlying etiology.[3,5] This enhancement can follow two patterns (Fig 1). Pachymeningeal enhancement involves the dura mater and is visible abutting the inner

table or along the dural reflections in the falx cerebri, tentorium cerebelli, falx cerebelli and cavernous sinus.[5] This is distinct from leptomeningeal enhancement which involves the pia and arachnoid layers and follows brain surface or the subarachnoid space.[5] Both pachymeningeal and leptomeningeal enhancement can be seen in the setting of meningitis.

Communicating hydrocephalus (Fig 1) is the most common complication seen with meningitis, resulting from the presence of a purulent exudate which prohibits the Pacchionian granulations, perivascular spaces, and brain from performing their normal function of resorption of CSF.[3] Hydrocephalus related to meningitis is often transient, but can evolve into chronic hydrocephalus and occasionally necessitates CSF shunting.[3]

Ventriculitis (Fig 1) results from significant infection of the ependymal lining of a ventricle, often in concert with severe meningitis of the basilar cisterns or with intraventricular rupture of a cerebral abscess. Imaging findings of ventriculitis include thick enhancing ependyma lining the affected ventricle, often with hyperintense signal of the ventricular wall on T2-weighted images.[3] Ventriculitis can lead to asymmetric or unilateral hydrocephalus involving only the affected ventricle (Fig 1).[3] Complex debris layers in the affected ventricle and intraventricular septations often can develop.[3]

Abscesses most commonly develop in the region of the gray-white junction of the frontal and temporal lobes.[3] Over a period of weeks a cerebral abscess can evolve from a cerebritis and progress to necrosis.[3] Cerebritis on CT appears as regional hypodensity compatible with parenchymal edema (Fig 1).[2] Cerebritis manifests on MRI as a spectrum of findings including varying degrees of abnormal parenchymal hyperintensity on fluid sensitive sequences with or without associated abnormal enhancement.[3]

With time, early abscess formation may be seen as a poorly defined or incomplete ring of peripheral enhancement.[5] A mature abscess is characterized by a complete rim of smooth enhancement which often extends to the region of the ventricles (Fig 1) and demonstrates marked internal restricted diffusion.[3,5] Typically, the rim of a mature abscess is hypointense on T2-weighted sequences and there is extensive surrounding cerebral edema.[3] MR spectroscopy can assist in differentiating pyogenic from atypical bacterial infection for a mature abscess, as a pyogenic abscess should result in a lack of choline and the presence of amino acids.[2] If a mature abscess is in close proximity to a ventricle raising a concern for potential intraventricular rupture, drainage is considered; however, some mature cerebral abscesses can be treated successfully with antibiotic therapy.[3]

Extra-axial fluid collections can also complicate meningitis. Sterile subdural collections can develop as a reaction to the underlying inflammation, as is the case with a subdural effusion. In contrast, subdural empyemas contain purulent material, as they are more commonly associated with bacterial meningitis as opposed to meningitis with a different underlying etiology. Subdural effusions are present in up to 1/3 of patients with meningitis and are usually frontotemporal in location.[6] A subdural effusion can evolve into a subdural empyema with time (Fig 2). In a child diagnosed with meningitis, an increasing head circumference must raise a suspicion for extra-axial collection development.[3]

Subdural effusions are thought to originate as a result of leakage of fluid from vein walls due to inflammation.[3] Drainage of an effusion might be necessary if the collection exerts mass effect on the underlying brain or if the collection is particularly large, although effusions typically resolve spontaneously upon treatment of the meningitis.[3,6] Fluid in an effusion should be similar to cerebrospinal fluid on most MRI sequences. The collection may appear slightly brighter than CSF on proton density and fluid attenuated inversion recovery (FLAIR) sequence images due to the presence of serosanguinous contents and may demonstrate peripheral and/or internal enhancement related to the presence of a fibrin network.[3] Differentiation between a subdural collection and benign prominent subarachnoid space of infancy can be assisted by identifying crossing veins in the fluid collection that would suggest a subarachnoid location.

