PANHANDLE HEALTH

A QUARTERLY PUBLICATION OF THE POTTER-RANDALL COUNTY MEDICAL SOCIETY

Winter 2017 | VOL 28 | NO. 1

Texas Tech Case Studies

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On The Cover: "Sounds of Silence", by Marsha Clements.

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President's Message:
In Closing...(December 2017)

by Rouzbeh K. Kordestani, M.D., MPH

I write this letter as a close to my year as the President of the medical society in the Panhandle. I hope it is not the "Swan Song" as Cindy our executive director put it. But I can say that this year has been educational.

I initially approached this year with hefty ideas, ready to take on the world and take on change. I was not looking forward to a year with a President I did not like. However, like any American, I realized that this was the new President and he must be given a fair chance. Months later, I realize that he is a mess and problematic. But then I realized as a physician, I have a great deal of control and ability. I realized that our patients and "normal" people do not. I realized we have to be strong for our patients in a sea of confusion, regardless of who or what is on the horizon. I realized that it's not about me, but more about them, the patients and more about US—the patients and the caretakers in the health system. That emboldened me--a sense of responsibility always tends to sharpen my focus.

Then came the unexpected retirement/sabbatical. I had to undergo a few unexpected operations on my arms and elbows. Because of that, I had to retire. Thankfully, I am now recovering. But through the process, I realized (as every physician needs to contemplate), that this career could/will end. I realized I was poorly prepared mentally for the journey. In speaking to others, many are not prepared at all, mentally or, worse, financially. I encourage you all to look at yourself with an open mind and make preparations because retirement will happen to all of us.

Two other revelations then came to pass in the last few months. The first is that I realized the medical/health landscape is now thinly populated. The new generation of physicians and medical students are too ready to dodge responsibility or to choose the easy way out. My generation (you guys) is too comfortable in keeping the responsibility and the mantle--this is a mistake. We need to teach better and to delegate; we need to expect more; we need to make the new generation ready; we need to choose good apprentices and start teaching them what we know and need to pass on. This is a must--the system must endure well past our generation and us.

The other thing I have realized in the last several months is that modern medicine/surgery is under assault. It seems that everyone seems to say the system is "bad" or "broken." Yet no one actually wants to fix it. I believe the only people who can fix it are the patients and the health care givers. To this end, doctors need to band together and make choices. We do not need another society of sorts. We need to assume the responsibility and the power that comes with numbers. By this, I mean entities such as Unions/ Cooperatives. If college football players can unionize, why can't physicians? People say that is "collusion." I beg to disagree. In our collective, we may have the largest collection of intelligence in this country. We were given the responsibility of keeping other human beings alive--that is the greatest of honors, the highest of privileges. Why then not turn that intelligence inwards? Moreover, I would argue that every hospital/system/political group has tried to fix the medical system and in serial fashion has failed. The field of medicine, both the art and science, belongs to us. Health care started with the patients and us. Why not give us the chance to fix it? I urge you all to accept the mantle of responsibility, to join together, to band together for yourself and for your patients and help fix the system. The ultimate fix comes from us, each and every one of us. Please think about it seriously and with conviction and stand to make the change. If you do not, the system will fail and you will have failed yourself and all those people who trusted that you would not falter.

Our Next Issue Of

Panhandle Health

Features:

Veteran's Health Care

Alliance News

by Irene Jones, Co-President

The Potter-Randall County Medical Alliance recently had their end of the year membership quarterly meeting at Crush Wine Bar. I want to thank everyone who has supported the Alliance in the past and especially in the last 2.5 years during my time as president. I feel honored to be in this medical community as a spouse and thankful to have met so many wonderful physicians, physician spouses and healthcare workers. The Alliance successes are plentiful this year due to our board, membership involvement and fundraising team. I know we will continue serving the community as Kristen Atkins takes leadership in 2018. I hope to see you at one of our Alliance quarterly meetings next year. Bring a friend, learn about our Alliance and serve in any capacity you can.

2017 Successes:

~$12,000 Donated to Our Children's Blessing

~$5,000 Children's Miracle Network

~Twelve Ronald McDonald House Meals provided for over 60 families

~$750-$1,000 worth of Hygiene Products delivered to ACTS in San Jacinto

~Fifty formal dresses donated to Palo Duro H.S.

~$635 raised at Doctor's Day for Bicycles and Hard Hats

~$1,200 worth of Bikes and Scooters purchased for Hard Hats Give away

~300 Hard Hats fitted

~300 Backpacks stuffed and filled with Supplies for Heal the City

~$2,500 grant for Heal the City

~$3,500 Susan G. Komen West Texas Race for the Cure

~Several College Scholarships

SHOUTOUTS!

Thank you Ana Rodriguez for stocking the Hygiene Closet and to Melissa Albracht for providing a meal to the Ronald McDonald House in October.

Hope to see you at our upcoming NYE Gala!

~December 31st: New Year's Eve Gala at The New Downtown Embassy Suites Hotel.

(please visit our website for more information www.potterrandallalliance.com)

Benefitting Heal the City, Our Children's Blessings and PRCMA.

Sincerely,
Irene Jones-PRCMA President

Executive Director's Message

by Cindy Barnard, Executive Director

The articles in our Winter issue of *Panhandle Health* are case studies by physicians and residents at Texas Tech. A case study is an "in-depth study of one individual". Ideally, a case study details a particular medical case and describes the background of the patient and discusses investigations undertaken in order to determine a diagnosis/diagnoses. A case study also might indicate a previous course of treatment the patient underwent. In general, case studies are informative and a useful part of every physician's medical education, both during training and on a continuing basis. "By reviewing case studies, physicians may gain a broader understanding of clinical diagnoses, treatments and outcomes." As the year ends, I want to thank the 2017 Board of Directors for their service and dedication to our Society. Under the leadership of our President, Dr. Rouzbeh Kordestani, 2017 has been an exceptional year. The following physicians deserve a big thank you for their support as well:

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Robin Martinez, M.D. – Physician Health and Rehabilitation

Another thank you goes to the 2017 *Panhandle Health* Editorial Board, led by Dr. Traci Crnic, Editor, and Dr. Paul Tullar, Associate Editor. Other members are Walter Bridges, M.D., Tarek Naguib, M.D., Steve Urban, M.D., Ellen Hampsten, M.D., and Mary Elhardt, M.D.

A final thank you goes to our 2017 "Circle of Friends" for their continued financial support and generosity . Their commitment is absolutely essential to the success of all our events. They are Amarillo National Bank, Baptist Community Services, Duncan & Boyd Jewelers, Neely, Craig & Walton Insurance Agency, Texas Medical Association Insurance Trust, Texas Medical Liability Trust, Interim Healthcare, Happy State Bank, Panhandle Eye Group, L.L.P., Cenveo, Daryl Curtis, CLU, CHFC, and Physicians Financial Partners.

Our cover for this issue is by Marsha Clements.

Attention: Active, Retired And Resident Members Of Potter Randall County Medical Society

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Reservations must be made by Tuesday, January 9, 2018

Invitation in the mail to follow

Guest Editor's Message

by Ellen Hampsten, M.D.

In the Panhandle, we are uniquely gifted to have an academic center of medicine. Usually associated with bigger cities, academic medical centers bring in cutting edge medical care, research and leadership. For a region like the Panhandle, this is important. Not only are we training physicians to care for patients with the most up-to-date guidelines and strategies, but we are also able to recruit and maintain those physicians.

This issue of *Panhandle Health* contains articles from residents, medical students and faculty physicians at Texas Tech University Health Sciences Center here in Amarillo. We wanted to share with you what they have been seeing and learning from our region. Please, enjoy this issue created by our young colleagues.

POTTER RANDALL COUNTY MEDICAL SOCIETY (PRCMS) OFFERS HELP TO ADDICTED PHYSICIANS

If you, or a physician you know, are struggling with addiction and are unsure what to do or whom to contact, the Potter-Randall County Medical Society is here to help. We offer face-to-face confidential sessions with the PRCMS Physician Health and Wellness Committee, made up of your physician peers who know and understand recovery. Please don't struggle alone when help is a phone call or an email away. Whether you are calling for yourself, your practice partner, or as a family member of a physician, contact Cindy Barnard, PRCMS Executive Director, at 806-355-6854 or prcms@suddenlinkmail.com. Membership in PRCMS is not required.

NEW MEMBER SPOTLIGHT 2017

BOARD OF CENSORS REPORT: The following were approved for membership on November 14, 2017

FIRST YEAR MEMBERSHIP:

CARTHEL, KADE, M.D. – CD/IC
6200 I40 West, Amarillo TX 79106.
Graduated from Texas Tech University Health Science Center, Lubbock TX 2010. Internship and Residency at Scott & White Hospital, Temple TX 2011-2013. Fellowship (Cardiology) at University of Texas Health Science Center, Houston TX 2013-2016. Fellowship (Interventional Cardiology) at University of Texas Health Science Center, Houston TX 2016-2017.

PATEL, SHRESTHA, D.O. – ON
1000 S. Coulter, 79106.
Graduated from Des Moines University College of Osteopathic Physicians & Surgeons, 2010. Internship & Residency at University of Texas Medical Branch, Galveston TX 2010-2013. Fellowship (Medical Oncology) at University of Texas Medical Branch, Galveston TX 2015-2017.

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Transfer from Bexar County Medical Society.
6826 Plum Creek Drive, Amarillo TX 79124.
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2101 Coulter (ER NOW), Amarillo TX 79106.
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MURRELL, BRIAN, M.D. – APM
4104 SW 33rd St., #200, Amarillo TX 79109. (Rejoined.)
Graduated from Wayne State University School of Medicine, Detroit MI 1983. Internship and Residency at Sinai Hospital, Detroit MI 1983-1987. Fellowship at Medical College of Ohio, Toledo OH 1098-1988.

OBOKHARE, I. DANIEL, M.D. – CRS/GS
1400 Coulter, Amarillo TX 79106. (Rejoined).
Graduated from Howard University College of Medicine, Washington DC 2006. Residency at Case University Hospital of Cleveland, Cleveland OH 2008-2011. Colorectal Fellowship at Ochsner Clinic, New Orleans LA 2011-2012.

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6100 Purple Sage Circle, 79124.

But It's Not MS!

by Eugene Nwankwo, MS3; Jenny K. Nguyen, M.D.; John Mark Tohlen, M.D.; Evelyn Sbar, M.D.

INTRODUCTION:

Demyelination is a condition that involves a loss or destruction of neuronal myelin with preservation of axons. Usually, this is a result of diseases known to damage myelin sheaths or cells that form them. Demyelination disorders may be of congenital origin, or more commonly, acquired in association with other systemic or infectious diseases. In rare cases, demyelination may present as a primary problem (without prior medical condition).

CASE PRESENTATION:

A 29 year-old Caucasian male with no significant past medical history presented with 2-3 months of persistent and progressive neck pain associated with weakness that worsened abruptly 2 weeks before presentation. Additionally, he began to suffer from flu-like symptoms of headache, myalgias, malaise, worsening generalized weakness, and fever. He reported an ascending pattern of muscular weakness. Two days before admission, the patient became unable to walk or get out of bed. He denied any recent illness, toxic exposure, or recent travel. He denied family history of multiple sclerosis or other neurologic disease. He worked as a tree trimmer and part-time fire fighter. Past medical, family, and social history were otherwise unremarkable. Past surgeries included an appendectomy and cholecystectomy. NKDA. Vital signs included: BP 156/93, HR 91, RR 20, T 98.6F, O2 Sat 95% on RA, Wt 86 kg. Physical examination showed him to be somnolent but was oriented x3 when aroused. Strength 1/5 in lower extremities and 3/5 in upper extremities. Diminished reflexes globally. Neck stiffness was present. Labs: CBC and CMP normal with exception of ALT of 100. Imaging: Head MRI from an outside facility was normal with no lesions noted.

Hospital course: LP with CSF analysis was performed initially and patient was empirically placed on vancomycin, ceftriaxone, and acyclovir. Infectious disease (ID) consult was ordered. Neurology consult was placed as well but neurologist was out of town. ID made diagnosis of aseptic meningitis based on CSF results. During initial few days of admission, patient had minimal improvement in muscle weakness and began to complain of abdominal pain and distention. An ileus was suspected prompting consults to surgery and GI. NG tube and bowel regimen were instituted resulting in resolution of symptoms. Further CSF results returned and were positive for 3 oligoclonal bands. MRI of spine was negative for lesions. Antibiotics were de-escalated but patient's weakness still did not improve with management for aseptic meningitis. Eventual neurology consult resulted in an EMG that confirmed peripheral demyelination disease and a statement that patient had "good potential for full recovery." Patient had received IV antibiotics, antiviral treatment, and 2 days of high dose steroids with very little improvement. He was then started on IVIG for 5 days, which he completed in the hospital. He began to have very slow improvement in muscular strength but suffered from postural hypotension, neurogenic bladder, and stool incontinence. Ultimately, he was discharged to LTAC for continued physical therapy with a diagnosis of demyelinating polyneuropathy subvariant, eventually classified as Miller-Fisher variant of Guillain Barre syndrome.

DISCUSSION:

Although rare, infectious diseases such as encephalitis, myelingoencephalitis, myelitis, radiculopathy, and neuropathies have been known to affect the peripheral nervous system (1). Given the similarities in presentation between the infectious and autoimmune etiologies, careful attention must be paid to the patient's symptoms (2). Our patient initially presented with signs that led to suspicion of viral meningitis. Common signs include fatigue, somnolence, headache, neck stiffness, and abdominal pain (3). However, the patient presented with a far longer duration (over 2 months) of symptoms than would be expected for aseptic meningitis, which is usually acute and self-limiting. Furthermore, an ileus is relatively rare in meningitis patients (4)(5). Given the patient's unique symptoms, it was crucial to broaden the investigation for immunologic and other inflammatory agents.

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a form of chronic neuropathy that can be diagnosed based on electrodiagnostic and pathological studies. CIDP may be associated with of anti-myelin associated glycoprotein antibodies or multifocal motor neuropathy, which indicates multifocal weakness and motor conduction blocks (6). The major clinical feature is a symmetric involvement of extremities, with motor being affected more than sensory. There is often more proximal than distal muscle weakness (1)(2). In addition, autonomic dysfunction (urinary retention, constipation, orthostatic hypotension) may be observed (7).

CIPD and multiple sclerosis (MS) display similarities in clinical course and pathogenesis. In some cases, there have been reports of co-occurrence of the two disorders (8). A finding of two or more oligoclonal IgG bands (OB) in CSF reflects a local B-cell response accompanying central nervous system inflammation. More than 95% of patients with MS have CSF OB (9). However, presence of oligoclonal bands can be indicative of various other neuro-inflammatory conditions ranging from neurodegeneration to infection (10).

CIDP can further be classified as acquired or hereditary. Acquired variants are most amenable to immune modulating treatment (11). The first line treatment is steroids. However, an important consideration is that patient response is often affected by the stage of the disease. Newly diagnosed patients are most responsive to steroids owing possibly to a higher level of pro-inflammatory cytokines and higher levels of alternative splicing early in the inflammatory disease process (12)(13). Either steroids or IVIG are shown to be of similar efficacy with improvement expected in 50 – 80% of patients (14)(15)(16)(17). The high cost and limited avail-

ability of IVIG makes it a less realistic option for many patients.

CONCLUSION:

Infectious diseases may affect the peripheral nervous system. Given the similarities in presentation between the infectious and autoimmune etiologies, careful attention must be paid to both the patient's symptoms, as well as their histories, to reach the appropriate diagnosis and provide the most beneficial treatment plan.

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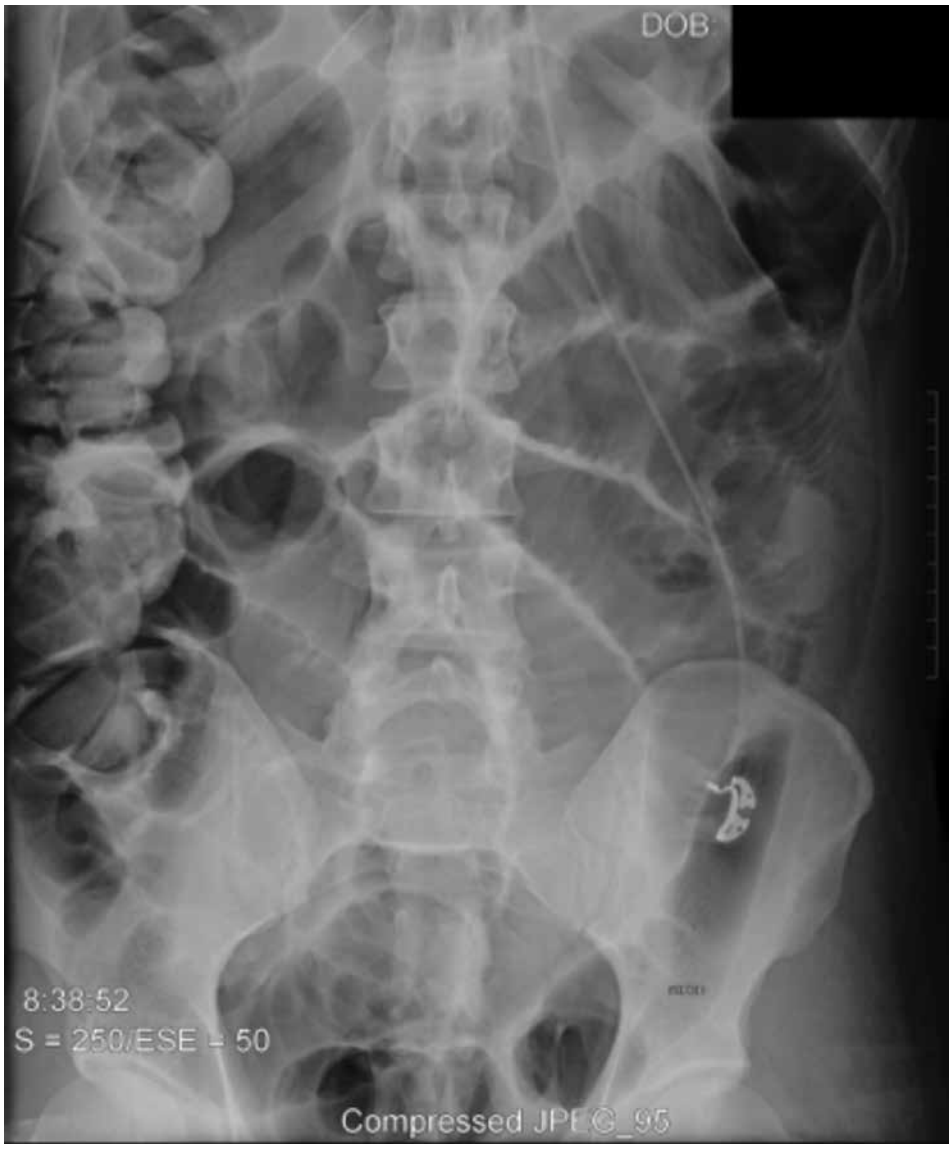

Figure 1. Abdominal x-ray showing bowel impaction.

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Infective Endocarditis in Two Patients with Congenital Heart Disease: A Review of Guidelines for Prophylaxis

by Mehar Hoda M.D., Alya Abuhantesh M.D., Srilatha Alapati M.D.

ABSTRACT

Despite advances in diagnosis, antimicrobial therapy, surgical techniques, and management of complications, patients with infective endocarditis (IE) still have high morbidity and mortality rates. The guidelines for IE prophylaxis continue to evolve as high quality literature on the subject surfaces. We present two cases of tetralogy of fallot with absent pulmonary valve syndrome that developed significant complications from infective endocarditis, and review the latest guidelines on prophylaxis.

INTRODUCTION:

Infective endocarditis (IE) is an uncommon but potentially lethal disease with an annual incidence ranging from 3 to 7 per 100,000 persons (1). Clinical findings are related to four underlying phenomena, namely, bacteremia, valvulitis, immunologic responses, and/or emboli and therefore present a wide range of symptoms, making the diagnosis difficult. Despite advances in diagnosis, antimicrobial therapy, surgical techniques, and management of complications, patients with IE still have high morbidity and mortality rates. The American Heart Association (AHA) guidelines for IE prophylaxis have been in a process of evolution for more than 50 years with the first document on this subject released in 1955. Since then, the recommendations have evolved to include the changing epidemiology and microbiological resistance pattern (2, 3). It is notable that much of the earlier rationale was based largely on expert opinion and what seemed to be a rational and prudent attempt to prevent a life-threatening infection. Over the years there has been a shift in the basic principles that drove the formulation of the AHA guidelines as high quality literature surfaced. However, there continue to be differences in guidelines between the United States and the rest of the world. We present two patients with Tetralogy of Fallot (TOF) with absent pulmonary valve who developed IE with different presentations but significant comorbidies and review the current guidelines from the AHA on the prevention of IE.

CASE 1:

Our first case is a 17-year-old female who presented to an outlying facility with fever and sore throat. She has been diagnosed with TOF with absent pulmonary valve syndrome and atretic left pulmonary artery segment at birth and had undergone Ventricular Septal Defect (VSD) repair, RV-PA homograft conduit and reduction arterioplasty of the right pulmonary artery at the age of nine with no residual VSD. An x-ray of the chest revealed increased pulmonary vascular markings, and she was then transferred to pediatric intensive care unit.

On admission she was found to be anemic and thrombocytopenic. Transthoracic echocardiogram (TTE) showed a mobile echogenic mass on the leaflet of the pulmonary valve measuring 2.2 cm x 0.65 cm (Fig:1) with pulmonary insufficiency. Blood culture grew *Streptococcus viridans* and she was started on IV vancomycin and IV ceftriaxone but continued to have persistent heart failure requiring inotropic support. During the course of treatment she developed acute kidney injury and a ventricular dysrhythmia with torsades de pointes and suffered cardiopulmonary arrest for approximately ten minutes. She was started on amiodarone to control her ventricular dysrhythmia. After cardioversion, she was transferred to Dallas, where she underwent transvenous implantable cardioverter defibrillator placement.

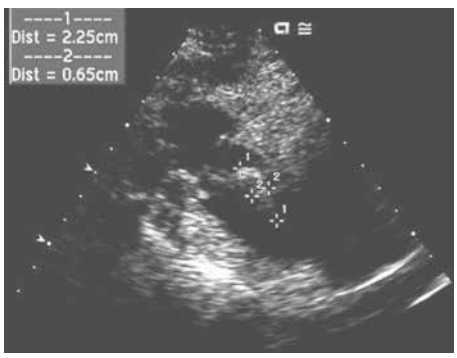

Fig: 1 - Parasternal short axis showing the vegetation measuring 2.2 cm x 0.65 cm on pulmonary valve.

Head imaging showed anoxic changes consistent with anoxic encephalopathy. Since that episode she has developed several neurological issues including psychosis, disorientation and insomnia, followed by neurology and psychiatry. Six months after treatment she underwent pulmonary valve replacement with porcine heterograft and reduction arterioplasty of right pulmonary artery. Four-year follow up revealed neurological improvement and normal cardiac function. She is a candidate for endocarditis prophylaxis.

It is notable that this patient had dental braces and had undergone a dental procedure a couple weeks prior to onset of symptoms. The procedure along with daily mucosal disruption with resultant bacteremia was believed to have been the cause of IE in this patient.

CASE 2:

Our second case is a 19-year-old male who presented to a local emergency department complaining of one week of RUQ and LUQ abdominal pain, vomiting, diffuse muscle weakness, and fatigue. He has a past medical history of TOF with absent pulmonary valve syndrome at birth and underwent complete repair with VSD closure and RV-PA conduit. Later in life the RV-PA conduit became stenotic and was replaced with a Edwards Perimount pulmonary valve; which then became stenotic and was replaced with a Melody valve. He was admitted to the adult intensive care unit, where a TTE showed significant enlargement of the right ventricle

(Fig: 2), with vegetations on the Melody valve and acute right ventricular systolic failure and moderately depressed LV systolic function. He was found to have MSSA septicemia and soon developed septic shock and acute cholecystitis, shortly after which he was transferred to Dallas for further management.

For the endocarditis and MSSA septicemia he was treated with IV naficillin for a total of 6 weeks. He then underwent removal of the Melody valve and RV-PA conduit replacement. For the cholecystitis he had a cholecystostomy with planned cholecystectomy 6 weeks after the initial presentation of cholecystitis.

During his course he developed acute kidney injury, post-op atelectasis, hypokalemia, and hyperbilirubinemia which all resolved. He developed anemia secondary to post-operative bleed and hemorrhoids; he was transfused during the admission and was placed on daily iron on discharge. He also developed anxiety regarding his health and was started on an antidepressant. One month follow-up EKG showed a stable right bundle branch block, and cardiac function on TTE was improved.

Of note, in retrospect, it is believed that his original source of bacteremia was from skin lesions that he had developed a few weeks prior to onset of his symptoms.

DISCUSSION

The diagnosis of IE is often not a straightforward one. While many cases may be preceded by an infection, a large fraction of the patient population has no known preceding source. Presentation of this disease covers a large spectrum extending from fever, new-onset murmur, anemia and thrombocytopenia to septic emboli affecting many organ systems, for example the lungs, brain, kidneys, skin, spleen and the musculoskeletal system (4).

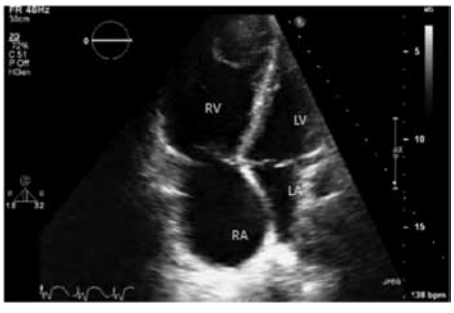

Fig: 2 – Apical 4 chamber view showing severely dilated right atrium (RA) and right ventricle (RV).