An empyema differs from an effusion clinically and radiologically. Clinically, a patient with an empyema tends to exhibit persistent abnormal findings on neurologic examination.[3] On MRI, an empyema tends to be hyperintense relative to CSF on T1-weighted images, demonstrates peripheral enhancement with or without septations, and markedly restricts diffusion internally. Restriction of diffusion is an important tool to help distinguish an empyema from a sterile effusion which should be dark on diffusion weighted imaging. The presence of signal abnormality in

the adjacent cortex and associated findings such as an abscess or venous thrombosis are also suggestive of an empyema as compared to an effusion.[3]

Vascular complications of meningitis can affect the veins and the arteries. Children commonly become dehydrated with meningitis, increasing the risk of venous thrombosis in the pediatric population. This is important because up to 30% of patients with venous thrombosis suffer a venous cerebral infarction as a result.[3] Thrombophlebitis is typically seen with meningitis or with mastoiditis which can also cause meningitis. Meningitis can also induce arterial spasm resulting in territorial cerebral infarction. Alternatively, direct spread of infection or inflammation to the arteries can have the same effect.[3] The arteries most commonly affected are those within the perivascular spaces. As a result, while involvement of larger arteries can produce large territorial cortical infarctions, most infarctions secondary to meningitis-induced vasculopathy involve the basal ganglia.[3]

Parenchymal imaging findings in venous infarct related to meningitis can be difficult to identify prospectively, as the appearance can be very similar to cerebritis. CT might demonstrate regional hypodensity reflecting parenchymal edema. On MRI, findings include regions of hyperintense signal on fluid sensitive MRI sequences that do not correspond to a typical arterial distribution.[3] T1-weighted images might be helpful in identifying thrombosed cortical veins or thrombosed dural venous sinuses, both of which could appear hyperintense.[3] MR venography and post contrast imaging can also be helpful in identifying venous thrombosis as a hypointense filling defect in a vein or venous sinus (Fig 3).[3] Diffusion weighted imaging is not always reliable as a means of identifying regions of venous infarct.[3]

Prior to the development of cerebral infarction in an arterial vascular distribution, MR angiograpy may reveal abnormalities related to meningitis-induced vasculopathy such as narrowing of large vessels, irregularity of medium vessels, and occlusion of distal branches.[3] Infarctions resulting from this vasculopathy typically follow an established vascular distribution. With time, such a vascular insult evolves as expected from an area of edema to laminar necrosis to eventual encephalomalacia with ex-vacuo dilatation of the adjacent ventricle (Fig 3). Diffusion weighted imaging is very helpful in identifying regions of ischemia related to meningitis-induced arterial vasculopathy.

DISCUSSION

Bacterial meningitis affects the pediatric population twice as much as the adult population in the United States.[2] The diagnosis of meningitis is achieved by a combination of good clinical history, physical examination, and lumbar puncture with laboratory evaluation of the CSF.[3] Rapid diagnosis and recognition of potentially complicated meningitis is paramount in treating children with bacterial meningitis because untreated or complicated meningitis can have permanent life-altering sequelae. Patients can lose hearing or motor function or develop epilepsy as a result of bacterial meningitis in childhood.[1] Complicated meningitis might be suspected for a variety of reasons including physical examination findings or neurologic abnormalities.

CT is often employed as a first line cross-sectional imaging study to evaluate for hydrocephalus in cases of suspected complications of meningitis.[2] Additional complications related to meningitis might be seen on CT, such as edema, mass effect, or extra-axial fluid collections. If more complicated meningitis is suspected MRI examination of the brain is a very useful tool.[2,3] MRI demonstrates the complexity of extra-axial fluid collections in greater detail than CT. In addition, MRI may reveal the true extent of parenchymal edema and the stage of cerebritis and/or abscess formation that the meningitis has reached. Early ischemia related to meningitis-induced vasculopathy is more readily identified by diffusion weighted imaging on MRI rather than CT. Venous infarction, however, can be more challenging to diagnose as the associated edema does not necessarily restrict diffusion the way a territorial infarction does. Vascular abnormalities might be identified prior to ischemic change by MR angiography or MR venography.

All physicians caring for children and all radiologists reading pediatric neuroimaging studies must be aware of the potential complications related to meningitis. Prompt imaging to establish the extent of these complications contributes to efficient healthcare decision making, treatment, and evaluation of prognosis.

REFERENCES

1. Jan W, et al. Diffusion-weighted imaging in acute bacterial meningitis in infancy. Neuroradiology 2003;45:634-39

2. Slovis TL, et al. Caffey's Pediatric Diagnostic Imaging. Chapter 51:Brain Infections. 11th Edition. Philadelphia: Mosby Elsevier 2008, p751-753
3. Castillo M. Imaging of Meningitis. Seminars in Roentgenology. 2004 Oct;39(4):458-64
4. Barkovich AJ and Raybaud C. Pediatric Neuroradiology. Chapter 11: Infections of the Developing and Mature Nervous System: Meningitis. 5th Edition. Philadelphia:Lippincott Williams and Wilkins 2102, p975-990
5. Smirniotopoulos JG, et al. From the Archives of the AFIP: Patterns of Contrast Enhancement in the Brain and Meninges. Radiographics. 2007;27:525-551
6. Mohan S, et al. Imaging of Meningitis and Ventriculitis. Neuroimaging Clinics of North America. 2012;22:557-583

Figure 1. CT with purulent exudate (top left), tuberculoma (top right), ventriculits with hydrocephalus (bottom left) and parenchymal abscess (bottom right)

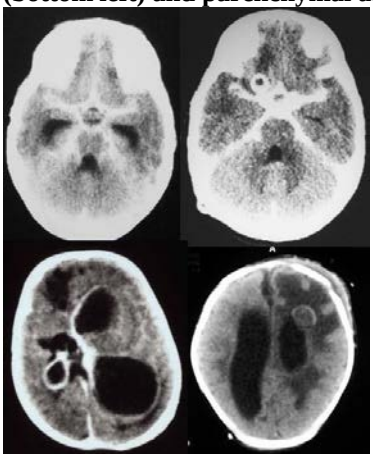


Figure 2. CT subdural effusion (left) can evolve into a subdural empyema (middle) which is seen as hyperintense compared to CSF on T1-weighted MRI (right)

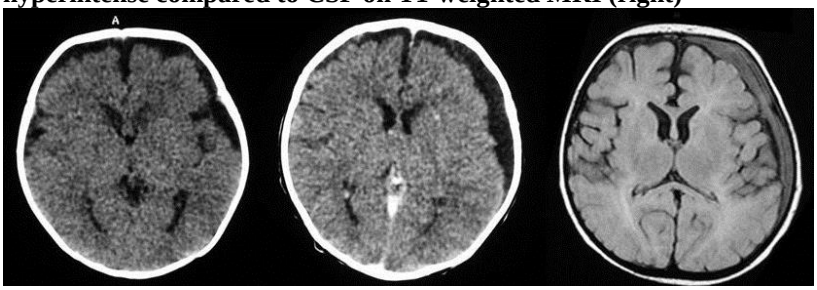
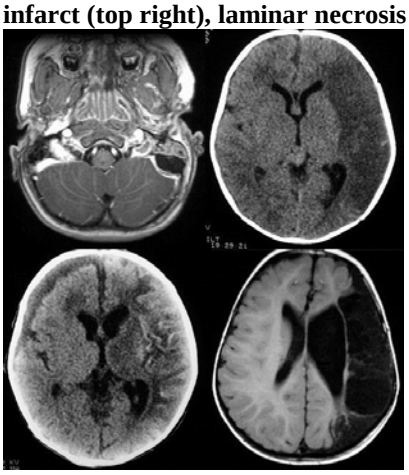


Figure 3. MRI with left mastoiditis/epidural abscess with sigmoid sinus & IJ thrombosis (top left), left MCA infarct (top right), laminar necrosis (old infarct-bottom right) and encephalomalacia (old infarct-bottom left)



Review Article: Primary Small Cell Carcinoma of the Bladder – Case Report with Review of the Literature

Shefali Ballal, MD, Steve Hou, MD
Drexel University College of Medicine: Department of Pathology and Laboratory Medicine

INTRODUCTION

Small cell carcinoma of the urinary bladder is a rare, poorly differentiated neuroendocrine epithelial tumor associated with a more aggressive behavior and poorer outcome than bladder transitional cell carcinoma.[1] The first case was described in 1981 by Cramer et al.[2] Up to 25% of patients present with metastatic disease and up to two thirds develop distant recurrence.[3]