Viridans group streptococci cause approximately half of all cases of community-acquired native valve IE not associated with intravenous drug use (5). In the 19th century, the association between bacteremia and surgery, poor oral hygiene and dental extraction was discovered, and as a result the AHA guidelines recommended antimicrobial prophylaxis to prevent IE in patients with underlying cardiac conditions who underwent bacteremia-producing procedures (6). However, overview of published evidence suggests that, of the total number of cases of IE that occur annually, an exceedingly small number are caused by bacteremia-producing dental procedures. Accordingly, only an extremely small number of cases of IE might be prevented by antibiotic prophylaxis even if it were 100% effective. The vast majority of cases of IE caused by oral microflora most likely result from random bacteremias caused by routine daily activities, such as chewing food, tooth brushing, flossing, use of toothpicks, use of water irrigation devices, and other activities (7-9). Other primary sources include pharyngitis or skin lesions (10).

The most recent guidelines have therefore limited the population that is recommended to receive IE prophylaxis to those with cardiac conditions with the highest risk of adverse outcome. These include patients with a prosthetic cardiac valve or prosthetic material for cardiac valve repair, previous history of IE, unrepaired cyanotic congenital heart disease including palliative shunts and conduits, completely repaired congenital heart defect with prosthetic material or device (whether placed by surgery or by catheter intervention) during the first 6 months after the procedure, repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization) and cardiac transplantation recipients who develop cardiac valvulopathy.

The antibiotic for prophylaxis should be administered in a single dose before the procedure. If the dosage of antibiotic is not administered before the procedure, the dosage may be administered up to 2 hours after the procedure. However, administration of the dosage after the procedure should be considered only when the patient did not receive the pre-procedure dose. All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa qualify for prophylaxis.

Amoxicillin is the preferred choice for oral therapy because it is well absorbed in the GI tract and provides high and sustained serum concentrations. For individuals who are allergic to penicillins or amoxicillin, the use of cephalexin or another first-generation oral cephalosporin, clindamycin, azithromycin, or clarithromycin is recommended. Because of possible cross-reactions, a cephalosporin should not be administered to patients with a history of anaphylaxis, angioedema, or urticaria after treatment with any form of penicillin, including ampicillin or amoxicillin. Patients who are unable to tolerate an oral antibiotic may be treated with ampicillin, ceftriaxone, or cefazolin administered intramuscularly or intravenously.

CONCLUSION

IE continues to be an uncommon yet potentially fatal illness with high morbidity and mortality. However, many of the previously believed risk factors and preventive strategies are no longer believed to be true. The latest guidelines restrict IE prophylaxis to individuals with high risk of adverse outcomes.

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A Case Series:

Wolf Hirschhorn Syndrome (WHS)

by Surya Jacob M.D., Shannon Herrick M.D., Mubariz Naqvi, M.D.

ABSTRACT:

Wolf–Hirschhorn Syndrome is a contiguous gene deletion syndrome involving variable size deletion of the short arm of chromosome 4 at 4 p 16.3 region. It is characterized by specific pattern of craniofacial features, delayed growth and development, intellectual disability and seizures. We present 2 cases of Wolf Hirschhorn Syndrome in Amarillo, which were evident only by intrauterine growth restriction during prenatal period, found to have multiple physical defects at birth and on further genetic evaluation diagnosed to have Wolf Hirschhorn Syndrome. Our cases are presented with the purpose of increasing clinical knowledge and helping clinicians to facilitate the management of children with WHS with better understanding of the various features associated.

INTRODUCTION

Wolf-Hirschhorn Syndrome, also called 4 p deletion syndrome, is a congenital malformation syndrome caused by deletion of the short arm of chromosome 4 (4p-). The prevalence of WHS is 1:20,000 to 1:50,000 births; it is more common in females, with a male to female ratio of 1:2. WHS is characterized by a specific pattern of craniofacial features called the "Greek warrior helmet-like facies" including microcephaly, broad flat nasal bridge, high forehead, hypertelorism, highly arched eyebrows, dysplastic ears, pre-auricular skin tags, short philtrum and micrognathia. Other prominent features include prenatal and postnatal growth delay, intellectual disability, seizures, midline defects in the brain, heart, palate or genitalia, congenital heart defects, renal anomalies, and immunological deficiencies. We present 2 cases of Wolf-Hirschhorn Syndrome(WHS), which was evident only by intrauterine growth restriction during the prenatal period in a baby, who was found to have multiple physical defects at birth and on further genetic evaluation diagnosed with WHS.

CASE REPORT I

Initial presentation:

Early term (37 weeks) female was born to a 37 year old Hispanic G3P2A1 mom via elective repeat C-Section. Mom was type 2 diabetic and was on glyburide during the pregnancy. The onIy complication during the prenatal period was intrauterine growth restriction. Pertinent physical findings at birth were: small for gestational age with birth weight of 2034 g (3%), microcephaly with head circumference of 30 cm (2%) and birth length of 45 cm (15%) which was within normal range. She had frontal bossing, broad nasal root, hypoplastic right ear, bilateral cleft lip and cleft palate, mild hypotonia, moderate PDA with left to right shunt, small left kidney, sacral dimple and multiple cutaneous hemangiomas. She also had transient tachypnea of newborn (resolved) and poor oral feeding due to cleft lip and palate during her NICU course.

Diagnosis:

Chromosomal microarray revealed 8.18 MB sized terminal deletion of 4pter à4p16.1 consistent with WHS.

Progress at 4 years of age:

She suffers from failure to thrive and is currently on G-tube feeds. For the developmental delay she is receiving Speech, Occupational, and Physical therapy. She is status post bilateral cleft lip repair which was done at 5 months of age and status post cleft palate partial repair which was done at 2 years of age. She has refractory seizures which presented initially with febrile seizures at 6 months of age then developed into convulsive and non-convulsive types. She is currently managed with ethosuximide and clobazam but never been seizure free. Genetic counseling was done. The maternal chromosomal analysis excluded 4p deletion. Paternal chromosome analysis is still pending. If dad carries translocation, the couple has 10% risk of future children with WHS and 40% risk for miscarriage. Pre-implantation genetic or prenatal diagnosis in future pregnancies has been recommended.

CASE REPORT II

Initial presentation:

37+2 weeks female born to 26 year old Caucasian G1P1 mom via vaginal delivery. The only complication in the prenatal period was intrauterine growth restriction. The pertinent physical findings at birth were small for gestational age with birth weight of 1920 g (1%), microcephaly with head circumference of 28.5 cm (1%) and birth length of 46.5 cm (34%) which was within normal range. She had misshapen cranium with frontal bossing, left eye with small rudimentary globe (microphthalmus), right optic nerve glioma, broad nasal root, right preauricular pit, mild retrognathia, generalized hypotonia, PDA with bidirectional shunt, small atrial septal defect, moderate bilateral hydronephrosis, cervical butterfly vertebrae, deep sacral dimple, spina bifida occulta, lateral drift of toes of left foot, rudimentary nuchal skin and multiple cutaneous hemangiomas.

She also had left pneumomediastinum associated with respiratory distress syndrome (resolved) and poor oral feeding during the NICU course.

Diagnosis:

Chromosomal microarray revealed 24.89 Mb sized terminal deletion in the short arm of chromosome 4, with a breakpoint at p15.2, consistent with WHS.

Progress at 7 months of age:

She suffers from failure to thrive due

to poor oral feeding and is on G-tube feeds currently. For neurodevelopmental delay she is receiving speech, occupational, and physical therapy. She is anticipating prosthetic left eye placement for the microphthalmia. Bilateral tympanostomy tubes were placed for middle ear effusions, and she is being following up pediatric ENT for failed left ear hearing screen. PDA closed spontaneously but the small ASD persists. The multiple cutaneous hemangiomas are resolving on propranolol. Immunoglobulins A, E, and G deficiencies were revealed on further evaluation. Genetic counseling and parental chromosomal analysis is still pending

>75% – Wide nasal bridge – High-arched eyebrows – Widely spaced eyes – Microcephaly – Distinct mouth – Short philtrum – Micrognathia – IUGR/post natal growth deficiency – Intellectual disability – Hypotonia – Muscle hypotrophy – Seizures – Feeding difficulties – Abnormal ears	50-75% – Distinctive EEG abnormalities – Skeletal anomalies, skin changes, craniofacial asymmetry – Abnormal teething – Ptosis – Antibody deficiency 25-50 % – Heart defects – Hearing defects – Eye/optic nerve defects, cleft lip, structural brain anomalies – GU tract defects < 25% – Liver/gall bladder/gut/diaphragm/ esophagus/lung/aorta anomalies

• *Frequency of Clinical features of WHS*

DISCUSSION:

Wolf Hirschhorn Syndrome is a contiguous gene deletion syndrome involving variable size deletion of the short arm of chromosome 4 at 4 p 16.3 region. 87 % of cases are caused by a de novo deletion (~80% of which involves the paternal chromosome). In a small percentage of people, WHS is associated with unbalanced translocation or ring chromosome 4 as well.

Prominent features of Wolf-Hirschhorn Syndrome include growth deficiency which includes prenatal and postnatal growth deficiency, short stature and slow weight gain (main causes include oral facial clefts, difficulty in sucking, poorly coordinated swallow with consequent aspiration, and gastroesophageal reflux and seizures). Ninety percent of affected children will have onset of seizures within first 3 years of life (with peak incidence around 6-12 months), and often triggered by fever (even of low degree). The most common seizures are generalized tonic-clonic seizures, tonic spasms, complex partial and clonic seizure. Other features include global developmental delay and generalized hypotonia associated with muscle hypotrophy of lower legs. Cognitive abilities and adaptive behavior skills are more severely impaired in WHS children compared to children with other deletions. In addition, WHS involves skeletal anomalies and abnormal tooth development, congenital heart defects including ASD, VSD, pulmonary stenosis, and PDA associated with aortic insufficiency, genitourinary (vesico-ureteral reflux, horseshoe kidney, uni-bilateral renal agenesis, cystic dysplasia or hypoplasia, hypospadias with uni or bilateral cryptorchidism in males, clitoral aplasia or hyperplasia, streak gonads and absent uterus or vagina (in females), conductive hearing loss, and antibody deficiencies.

Management:

There is no specific treatment. The mainstay of management is supportive care including physiotherapy, occupational therapy and speech therapy for developmental delay, seizure control, treatment of feeding problems and enrollment of patients in individualized rehabilitation program. Genetic counseling for parents and antenatal diagnosis for future pregnancies is recommended as well.

Natural History:

35% of patients die during the first year of life due to congenital heart defects. The first 2 years of life are often complicated by respiratory tract infection, failure to thrive and refractory seizures producing a mortality rate of 21%. The main causes of mortality include lower respiratory tract infections, multiple congenital anomalies, sudden unexplained death and congenital heart disease. The life expectancy is similar to that of persons with seizure disorders and developmental disability in the absence of major malformations.

CONCLUSION:

We should consider the diagnosis of Wolf-Hirschhorn syndrome in the setting of IUGR, especially when an infant presents with multiple birth defects and craniofacial features. An accurate identification of such patients can lead to the organization of the most appropriate laboratory testing, to the prediction of the prognosis with relative certainty, to the development of the most appropriate health maintenance and educational plans and to referral of the patient and the family to support groups..

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Fullminant Shock Secondary to a 10cm Cardiac Mass in a Young Male with Symptoms of Dyspepsia

by Nathuja Salagundla M.D., Juan Pablo Garrido M.D., Shayan Siddiqui M.D., Asadullah Mirza M.D.

INTRODUCTION

Cardiac tumors, either benign and malignant, represent rare diseases, and data on both management and outcome are limited. It is suggested that benign tumors account for more than 75% of primary heart tumors. These tumors do not usually metastasize, but they can have catastrophic effects due to weakening of cardiac structure and function, including precipitation of arrhythmias, embolism and acute heart failure.

CASE

A 43-year-ols Hispanic male with no significant past medical history presented to ED complaining of shortness of breath, abdominal fullness described as dyspepsia, and intermittent chest fullness. He reported not being able to not lie on his right side because of fullness in chest and abdomen. In addition, he reported near syncopal episodes for the last 2 months with dizziness, lightheadedness and orthopnea without chest pain. The patient had no history of any cardiac issues.

Initial vitals were all normal. Physical examination revealed weak S1 and S2. In the second intercostal space along the left sternal border, a crescendo-decrescendo harsh systolic murmur was appreciated. Lung exam revealed decreased breath sound in the bases bilaterally, with no wheezes or crackles. Initial laboratory workup was unremarkable. CT abdomen and pelvis with contrast revealed a large intracardiac filling defect, anasarca with moderate pericardial effusion, moderate ascites and minimal left pleural effusion.

Patient was admitted to ICU and cardiology was consulted immediately. A transesophageal echocardiography showed a mobile 10 cm x 4 cm mass that seemed to originate from the intra-atrial septum and to be adherent to the aorta with extension into the right ventricle. Initial impression was that the mass could be myxoma. Patient started to have difficulty breathing and became hemodynamically unstable. He was initially resuscitated with intravenous fluids but did not respond. He was intubated, and vasopressors and inotropes were started. Cardiothoracic surgery was consulted, but while arrangements for transferring to tertiary facility were being made, the patient passed away the next morning.

In short, a young patient with no PMH presented with vague symptoms of chest and abdominal fullness and developed fulminant cardiogenic shock within a short time due to a large intracardiac mass.

DISCUSSION

Cardiac neoplasms have the potential to remain clinically silent until they reach an advanced stage, limiting therapeutic options especially for those with malignant transformation. The majority of primary cardiac tumors are benign, with more than 80% being myxomas in various locations, and dyspnea being the most common reason for initial clinical consultation (as in this patient).

The signs and symptoms of cardiac tumors generally are determined by the location of the tumor and not by its histopathology. Cardiac tumors may cause symptoms through a variety of mechanisms: embolization (which is usually systemic but can be pulmonic), obstruction of the circulation through the heart or heart valves (which usually produces symptoms of heart failure), direct invasion of the myocardium (usually causing impaired contractility), arrhythmias, heart block, pericardial effusion with or without tamponade, and constitutional or systemic symptoms.

Tumors arising in the right atrium grow into the atrial lumen and obstruct blood flow, producing hemodynamic changes similar to those seen with tricuspid stenosis. Typical cardiovascular signs and symptoms are those of right heart failure (peripheral edema, fatigue, ascites, hepatomegaly, and prominent "a waves" in the jugular veins). Myxomas are the most common tumors of the right atrium; however, sarcomas (and in particular angiosarcomas) have been reported to arise from the right atrium.

When a probable diagnosis of myxoma has been made, early resection is required because of the risk of embolization or cardiovascular complications, including sudden death. The results of surgical resection are usually very good, with most series reporting an operative mortality rate below 5 percent.

This patient developed cardiogenic shock secondary to right sided heart failure in the setting of a large right atrial mass, most likely a myxoma, causing multi organ failure due to poor stroke volume and cardiac output.

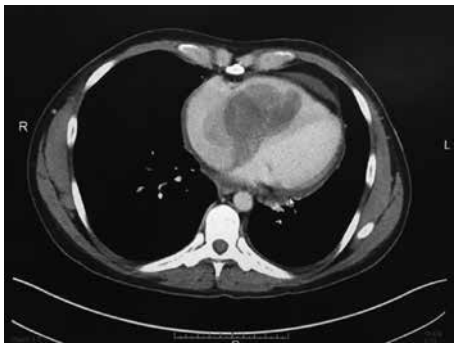


Figure 1 – CT with contrast

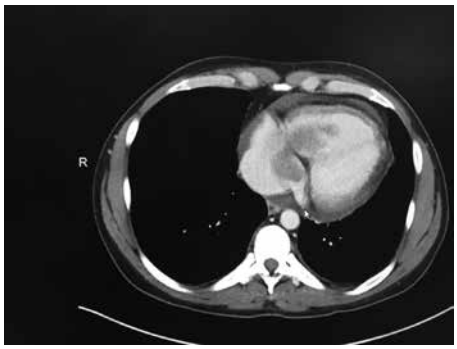
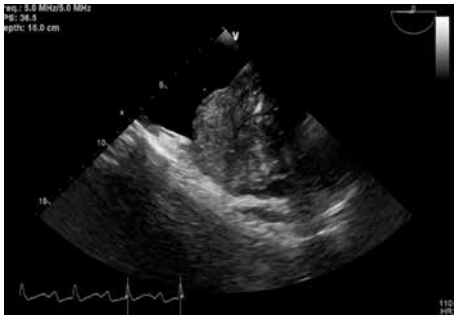
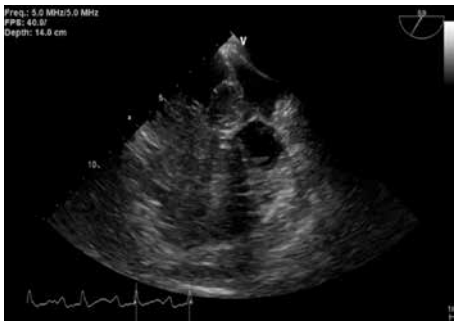


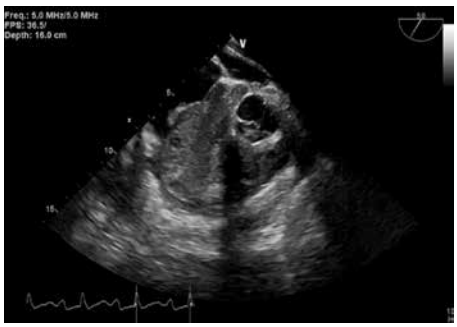
Figure 2 – CT with contrast



TEE 1 – Transesophageal echocardiogram



TEE 2 – Transesophageal echocardiogram



TEE 3 – Transesophageal echocardiogram

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In Memorium

Dr. Luther Nelson, Radiologist,

died June 29, 2017 at the age of 91.

He was a member of the Potter-Randall County Medical Society for 39 years.

Cardiac Salmonellosis

by Jenny K. Nguyen, M.D., Kamel Azhar, M.D., David Pearson, M.D., Evelyn Sbar, M.D., John Slaton, D.O.

INTRODUCTION:

Salmonella is a gram-negative, facultative anaerobic bacillus. Non-typhoidal *Salmonella,* such as *Salmonella enteritidis*, usually causes a self-limiting gastroenteritis secondary to food contamination of products like chicken or eggs. Extra-intestinal infection is possible but rare. Of high concern, bacteremia may occur with cardiac manifestations and may result in high morbidity and mortality.

CASE REPORT:

A 24 year old African American male with no past medical history presents to the ER for abdominal pain for 14 days. A few days after his pain started, he went to an urgent care, where he was diagnosed with colitis and started on Cipro and Flagyl for 10 days. However, after beginning antibiotic treatment, he had no improvement and started having diarrhea; so he went to clinic. Stool studies were positive for *Salmonella* and completion of previous antibiotic course was recommended. Patient went to ER on day 8/10 of antibiotic course as he still had no improvement. Abdominal pain was epigastric and in RUQ. It was a 6-7/10 on the pain scale. Patient denied fevers, nausea, vomiting, jaundice, or pruritus. Meds: Cipro and Flagyl. Allergies: NKDA. Negative medical/surgical/social/family history. Physical exam: BP 143/72, HR 93, RR 23, T 101.0ºF, SpO2 100% RA. Lungs: CTAB with good air entry. Cardiovascular: tachycardia, parasternal heave and friction rub but otherwise normal rhythm and normal S1 and S2. GI: abdomen soft, nondistended, RUQ and epigastric tenderness, BS normal. Pertinent labs: WBC 7.4, CMP normal, CRP 4.685, troponins negative, blood cultures negative, stool studies/cultures negative. Imaging: CXR showed interval development of cardiomegaly. CT abdomen/pelvis with IV contrast showed moderate to severe pericardial effusion. EKG showed sinus rhythm and nonspecific T wave changes. Echocardiogram showed EF 60-65%, abnormal diastolic function, and large pericardial effusion >750ml with early signs of tamponade. RUQ ultrasound and HIDA scan negative.

Serogroup	Serotype	Manifestation
A	S. paratyphi A	Enteric fever
B	S. paratyphi B	Enteric fever Gastroenteritis
B	S. typhimurium	Gastroenteritis
B	S. heidelberg	Gastroenteritis Bacteremia
C	S. paratyphi C	Enteric fever
C	S. choleraesuis	Bacteremia
C	S. newport	Gastroenteritis
D	S. typhi	Enteric fever
D	S. enteritidis	Gastroenteritis
D	S. dublin	Bacteremia

Figure 1.
Different strains of *Salmonella* and their common manifestations.

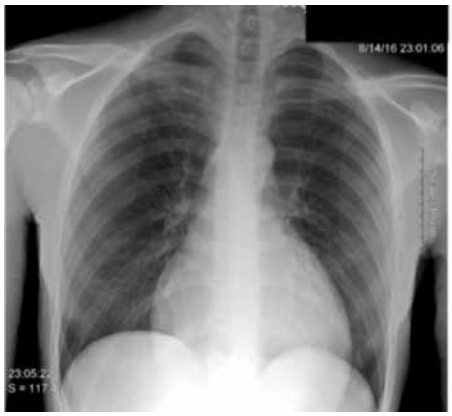


Figure 2.
Chest x-ray of patient shows cardiomegaly. Note the globular enlargement/water bottle shape of the heart. This is indicative of a pericardial effusion.

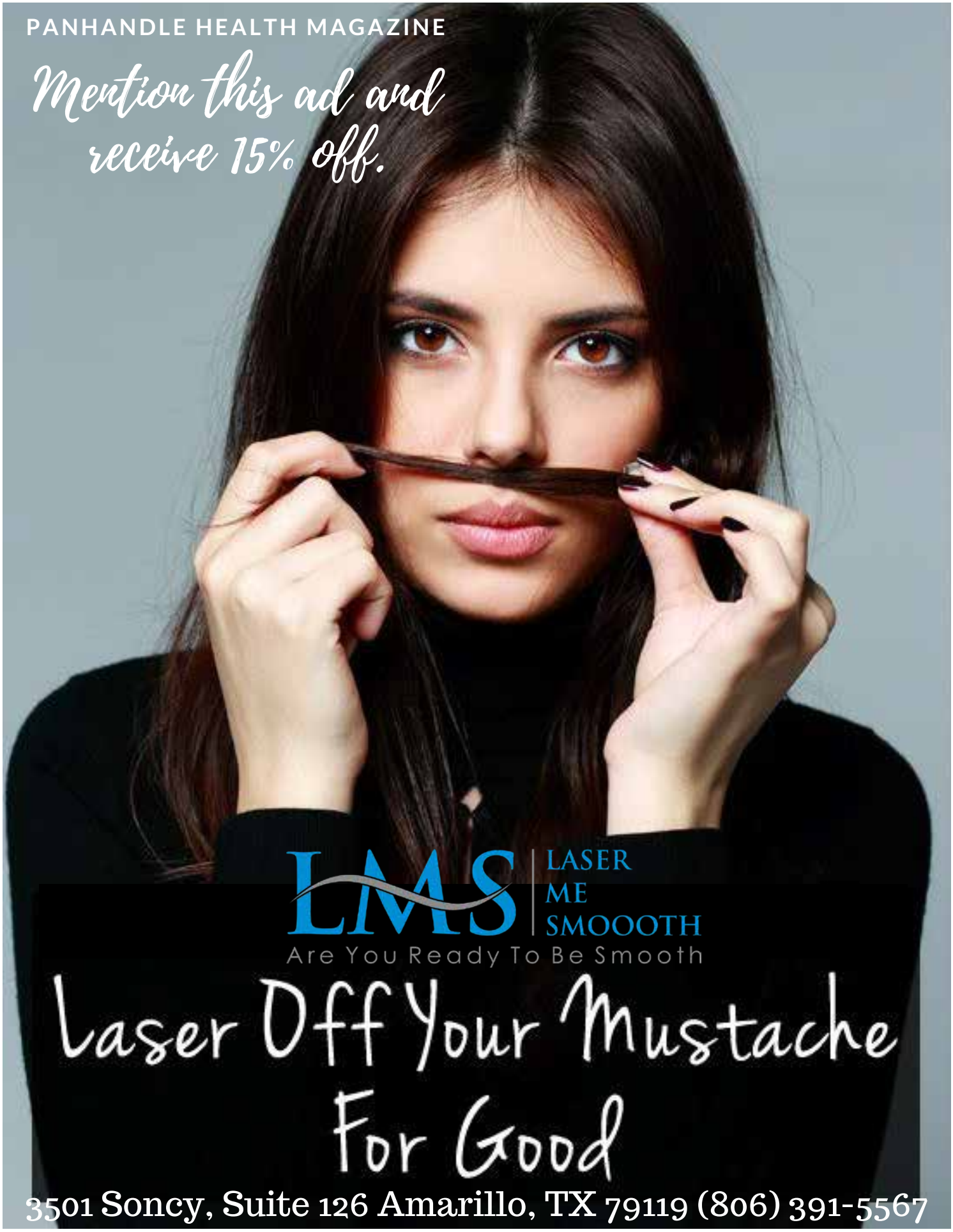
Hospital course: Since he failed outpatient therapy on Cipro and Flagyl and continued to have diarrhea that was previously positive for *Salmonella*, he was started on ceftriaxone. Cardiology was consulted and high dose aspirin was added with the diagnosis of pericarditis with pericardial effusion secondary to *Salmonella*. Pericardiocentesis was not performed, as patient was hemodynamically stable. RUQ pain was worked up but no gallbladder etiology uncovered. GI was consulted, as patient's diarrhea did not improve. EGD showed gastritis and prepyloric ulcer that was most likely from the high dose NSAIDs. Carafate and Protonix were started. He was discharged to a Long Term Acute Care facility for close surveillance and to complete a total of 14 days of ceftriaxone. Pericardiocentesis was performed 14 days later. Fluid cultures were negative, as expected after his extensive antibiotic use.

DISCUSSION:

Non-typhoidal *Salmonella* usually causes a self-limiting gastroenteritis. It can cause bacteremia in about 3-8% of cases. Cardiac salmonellosis is very rare and only occurs in approximately 1-5% of cases. There have only been 19 reported cases of cardiac salmonellosis according to *Texas Heart Institute Journal* and *Annals of Medicine and Surgery*. Appropriate treatment includes aggressive antibiotics along with corresponding cardiac surgery depending on the presentation. Case reports have shown antibiotic sensitivities/effective treatments with ceftriaxone, ciprofloxacin, levofloxacin, gentamicin, amikacin, ampicillin, and ceftazidime. Echocardiogram plays a central role in diagnosis of cardiac involvement and further classifying the extent of involvement. Surprisingly, patients with non-typhoidal *Salmonella* bacteremia or cardiac salmonellosis don't always have gastrointestinal symptoms on presentation. It is important to note that cardiac salmonellosis has high mortality, so it is imperative to have the patient promptly treated and under close surveillance.