CASE REPORT

We report here a case of small cell carcinoma of the urinary bladder in a 56 year-old man with past history of chronic hepatitis B. He initially presented with hematuria in April 2012. CT of the abdomen/pelvis without contrast on 10/7/12 showed a 4.3 x 2.2 cm mass-like lesion in the right bladder wall. He underwent a transurethral resection of the bladder tumor on November 6, 2012. Gross examination of the specimen showed multiple tan-pink fragments of soft tissue measuring 6.8 x 5.2 x 2.6 cm in aggregate. On light microscopy, the tumor cells were arranged in cohesive sheets with nuclear molding, high nuclear cytoplasmic ratio, and hyperchromatic nuclei (Fig 1). Muscle and lymphovascular invasion was present with areas of necrosis (Fig 2). The mitotic rate was high with a count of 92 mitotic figures/10HPF. Immunohistochemistry revealed that the tumor cells were positive for CD56, chromogranin, synaptophysin and TTF-1 and negative for CK7 and CK20 confirming the diagnosis of small cell carcinoma (Figs 3,4). Our patient had pure small cell carcinoma histology. There was no evidence of clinical metastatic disease. The patient was recommended to have 4 cycles of neoadjuvant chemotherapy with cisplatin (80mg/m2-Day1) and etoposide (100mg/m2–Day 1-Day 3) every 3 weeks followed by evaluation for cystectomy.

DISCUSSION

Carcinoma of the bladder is the second most common urologic malignancy. In the western world it is the fourth most common cancer in men and the eighth most common cancer in women.[4] Small cell carcinoma of the bladder is extremely rare and accounts for less than 0.7 % of all cancers arising from the bladder and a range between 0.35% and 1.8%.[5-10] The reported incidence is less than 1-9/1,000,000 habitants. Since 1980, less than 1000 cases of small cell carcinoma of the bladder have been diagnosed and reported in the literature up to July 2011.[1] The majority of patients are male with a mean sex ratio of 5:1 and a range between 1:1 to 16:1.[5-17] Most patients are in the sixth to seventh decade. The mean age at first diagnosis is 67 years with a range between 32 to 91 years.[8,11,13,14] Small cell carcinoma of the bladder is often associated with history of smoking[7,10,11,18] and the vast majority of patients are white.[8,18,14] Gross hematuria is the most common presenting symptom[8,11,18,14] with dysuria being the second most common symptom.[5,18] The pathogenesis of small cell carcinoma is not well defined. The three most important hypotheses used to explain the origin include 1) malignant transformation of bladder neuroendocrine cells, 2) arising from urothelial metaplastic changes, or 3) a multipotential common stem cell with an ability to differentiate into various cell types.[19,20] The diagnosis is based on the criteria established by the WHO classification system (2004) used for the diagnosis of small cell lung carcinoma.[1] Immunohistochemistry markers for neuroendocrine markers are extremely useful in establishing the diagnosis.[1] In most cases the diagnosis is made at advanced stages.[3] More than 95% of the cases are diagnosed at the muscle invasive stage of T2 or more.[8-11,13-14,18] The most frequent sites of metastasis were pelvic and retroperitoneal lymph nodes, liver, bone, brain and lung.[5,14] Most series have reported a higher incidence of mixed small cell carcinomas[3] with pure small cell histology having a worse prognosis.[1,9,10]

TREATMENT

There is no standard treatment for small cell carcinoma of the bladder due to the rarity of the disease and in the absence of randomized clinical trials.[1,3] Ismaili et al. in their review paper have the following recommendations for the treatment of small cell carcinoma: neoadjuvant chemotherapy followed by radical resection should be considered as the treatment of choice in surgically resectable small cell carcinoma of the bladder. This sequence can achieve a cure in 78-80% of the patients.[21-24] Neoadjuvant chemotherapy has four theoretical advantages: 1) the early treatment of micrometastatic disease, 2) the systemic treatment is better tolerated by allowing the preoperative administration of chemotherapy drugs in optimal doses with less toxicity, 3) small cell carcinoma of the bladder is highly chemosensitive, hence the majority of patients have good responses, and 4) downstaging, which facilitates the surgical resection.[25,26] Sequential chemoradiotherapy is a second treatment option which can achieve a cure in 36-70% of the cases.[7,27] In cases where the surgery has been performed first, adjuvant chemotherapy or adjuvant

chemoradiotherapy should be indicated.[8,9] In advanced stages, chemotherapy based on cisplatin should be considered as the treatment of choice for patients with good performance status and good renal function with a glomerular filtration rate >60 ml/min. The treatment should be based on neuroendocrine regimens type etoposide plus cisplatin or the sequential protocol of ifosamide plus doxorubicin at day 1 and etoposide plus cisplatin at day 21. In unfit patients, cisplatin should be substituted by carboplatin AUC 5 to 6.[1]