CONCLUSION:

Non-typhoidal *Salmonella,* such as *Salmonella enteritidis*, usually causes a self-limiting gastroenteritis secondary to food contamination. Cardiac salmonellosis occurs in approximately 1-5% of cases. There is high morbidity and mortality in patients infected with *Salmonella* that have cardiac involvement, so prompt treatment with antibiotics and appropriate cardiac surgery is recommended.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Intraventricular Neurocysticercosis Causing Acute Hydrocephalus

by Nethuja Salagundla M.D., Shayan Siddiqui M.D., Asadullah Mirza M.D.

INTRODUCTION:

Cysticercosis is a neglected parasitic infection caused by the larval form of the pork tapeworm *Taenia solium*. Neurocysticercosis is the most serious clinical manifestation of cysticercosis and is the leading cause of acquired epilepsy in disease-endemic counties, accounting on average for 29% of persons with epilepsy. It has been increasingly recognized as a cause of severe and preventable neurologic disease in the United States.

CASE PRESENTATION:

A 50 Y/O Hispanic male with no known past medical history presented with chief complaint of headache. Patient mentioned that he had had headaches for long time in the past but they usually resolved with ibuprofen. A day before presentation, he started to have a headache in the morning and by the time he returned from work, his wife noticed that he was becoming drowsy, started throwing up, and became confused and increasingly lethargic. No other associated symptoms. Social history: patient came to USA 4 years ago, grew up in Mexico, denied smoking, alcohol or drug history. Physical examination: initial vitals were unremarkable. The patient was confused and lethargic with no focal deficits; other systems examination was normal. Initial labs: CBC with differential and CMP normal. Imaging studies showed nonspecific foci of calcifications in the parenchyma and posterior portion of third ventricle which suggested proteinaceous material. Serological studies were positive for *T.Solium.*

Neurocysticercosis was highly suspicious based on symptoms, imaging and social history. Cyst in 3rd ventricle was obstructing drainage of CSF causing hydrocephalus. Neurosurgery was consulted, and immediately performed ventriculostomy with shunt to relieve increased ICP pressure. Initially the patient responded well with improvement in mentation. Infectious disease recommended starting antiparasitic therapy with albendazole and dexamethasone. After 3 days, the patient had persistent headache and developed diplopia. Repeat CT head showed increased dilation of 3rd ventricle and improved transependymal edema. Patient was transferred to tertiary care for further management. He underwent uncomplicated endoscopic removal of intraventricular cyst and was subsequently discharged home in stable condition. On follow up, patient has recovered well and is completely asymptomatic.

DISCUSSION:

Clinical syndromes related to *Taenia solium* are divided into neurocysticercosis and extraneural cysticercosis. Neurocysticercosis can be further divided based on location into subarachnoid, cisternal, parenchymal, intraventricular and spinal forms.

Management of intraventricular neurocysticercosis is distinct from the parenchymal form, as guidelines have not been validated in this subgroup of patients.

Intraventricular neurocysticercosis commonly presents with acute obstructive hydrocephalus secondary to either cyst entrapment in narrow foramina or ependymitis. Headache is the most common presentation along with nausea, vomiting, decreased visual acuity, altered mental status and CN palsies, which were all seen in our patient.

Diagnosis is usually made radiologically; MRI is the most useful modality. The visualization of scolex is diagnostic. If MRI scan does not reveal a cyst and there is suspicion of neurocysticercosis, CT ventriculography or neuroendoscopic exploration may be warranted. The inflammatory reaction in neurocysticercosis varies markedly with parasite location, so interpretation of biochemical and serological tests on blood and CSF in intraventricular disease is problematic, as it does not produce the same inflammatory reaction as intraparenchymal cyst. The current antibody test of choice is an immunoblot assay using *T.solium* antigen on serum or CSF samples. Direct detection of *T.solium* antigen has the advantage of being associated with active infection and disease severity; the best study method is ELISA using a monoclonal antibody against the *Taenia saginata* HP10 antigen. Studies on both serum and CSF have shown a high specificity for viable cysts. Antigenemia predicts the presence of viable cysts in patients who have calcified lesions on CT, making it useful in settings where MRI is not available. In intraventricular neurocysticercosis the antigen levels are often higher, with specificity close to 100%. Serial monitoring with the HP10 assay is a useful way of confirming the disappearance of viable cysts with treatment. Lumbar puncture usually shows CSF pleocytosis below 500 x 10^6 cells per litre and consists mainly of lymphocytes and infrequently eosinophils.

The treatment of neurocysticercosis includes antihelmintic drugs and surgical resection of lesions as well as symptomatic treatment with analgesics, anti-inflammatory drugs and anti-epileptics.

Surgical intervention is usually warranted acutely in intraventricular neurocysticercosis to relieve pressure and remove cysts. Neuroendoscopic cyst resection with ventriculostomy (with or without placement of an external ventricular drain or permanent shunt) has now become the surgical treatment of choice, resulting in fewer complications and higher cure rates; this may be curative even without anthelmintic. Ventriculostomy at the time of procedure is often enough to relieve hydrocephalus

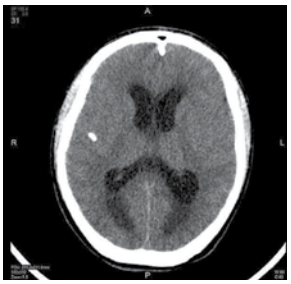


Figure 1:

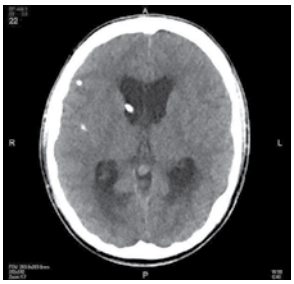
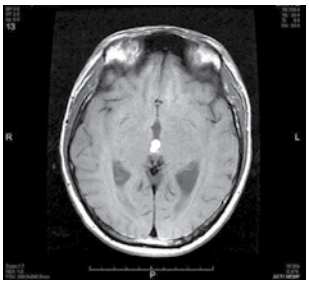


Figure 2:



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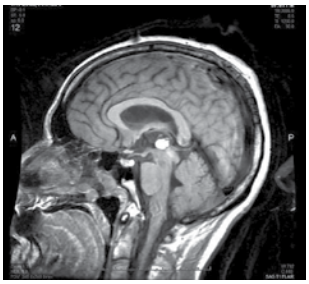

Figure 4:

without external drainage which carries a risk of bacterial infection, although it is still needed in patients with extensive disease and ependymitis. When a shunt is placed, there is a high risk of shunt failure by blockage, and administration of antihelmintic therapy reduces this.

No trials or guidelines are available on the antihelmintic treatment of the intraventricular form of neurocysticercosis, and it has not been determined if its use improves outcome when added to surgery. Most clinicians follow the guidelines for parenchymal neurocysticercosis from the American Academy of Neurology, which recommends albendazole (15 mg/kg/d for 8 d) and either dexamethasone or prednisolone. Praziquantel 50 mg/kg/d for 15 d is an alternative.

Treatment with anthelmintic may lead to a paradoxical worsening of symptoms and may unmask previously undiagnosed neurocysticercosis if treatment is given for another helminthic infection. Treatment should therefore be given under observation in hospital with concomitant corticosteroid administration, and in this setting it is thought to be safe. Corticosteroids have been shown to reduce the plasma level of praziquantel supporting the preferential use of albendazole.

CONCLUSION:

Intraventricular disease occurs in a significant minority of patients with neurocysticercosis, which is an infection no longer restricted geographically to resource-poor regions. The presentation is with features of raised intracranial pressure, and in untreated cases the mortality is high. The diagnosis is based on imaging with MRI, but serological tests, PCR, histopathology and direct surgical visualization are important supplementary diagnostic tools. Treatment is mainly surgical, preferably using a neuroendoscopic technique, but patients should receive antihelmintic treatment with concomitant corticosteroids to reduce incidence of shunt failure and to treat undiagnosed viable lesions elsewhere.

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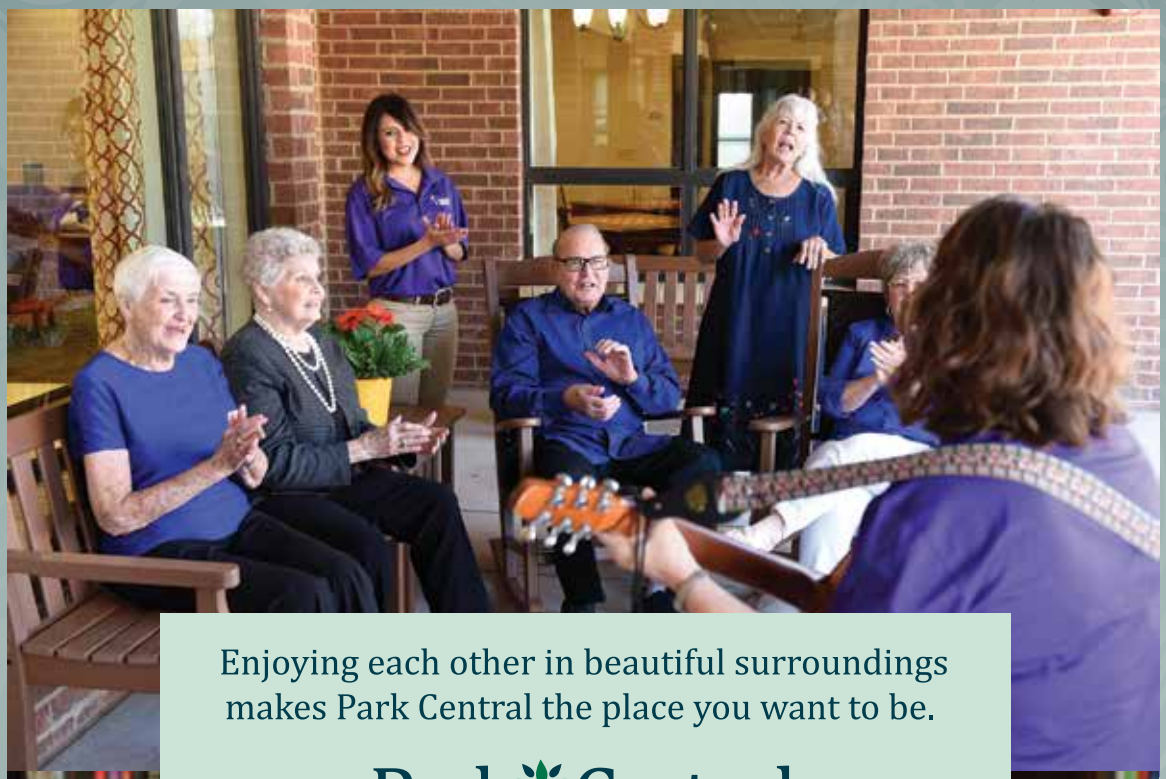

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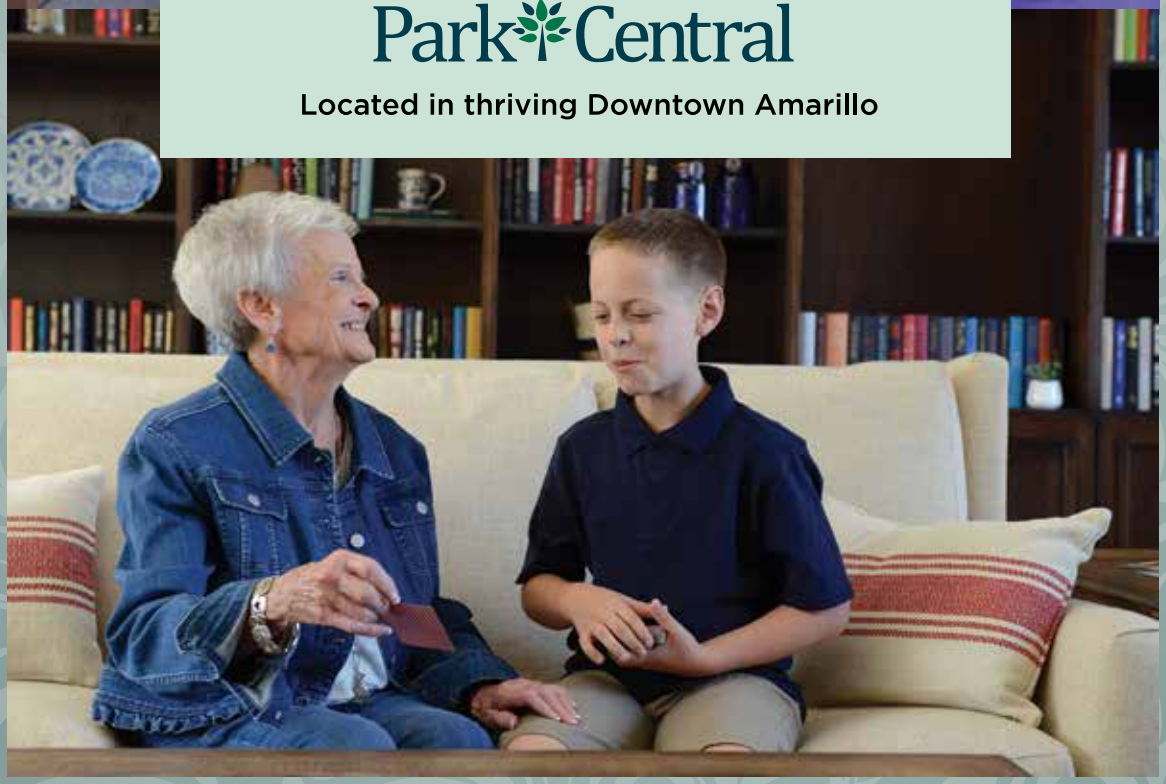

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Uses of Edinburgh (Post Partum) Depression Scale (EPPDS) in Post Partum Patient Care- Delivery to 6 Weeks Post Partum - Uses and Effectiveness

by Sarah Choksi, MS-III, Allison McCabe, MS-III, John Wilson, MS-III,
Teresa Baker, M.D., Robert P. Kauffman, M.D., Paul Tullar, M.D., Julia Wieck, R.N., M.S.N.

Introduction:

Post partum depression is a significant health problem in the post partum period. Untreated, it can result in death of the mother, baby or both. Several depression scales have been described & used to identify patients at risk. Texas Tech OB-GYN Dept. has used Edinburg Depression Scale (Post Partum) at 6 weeks post-partum visit for >10 years, even though PP depression/ PP psychosis peaks at several months post partum. Using some depression scale at 6 weeks post partum is considered "quality indicator" by insurance entities. The same insurance entities have decided that some depression scale administered at the immediate (3- 24 hours) at the delivering hospital is a new requirement as a "quality indicator" and NWTH began having PP nurses administer this 10/1/2016. Interventions are recorded in the medical record(s). This gives a chance to compare the two scores and interventions to see how consistent and how useful they were for outcomes and improvement of patient care.

Materials and Methods:

The target/study population is women who delivered at Northwest Texas Hospital (NWTH) from 10/1/2016 to 12/31/2016 and who did keep their 6-week post partum appointment at the Texas Tech OB Clinic. Women who came to their 6-week postpartum visit were identified via postpartum billing codes. Their EPDS scores, any interventions offered or completed, and any prior history of depression were recorded. The same women's EPDS scores at NWTH as well as any interventions offered or performed and any history of depression documented on their delivery records were recorded. 163 individual patients were identified with 102 completed data sets obtained due to the following exclusions: 19 patients who did not show up for their 6 week postpartum visit, 16 patients who did not have a EPDS screen performed at the hospital, 18 patients who did not deliver at NWTH and 4 patients who were incorrectly coded under the wrong scheduling code. The data was then analyzed to determine if there was a statistically significant difference between EPDS score post delivery and 6 weeks post partum and if the EPDS score is valid at 3-24 hrs. post delivery to predict future high scores on the EPDS.

Results:

The data were then split into two samples: sample 1 composed of the EPDS scores occurring at time of delivery and sample 2 composed of the EPDS scores occurring at the time of the 6 week postpartum visit. The sample size for both samples was 102 with the range of EPDS scores from 0-20 in both data sets. The median score in sample 1 (EPDS at time of delivery) was 3 with a 95% Cl for the median of 2-4 and an interquartile range of 1-6. The median score in sample 2 (EPDS at time of postpartum visit) was a 4 with a 95% Cl for the median of 3-5 and an interquartile range of 2-8. The Wilcoxon test determined 57 positive differences and 30 negative differences between the two sample sets with a large sample test statistic Z of -2.368022 and a two-tailed probability of 0.0179. The Wilcoxon signed rank test 1-tailed probability was performed (all ties were excluded due to nature of the test) resulting in a sample size of 87, a z-value of -2.368 and a p of 0.009.

Conclusion

The data collection for the study was difficult in that some women failed to show up for their 6-week postpartum visit, delivered at a different location or hospital other than NWTH, or did not have a EPDS screening performed at the time of delivery. There is a significant difference in the patient's scores post delivery and at their 6-week post partum visit. More analysis needs to be done to determine if the Edinburgh postnatal depression score is valid after delivery or if it is too soon to predict a future diagnosis of post partum depression. Future research on the effect of treatment after a high EPDS score post delivery on patient outcome can also help lead to quality improvement in patient care.

EDINBURGH POSTNATAL DEPRESSION SCALE (EPDS)

The EPDS was developed for screening postpartum women in outpatient, home visiting settings, or at the 6 –8 week postpartum examination.

Name: _____ Date: _____ Address: _____ Baby's Age: _____

As you have recently had a baby, we would like to know how you are feeling. Please UNDERLINE the answer which comes closest to how you have felt IN THE PAST 7 DAYS, not just how you feel today.

Here is an example, already completed.
I have felt happy:
Yes, all the time
Yes, most of the time
No, not very often
No, not at all

This would mean: "I have felt happy most of the time" during the past week. Please complete the other questions in the same way.

In the past 7 days:

1. I have been able to laugh and see the funny side of things
   As much as I always could
   Not quite so much now
   Definitely not so much now
   Not at all

2. I have looked forward with enjoyment to things
   As much as I ever did
   Rather less than I used to
   Definitely less than I used to
   Hardly at all

*3. I have blamed myself unnecessarily when things went wrong
   Yes, most of the time
   Yes, some of the time
   Not very often
   No, never

4. I have been anxious or worried for no good reason
   No, not at all
   Hardly ever
   Yes, sometimes
   Yes, very often

*5. I have felt scared or panicky for no very good reason
   Yes, quite a lot
   Yes, sometimes
   No, not much
   No, not at all

*6. Things have been getting on top of me
   Yes, most of the time I haven't been able to cope at all
   Yes, sometimes I haven't been coping as well as usual
   No, most of the time I have coped quite well
   No, have been coping as well as ever

*7. I have been so unhappy that I have had difficulty sleeping
   Yes, most of the time
   Yes, sometimes
   Not very often
   No, not at all

*8. I have felt sad or miserable
   Yes, most of the time
   Yes, quite often
   Not very often
   No, not at all

*9 I have been so unhappy that I have been crying
   Yes, most of the time
   Yes, quite often
   Not very often
   No, never

*10. The thought of harming myself has occurred to me
   Yes, quite often
   Sometimes
   Hardly ever
   Never

EDINBURGH POSTNATAL DEPRESSION SCALE (EPDS)
J. L. Cox, J.M. Holden, R. Sagovsky From: *British Journal of Psychiatry* (1987), 150, 782-786.

| continued on page 29

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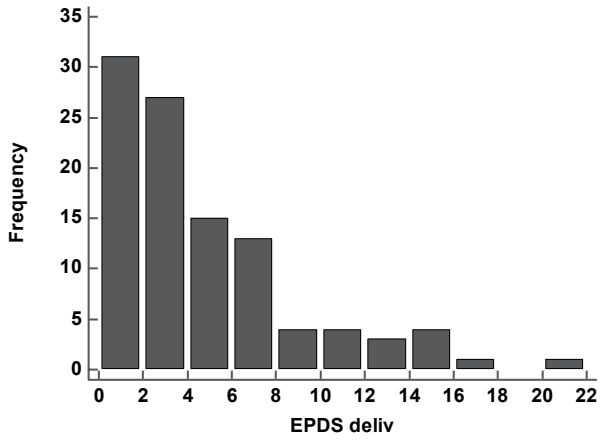
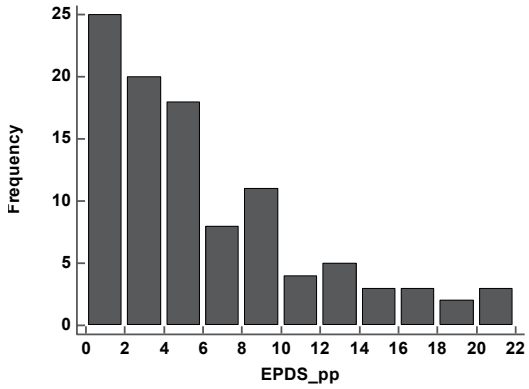
Plot of distribution of Points to Edinburgh Post Partum Depression Scale immediate PP vs 6 weeks PP



This is a distribution plot of Edinburgh Post Partum Depression Score (EPPDS) administered at or <24 hours after delivery. These are from PAIRED data (PAIRED with data below), so should not be considered independently, except as to the overall distribution of the EPPDS totals.



As above, the vertical (Y) axis represents number of people answering at this frequency of EPPDS score, while the horizontal (X) Axis represents the numerical score that these people's answers totaled. As above, these bar graphs could be misleading, as they are from PAIRED data. Use of these graphs together would be appropriate if Mann-Whitney test was used but not the Wilcoxon model.

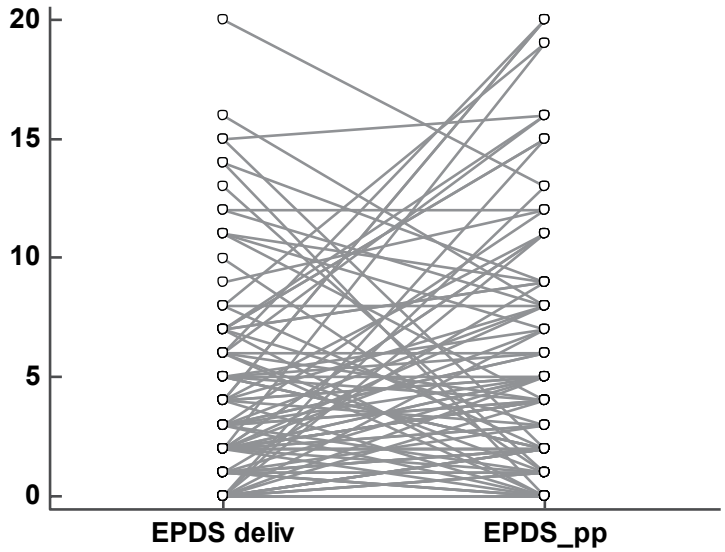


Figure 2: Dot and Line Diagram. This shows the data from the post delivery EPDS compared to the pospartum EPDS using the Wilcoxon test for paired samples

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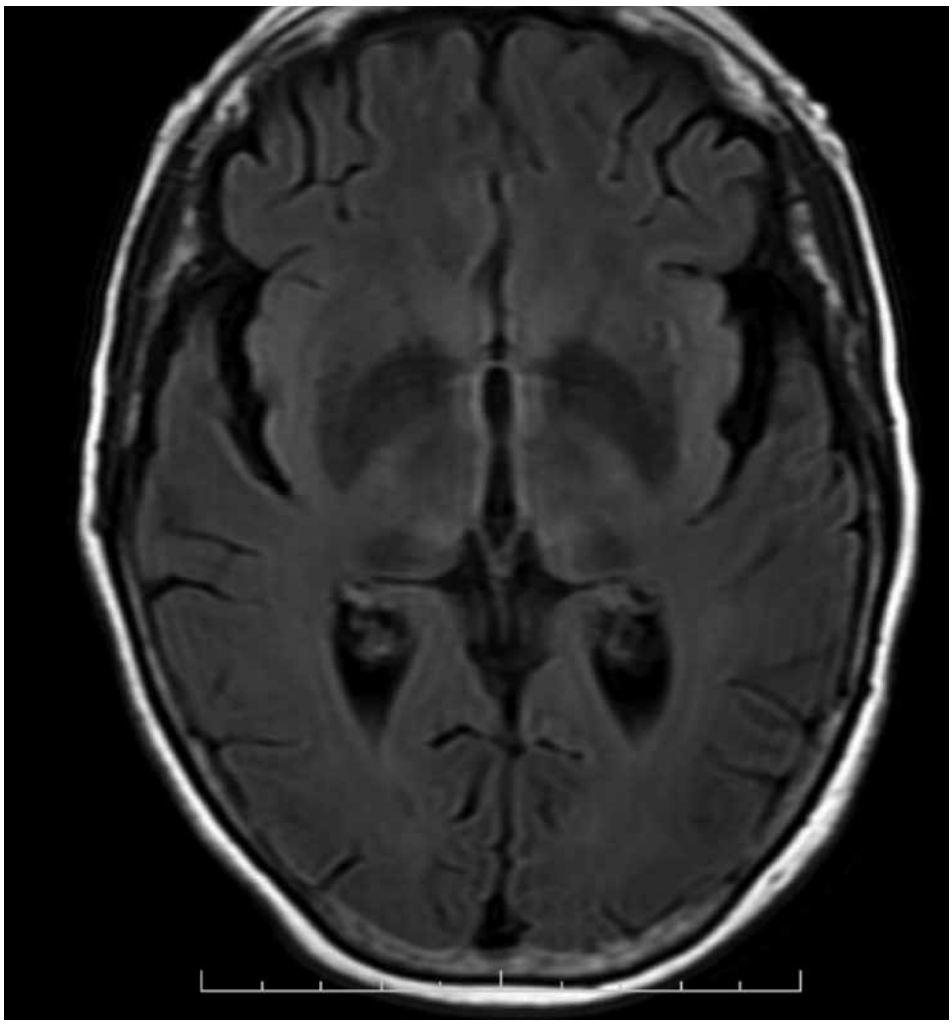
Case Study:

Think Pink: Encephalopathy from Overuse of Pepto-Bismol

by Sarah Ahmed M.D., Alena Prystupa M.D., Robert S. Urban M.D.