Currently, our patient is being treated with systemic chemotherapy using cisplatin and etoposide. The disease has a poor prognosis with the overall 5 year survival rate in all stages being 19% (16-25%).[8,11]

CONCLUSION

To our knowledge, primary small cell carcinoma of the bladder is a rare and aggressive tumor with no standard treatment. The prognosis of small cell carcinoma of the bladder is poor with a pure small cell histology having a poorer outcome than the mixed small cell histology.

REFERENCES

1. Ismaili. A rare bladder cancer-small cell carcinoma: review and update. Orphanet Journal of Rare Diseases 2011;6:75
2. Cramer SF, Aikawa M, Cebelin M. Neurosecretory granules in small cell invasive carcinoma of the urinary bladder. Cancer 1981;47(4):724-30
3. Ismaili N, et al. Small cell carcinoma of the urinary bladder: a case report and review of the literature. J Cancer Res Ther 2009;5(2):133-6
4. Haresh KP, et al. A prospective study evaluating surgery and chemoradiation in muscle invasive bladder cancer. J Cancer Res Ther 2007;3:81-5
5. Blomjous CEM, Vos W, De Voogt HJ, van der Valk P, Meijer CJLM. Small cell carcinoma of the urinary bladder. A clinicopathologic, morphologic, immunohistochemical and ultrastructural study of 18 cases. Cancer 1989;64(6): 1347-57
6. Holmang S, Borghede G, Johansson SL. Primary small cell carcinoma of the bladder: a report of 25 cases. J Urol 1995;153(6):1820-2
7. Lohrisch C, Murray N, Pickles T, Sullivan L. Small cell carcinoma of the bladder: Long term outcome with integrated chemoradiation. Cancer 1999;86(11):2346-52
8. Choong NW, Quevedo JF, Kaur JS. Small cell carcinoma of the urinary bladder. The Mayo Clinic experience. Cancer 2005;103(6):1172-8
9. Quek ML, et al. Radical cystectomy for primary neuroendocrine tumors of the bladder: the university of southern California experience.J Urol 2005;174(1):93-6
10. Ismaili N, Elkarak F, Heudel PE, Flechon A, Droz JP. Small cell cancer of the bladder: The Leon-Berard cancer centre experience. Indian J Urol 2008;24:494-497
11. Cheng L, et al. Small cell carcinoma of the urinary bladder: A clinicopathologic analysis of 64 patients. Cancer 2004;101(5):957-62
12 .Bex A, et al. Small cell carcinoma of bladder: a single-center prospective study of 25 cases treated in analogy to small cell lung cancer. Urology 2005;65(2):295-9
13. Iczkowski KA, et al. Small cell carcinoma of urinary bladder is differentiated from urothelial carcinoma by chromogranin expression, absence of CD44 variant 6 expression, a unique pattern of cytokeratin expression, and more intense gamma-enolase expression. Histopathology 1999;35(2):150-6
14. Siefker-Radtke AO, et al. Evidence supporting preoperative chemotherapy for small cell carcinoma of the bladder: a retrospective review of the M. D. Anderson cancer experience. J Urol 2004;172(2):481-4
15. Mukesh M, Cook N, Hollingdale AE, Ainsworth NL, Russell SG. Small cell carcinoma of the urinary bladder: a 15-year retrospective review of treatment and survival in the Anglian Cancer Network. BJU Int 2009;103(6):747-52
16. Mangar SA, et al. Small-cell carcinoma of the urinary bladder: 10-year experience. Clin Oncol (R Coll Radiol) 2004;16(8):523-7
17. Bex A, de Vries R, Pos F, Kerst M, Horenblas S. Long-term survival after sequential chemoradiation for limited disease small cell carcinoma of the bladder. World J Urol 2009;27(1):101-6
18. Abrahams NA, Moran C, Reyes AO, Siefker-Radtke A, Ayala AG. Small cell carcinoma of the bladder: a contemporary clinicopathological study of 51 cases.Histopathology 2005;46(1):57-63
19. Ali SZ, Reuter VE, Zakowski MF. Small cell neuroendocrine carcinoma of the urinary bladder: a clinicopathologic study with emphasis on cytologic features. Cancer 1997;79:356-361