Introduction: The heavy metal bismuth (Bi) is an active ingredient of a popular and relatively safe over-the-counter dyspepsia medication, Pepto-Bismol (active ingredient: bismuth subsalicylate). Chronic use of bismuth subsalicylate can rarely lead to bismuth intoxication, manifesting as memory change, confusion, depression, insomnia, ataxia, tremor, myoclonus, and even seizures and coma. We present the case of a woman whose self-medication with large doses of Pepto-Bismol led to a reversible encephalopathy and renal tubular acidosis. A detailed medication history (including dosage and frequency of over-the-counter medications) can help raise clinical suspicion for this rare syndrome.



MRI of brain showing subtle hyperintense signals in thalami bilaterally. There was no associated abnormality on diffusion-weighted imaging. For a full discussion of causes of bilateral thalamic lesions, see Rodriguez (4).

Case: A 61 year old previously healthy woman presented with 2 to 4 day history of confusion, confabulation, hand tremors, and gait ataxia. She was brought to the emergency room by concerned friends. Her initial history proved unreliable, as for instance she gave several conflicting versions of her marital status and family situation. Past medical history was positive for hypertension and self-diagnosed dyspepsia. She had not seen a medical practitioner in several years. Medication: Pepto-Bismol. Allergies: none known. Family history: negative. Social history: occasional alcohol. Initial physical exam revealed BP 160/108 but normal pulse and respiratory rates and no fever. General exam including abdominal exam was normal with the exception of mild copper wiring on funduscopic exam. There were no retinal hemorrhages and no evidence of papilledema. Neurological exam showed that the patient was alert but disoriented; speech was fluent but confabulatory. Strength and reflexes were normal (Babinski sign negative bilaterally) but gait was unsteady and Romberg testing revealed swaying both with eyes open and shut. Multifocal myoclonus was observed.

Initial workup: TSH, RPR, HIV serology and Vitamin B12 level were normal. Urine toxicology, blood alcohol, salicylate, and acetaminophen levels were negative. Chemistry panel revealed serum potassium of 2.1 mEq/L (normal 3.5-5.2), chloride elevated at 108 mEq/L and bicarbonate 16 mEq/L (normal 23-28 mEq/L), suggestive of nonanion gap metabolic acidosis. Urine pH was 7. The serum magnesium level was borderline low at 1.7 mEq/L. The patient's hypokalemia was severe and required over 120 mEq potassium chloride daily for several days before normokalemia was achieved. A diagnosis of renal tubular acidosis (RTA) was suspected and was treated successfully with oral sodium bicarbonate 650 mg TID. Non-contrast head CT showed age-related atrophy and stable sequela of small vessel disease. Brain MRI showed subacute or chronic bilateral thalamic

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hyperdensity and old lacunar infarctions involving basal ganglia (see image).

On further questioning, family members admitted that the patient had been consuming approximately one bottle (480 cc) of OTC Pepto-Bismol (bismuth subsalicylate) every other day for months to alleviate symptoms of dyspepsia. Given the neurological decline combined with new onset RTA and a history of chronic Pepto-Bismol use, bismuth toxicity was suspected. Serum bismuth level of 11 µg/L (normal 1-10 µg/L) and urine bismuth 405.7 µg/L (normal 0.3-4.6 µg/L) confirmed the diagnosis. Toxicology referral suggested a plain abdominal x-ray to assess bismuth burden in the gastrointestinal tract; only a tiny fleck of hyperdense material was seen in the left colon. Therefore, our consultant recommended conservative management in preference to chelation with 2,3 dimercaptopropane-1-sulfonic acid (DMPS). The patient was treated with a proton pump inhibitor and discontinuation of Pepto-Bismol. She was discharged with close follow-up. At time of office visit 4 weeks later she was much improved with resolution of tremor, gait ataxia and ankle clonus, as well as marked improvement of memory. Her serum bismuth level had fallen to 5 µg/L and urine bismuth level had decreased from 405 to 24 µg/L. Electrolytes were normal.

Discussion: Acute or chronic ingestion of toxic doses of bismuth (Bi) can lead to progressive confusion, myoclonus, lack of coordination, and speech disturbance (1, 3). In the CNS, Bi binds sulfhydryl groups and leads to white matter changes, although the thalamic MRI changes seen in our patient have not previously been reported. In the 1970s, over 1000 cases of Bi intoxication in France and Australia were described. These patients developed tremor, ataxia, myoclonus and confusion. In general, recovery occurred weeks to months after Bi discontinuation, although a few patients remained afflicted. Case reports from the United States have been infrequent and sporadic, but these cases have also been characterized by reversible memory deficit, myoclonus, and ataxia (2). In one case, chelation therapy with DMPS actually led to worsening symptoms (5). In general, therefore, management is symptomatic with a gradual but unpredictable improvement of symptoms after discontinuation of bismuth containing product.

Pathologically, Bi nephropathy is associated with proximal tubular damage. Although tubular damage with renal tubular acidosis and electrolyte wasting are common in other causes of heavy metal poisoning (e.g. cadmium), severe hypokalemia and metabolic acidosis, as seen in our patient, have rarely been ascribed to bismuth intoxication. On followup, however, our patient had normal potassium and bicarbonate levels without supplementation, suggesting that tubular damage from Bi was the probable cause.

A suggestive history, combined with high blood and urine bismuth levels, can confirm diagnosis of bismuth toxicity. In our patient, an extensive search for infectious and metabolic cause of encephalopathy was negative. Taking into consideration the history of chronic bismuth containing product use, high blood and urine bismuth levels, along with typical presentation (myoclonus, gait ataxia, RTA), we confirmed diagnosis of bismuth encephalopathy. Our patient was treated conservatively with close followup. She followed a typical course of bismuth encephalopathy with gradual improvement of symptoms at 4 weeks follow up. This case report calls attention to the fact that bismuth toxicity, while rare, should be taken into consideration in a patient presenting with progressive neurological decline and suggestive history. More detailed labeling of this apparently safe and readily available drug as well as raising patient awareness are important in avoiding bismuth toxicity.

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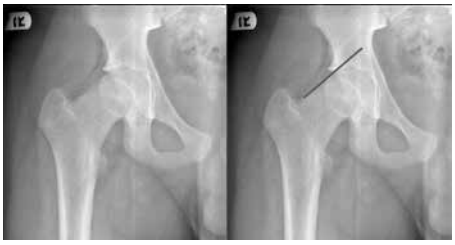
Classical and Atypical Presentations of Slipped Capital Femoral Epiphysis

by Raphael Mattamal, PGY-2, Johnnie Faircloth, M.D., Alison Lunsford, M.D.

Slipped capital femoral epiphysis (SCFE) is an orthopedic emergency caused by damage to or weakening of the growth plate (physis) in the proximal femur, leading to slippage of the overlying femoral head (epiphysis). It is a Salter-Harris type 1 fracture. Classically, it has been associated with the overweight or obese adolescent male with progressive onset of pain and limping. X-rays of the pelvis including AP and frog-leg lateral views show the classic "melting ice cream cone" appearance. Here we present two cases of SCFE, one meeting the classical description and one with an atypical presentation, which is still critical to diagnose in clinic.

Case One: This is a 13-year-old Hispanic male who presented to our sports medicine clinic for a 5-6 month history of isolated R knee pain that worsened after 3 days of football camp. Initial symptoms included a limp for which he was evaluated at a local ER with no imaging studies, and the family was told it was "growing pains".

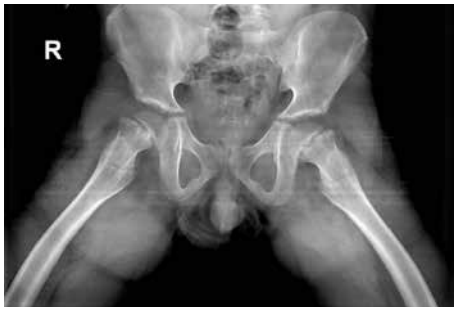
Case Two: This is an 11-year-old Hispanic male who presented to endocrine clinic for abnormal thyroid labs (outside TSH > 600 uIU/mL), but was also reporting a 2 month history of difficulty walking and keeping up with his family members. Initial symptoms included radiating pain down into his thighs, as well as needing to push up onto chairs, desks, or tables when rising from a sitting position.



13-year-old obese male with right-sided SCFE. Note Trethowan's sign ("melting ice cream cone" appearance).

Despite being considered the most common hip disorder in adolescence, SCFE often goes unrecognized (average duration of symptoms ~3.7 months). This is attributable to its varied presentation, which can include isolated or clustered groin, thigh, or even knee pain with or without hip pain due to adjustments to positioning and gait by the patient to avoid discomfort. The involvement of both hips in SCFE can be more common than initially thought, with estimates ranging from 1 in 5 children on initial presentation to 47-80% of children eventually with delayed evaluation / treatment. About 8% of children can have an atypical presentation of SCFE due to weakening of the physis secondary to other conditions such as an endocrine disorder (especially hypothyroidism, growth hormone deficiency, and panhypopituitarism), renal disorders (e.g. osteodystrophy secondary to CKD), Trisomy 21, or radiation therapy for neoplasms. Of note, classical SCFE more commonly affects males and atypical SCFE more commonly affects females. Proper height measurement is useful in SCFE evaluation, as height < 10th percentile has the best positive predictive value for underlying comorbid conditions. Evaluation of thyroid function and growth hormone should be considered in any short children with SCFE. A recent study in Pediatrics from February 2016 of 355 patients showed that underweight or normal weight individuals with SCFE, when compared to overweight or obese patients, were significantly more likely to present with unstable slips and a much shorter duration of symptoms (8.3 weeks versus 20.0 weeks), similar to these two SCFE cases presented.

These two cases seen in the clinic this year provide examples of both classical and atypical presentations of SCFE, and highlight the importance of understanding the underlying risk factors for either presentation. It is critical to fully evaluate knee or thigh pain in adolescents, consisting of a comprehensive history and physical examination including the hip, as well as imaging studies when indicated. SCFE requires prompt referral to orthopedist due to the long term sequelae of a missed diagnosis or delayed treatment, which can include avascular necrosis of the femoral head, hip osteoarthritis, gait abnormalities, and chronic lifelong pain.



11-year-old hypothyroid male with bilateral SCFE.

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Dr. Suhasini Nadesan, Oncologist,

died May 23, 2017 at the age of 71.

She was a member of the Potter-Randall County Medical Society for 8 years.

A Historical Note: The Case Report

by Tracy C. Crnic, M.D.

Abstract:

Objective: This paper discusses the case report as a form of publication in the development of medical literature. It includes a review of historical case reports and discusses the evolution of attitudes regarding the case report itself.

Methods: Pub Med, multiple publications summarizing and discussing the history of reporting in medical literature. Relevant online and printed manuscripts were reviewed and summarized.

Discussion: The case report has evolved through variations of value and content. This review serves to highlight some examples of case reports in history as well as to correlate how they reflect social and ethical issues in medicine at certain periods of history. Medical literary founders (including Hippocrates, Osler, and Galen) through their case reports reflect social and literary attitudes of their respective eras.

Conclusion: Case reports are amongst the earliest known recordings of medical information and development. Understanding the evolution of the format and appreciation of the case report helps to define and explain its use in history and its future in medical literature.

Key words: case report, Hippocrates, Osler, Galen, publication, learning, medicine, documentation, attitudes.

Introduction:

Case reports or studies are a genre of medical literature designed to concisely describe a particular event or observation. Case reports as a medical genre have engendered much debate regarding their place in medical publication [2]. However they remain one of few ways to bring a uniquely observed condition or new etiology to the attention of the scientific community. Reviewing these excerpts reveals broad variations in history regarding the purpose of writing a case report, what it should include or exclude, and its observed value in contributing to medical knowledge. Case reports have been described as one of the best ways for authors to get started in scholarly writing [3, 4], as well as having a diminutive effect on a journal's impact factor [5]. For example, in 1985, the American Medical Association reprinted 51 papers from the Journal of the American Medical Association that had significantly changed the observation of science and practice of medicine during the 150 years of the organization's existence; 5 of these papers were case reports. This serves as evidence of this genre's value. [1, 7] On the opposite end of the spectrum, some journals prefer not to publish cases because of "the low level of general application to the practice of evidence based care" [8]. This paper serves as a brief introduction to the origins and development of the Case Study. Excerpts from publications that predate the writings of Hippocrates along with other historical examples through to modern times are reviewed, and the social norms that helped them are discussed.