20. Oesterling JE, Brendler CB, Burgers JK, Marshall FF, Epstein JI. Advanced small cell carcinoma of the bladder. Successful treatment with combined radical cystoprostatectomy and adjuvant methotrexate, vinblastine, doxorubicin, and cisplatin chemotherapy. Cancer 1990;65(9):1928-36
21. Pan CX, Zhang H, Lara PN Jr, Cheng L. Small cell carcinoma of the urinary bladder: diagnosis and management. Expert Rev Anticancer Ther 2006;6(12):1707-13
22. Ismaili N, et al. Small cell cancer of the bladder : pathology, diagnosis, treatment and prognosis. Bull Cancer 2009;96(6): E30-44
23. Pant-Purohit M,Lopez-Beltran A, Montironi R, MacLennan GT, Cheng L. Small cell carcinoma of the urinary bladder. Histol Histopathol 2010;25(2):217-21
24. Wang X, MacLennan GT, Lopez-Beltran A, Cheng L. Small cell carcinoma of the urinary bladder-histogenesis, genetics, diagnosis, biomarkers, treatment and prognosis. Appl immunohistochem Mol Morphol 2007;15(1):8-18
25. Ismaili N, et al. The role of chemotherapy in the management of bladder cancer. Prog urol 2011;21(6):369-82
26. Ismaili N, et al. Neoadjuvant or adjuvant chemotherapy: what is the best treatment of muscle invasive bladder cancer? Oncol Rev 2011;5(3):185-189
27. Bex A, de Vries R, Pos F, Kerst M, Horenblas S. Long term survival after sequential chemoradiation for limited disease small cell carcinoma of the bladder. World J Urol 2009;27(1):101-6

Figure 1. H&E stain with scant cytoplasm, nuclear molding and increased mitotic figures

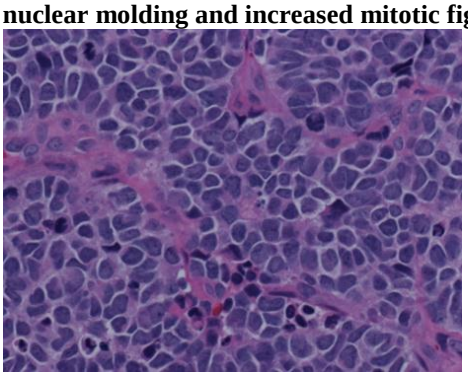


Figure 2. H&E stain showing infiltration of the muscle by tumor cells with hyperchromatic nuclei

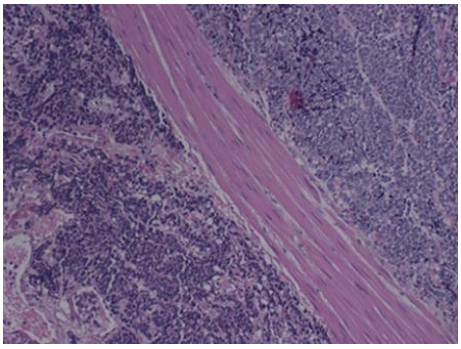


Figure 3. Neuroendocrine marker CD56 positive tumor cells

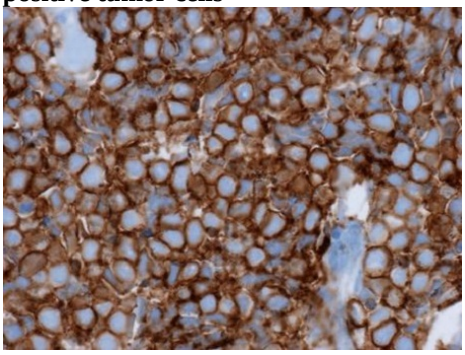


Figure 4. Neuroendocrine marker synaptophysin positive tumor cells