Illustration of historical examples of the Case Study:

Some of the earliest examples found in the literature of clinical case reports date as far back as ancient Egypt. The Edwin Smith papyrus from circa 1600 B.C. includes 48 separate cases described as "knowledge gained from practical experience"[6]. The cases describe various surgical interventions and treatments including magical spells. The Hippocratic corpus, dated 400 BC, in "Of the Epidemic" reports several cases emphasizing objective descriptions and clinically relevant findings in a chronological fashion. These vary from the Egyptian cases in that they omit details from the patient's perspective, are purely descriptive, and lack any reference to the supernatural [9, 10]. In the second century, Galenic case reports (estimated around 200 AD) illustrate a different style. His case discussions have more of a conversational tone. His documentation uses the first person and includes specific conversations with other physicians. Hurwitz [9] describes Galen's work as somewhat self-absorbed "that he does not shy away from presenting even his autobiographical history as in one occasion he documents his own abdominal pain". Another example dated near 900 AD written by Rhazes [11] describes a patient who suffered a traumatic neck injury resulting in sensory loss to his two and a half ulnar fingers. He described this as resulting from a lesion to "the nerve located after the seventh vertebra that innervates the little finger, the ring finger including the cutaneous area surrounding them", now known as cervical nerve [8].

Now we glimpse forward to the 17th and 18th centuries. Reports appear to have shifted in theme toward the more dramatic, almost as to appear more entertaining. Titles of reports are livelier, and physician-authors delay the moment of diagnosis or the outcome of the story as if to create suspense. Also emerging in these reports is interpretation of the patient's subjective experiences. This is illustrated in a case describing a three year old girl who survived underwater for a quarter of an hour without drowning, published in Philosophical Transactions in 1739. This style drew negative attention regarding the validity of case reporting, with criticism of the case report by the Royal Society of London for the Improvement of Natural Knowledge as "lacking in plain speech" and as being "threatening to the reporting of scientific observations" [9]. This sentiment carries forward into the 19th century as texts again swing back to avoid the subjective, with more emphasis on technical terminology. The cases specifically avoid inclusion of patient responses or expressions, deeming them unscientific. They are organized into sections that are more clearly delineated including demographic data, an outline of the clinical course, and presentation of autopsy findings. Also included is emphasis on medical terminology, such as that used by Sir William W. Gull in his mid-1800 description of myxedema [12].

How do these examples help us understand medical practices and how do their

| continued on page 38

descriptions reflect social understanding of medicine?

A perusal through these historical examples serves to correlate the art of medicine with the social context of the time. For example, ancient Egyptian medical care was distributed through a hierarchal fashion representative of the social structure of the time. Interventions were a combination of practical measures and magical spells and prayers. Many physicians of the times served as both medical doctors and priests [6]. In Egyptian medicine, disease was viewed as a manifestation of nature; therefore the physician's role was observed as helping nature do its job. Hippocratic reports are more inclusive of specific physical and mental disorders, though some of the conclusions seem somewhat fantastical. The reports take on a more conservative description fitting similar attitudes of the time [13]. Six centuries later Galen (a follower of Hippocratic medicine – the humoral theory) places additional emphasis on detail in discussing clinical, anatomical, and physiological knowledge. Brock depicts Galen as, "an encyclopaedist in whose works we find the essentials of the whole development of medicine" [14]. Galen's methods remained the standard of reporting over the following twelve centuries.

Throughout the Middle Ages, medical knowledge and its documentation becomes more politically influenced by the Catholic Church, which prohibited autopsies and dissections of the body. Near the same timeframe, Millan points out discrepancies in Arabic medical literature between the advocated practice in theoretical texts and actual medical performance, suggesting that more value is gained in case histories than theoretical treatises as they were designated. She goes on to explain her interpretation of care reports [15] as serving several purposes, first as a physician's private collection of experiences, second as a didactic tool for teaching, and finally as a means of self-assertion and promotion. Authors in this time begin to attain notoriety through creation of "elite funded learned medical literature". This is to say, the ruling elite funded learned medical literature in order to propagate a political and financial agenda. This introduced both financial and political incentive to publish.

In the late 1700's, as religious attitudes began to have less effect on the practice of medicine, case reports became more descriptive and inclusive of physical findings in examinations and autopsy reports. The structure of the report also continued to evolve, to a format including a presenting complaint, demographic information, past medical history, details of an examination, initial treatment and observations of its effect, and a summary of the course [16]. Throughout the 18th and 19th centuries case reports became gradually regarded as more acceptable: "the one genre of medical research writing which has been the common stock-in-trade of doctors over the centuries" according to Atkinson [16].

At the end of the 19th century we again see controversy arise, as case studies began to deviate from traditional medical reporting. Examples include the lengthy elaborate descriptions of Freud's course of psychoanalytic treatment interwoven in his reflections and hypotheses about his patients' suffering. Freud experienced self-doubt and anticipated critique before the publication of *Studies on Hysteria* and describes his cases as reading like short stories. He quotes "one might say they lack the serious stamp of science" [17]. Hunter described Freud's narratives as "antidotes and supplements to the standard case history so as to embody the authors' enrichment and extension of their medical fields". His studies and theories remain an integral part of the foundation of psychology and its current practice. The 20th century redirects the standard of case reporting toward neutralization of authors and conventionalization of their structure. Thus evolves the current format of a case report to begin with an introduction, followed by the case and discussion sections as is seen even today [18].

Discussion:

How does this review reflect on case reporting and its place in medical literature?

Understanding the origins of reporting medical observations in literature helps us to develop an appreciation for some of their limitations. It also enables us to observe changes in social mores and their effect on the evolution of medical education. Science has always valued evidence based information in establishing current practice patterns and treatments as they evolve. Case reports serve as descriptions of specific instances and opinions of their authors. They provide new perspectives and serve as a template to introduce previously undescribed findings or combinations of them. They also serve as initial evidence of topics to be further investigated by larger scale studies. Authors such as Freud introduced the science of mental healthcare and have had a great impact on its acceptance as a treatable genre. While these facts do not imply that case reports should replace other forms of scientific reporting, they do illustrate that they retain value as a method of doing so. Sharing these cases succinctly serves to encourage insight from others in our community regarding similar situations. This structure therefore acts to add a branch to the differential diagnosis. As an observer of medicine in the era of the internet, one need not look much further than available collections such as OMIM (Online Mendelian Inheritance in Man) or PubMed (an online index provided by the US National Library of Medicine and National Institutes of Health) to gain an appreciation of the wealth of documentation. Even noting a slight variation of a previously described case opens unimaginable possibilities.

In Summary:

As medical professionals, we must hold ourselves responsible for elevating standards of care not only by teaching through publication but also by striving to learn from them. Studying journals in our own and other specialties of medicine is not only a way to incorporate new material but also helps us apply standards our decision making and treatment protocols. Case reports were among the first line of documentation of medical phenomena. They are only one way of publishing things we have learned, but remain important in beginning to cataloging information. Reviewing the history represented in case reports allows us to put the breadth of medical knowledge and its change over centuries into perspective. As Sir William Osler said, "Always note and record the unusual….Publish it. Place it on permanent record as a short concise note. Such communications are always of value" [19].

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NEW MEMBER SPOTLIGHT 2017

BOARD OF CENSORS REPORT: The following were approved for membership on September 19, 2017

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2101 S Coulter, Amarillo TX 79106

Graduated from University of Texas Southwestern Medical School Center, Dallas TX 2012, Internship and Residency at University of Washington, Completed 2017.

BHASKER, CHAND, M.D. – FM

6715 Sandie Court, Amarillo TX 79109. (Heal the City).

Graduated from University of Kansas School of Medicine, Kansas City KS 1973, Internship at U.S. Public Health Hospital, San Francisco CA, 1973-74, Residency at Texas Tech Health Sciences Center, Amarillo TX 1976-78.

LONGHOFER, LISA, M.D. – HS/ORS

1600 Coulter, B, 79106

Graduated from Texas Tech University Health Science Center, Lubbock TX 2008, Internship and Residency Kansas University, Wichita KS 2008-13, Fellowship, Hand Center of San Antonio (Hand Surgery) 2013-14.

RESIDENTS ADVANCING TO REGULAR MEMBERSHIP:

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MATTAMAL, RAPHAEL, M.D. – PD
SULIMAN, ABDELRAZIG, M.D. – IM

by Tarek Naguib, M.D., M.B.A., F.A.C.P.

Medical Student Average Debt Texas Med (10/1) – Average debt per medical school graduate in 2016 is $190,000.

Top Most Affordable Medical Schools Texas Med (10/1) – Texas A&M is the most affordable medical school in the United States in 2015-16. It is followed by The University of Texas School of Medicine in San Antonio and The Texas Tech University Health Sciences Center School of Medicine in Lubbock.

New Combination Drug for Gout JAMA (10/3) – Marketed as Duzallo, a new oral medicine for gout, a combination of allopurinol and lesinurad, has been approved by FDA. The latter portion in the drug, lesinurad helps excrete uric acid in the urine.

Oxygen Does not Help in Stroke JAMA (9/26) – The preventive use of oxygen did not reduce death or disability at 3 months in patients with acute stroke.

Use, Misuse of Prescription Opioids in US Ann Intern Med (9/5) – More than one third of the US civilian, noninstitutionalized adults reported prescription opioid use in 2015. The use was mainly motivated by relief from pain. A substantial number reported misuse of the drugs.

Ibuprofen Equivalent to Hydrocodone JAMA (11/7) – In patients presenting to emergency department for acute extremity pain, there was no difference in pain reduction at 2 hours among single dose treatment with ibuprofen and acetaminophen or 3 different opioid and acetaminophen combinations. The opioids used included hydrocodone, codeine, and oxycodone.

Federal Task Force to Address Pain Management JAMA (10/24) – The US Department of Health and Human Services, the VA and the Department of Defense are working on forming a task force to devise a strategy for disseminating information about best pain management practices to the medical community.

New Data on Suicide Risk JAMA (10/24) – The suicide rate among female US military veterans has shown a dramatic increase. Twenty US veterans took their own lives each day in 2016. There was a 62% increase in female suicide since 2001. This figure is 2.5 times higher than the rate among nonveteran US women. By contrast, the male US veterans suicide rate has increased by only 30%. Two-thirds of the veterans who died by suicide were aged 50 years or older.

Suicide Rates Higher in Rural Areas JAMA (11/28) – Suicide rates remain consistently higher in rural counties than in urban counties in the US, a trend seen in all sexes, age groups, and racial/ethnic groups.

Lead in Healing Bracelet JAMA (11/14) – Lead poisoning of a 9-month-old baby girl in Manchester, Connecticut, last fall has been traced back to an unexpected source-a metal bracelet. Small spacer beads on the bracelet contained the lead. Neither the manufacturer of the beads or the maker of the bracelet could be identified.

High Mercury Levels Found in Women Around the World JAMA (11/21) – In women of childbearing age in 25 countries across the globe, hair samples revealed high mercury levels. The cause was either contaminated fish as in Pacific islands and small scale gold mining as in Indonesia and Kenya.

Body Mass Index (BMI) Affects Pregnancy Outcome JAMA (11/14) – Among pregnant women in Washington State, low and high BMI before pregnancy were associated with increase of severe maternal morbidity and mortality, compared with normal BMI.

Pet Rat Infected Family in Tennessee JAMA (11/28) – A mother and daughter were infected with Hantavirus traced back to an outbreak among rat breeders in Wisconsin and Illinois. The virus kills about 1% of those infected.

Stool Bacteria Given by Mouth Effective JAMA (11/28) – Fecal Microbiota transplantation worked well to prevent recurrence of *C. Difficile* colitis whether introduced via colonoscopy or by oral capsules.

Death and Cardiac Arrest in US Triathlon Ann Intern Med (10/17) – Death and cardiac arrests during the triathlon are not rare; most have occurred in middle-aged and older men. Most sudden deaths in triathletes happened during the swim segment, and clinically silent cardiovascular disease was present in an unexpected proportion of decedents.

Dr. Robert Gulde, Cardiologist,
died June 6, 2017 at the age of 83.
He was a member of the Potter-Randall County Medical Society for 33 years.

Dr. Walter Dickinson, General Surgeon,
died October 19, 2017 at the age of 78.
He was a member of the Potter-Randall County Medical Society for 45 years.

Anemia

by Tarek Naguib, M.D., M.B.A., F.A.C.P.

What is anemia?

Anemia is defined as having a low hemoglobin level in the blood. Hemoglobin is the substance that gives the blood its red color. It functions as the oxygen carrier from the lungs to the body organs all over. Low hemoglobin levels cause the symptoms of low oxygen delivery outlined below.

How do I suspect having anemia?

The symptoms of anemia (low oxygen delivery to body organs) are somewhat vague but usually are consistent among people. They include generalized weakness and fatigue, and shortness of breath. Occasionally spots before the eyes appear and lack of mental focus is noted with severe cases. The person may appear pale to others. These findings warrant medical attention, as not only may anemia have consequences but also it may herald other underlying conditions.

How to diagnose anemia?

The diagnosis of anemia is usually done by having a simple blood test to check the hemoglobin level. The test is very cheap and can rule out anemia if normal. If low, it will prompt a further search into the reason for the anemia.

What are the causes of anemia?

Anemia can be caused by immediate causes (called acute) as in cases of blood loss or long-standing causes (called chronic) like diabetes, kidney disease, liver disease, cancers, etc. Also, anemia can be hereditary as the young persons with sickle cell anemia and Mediterranean Sea anemia. In addition, certain degree of anemia is expected as the age advances. Also, anemia can take place in simple problems like heavy monthly menstrual outflow and low vitamin levels like vitamin B and folate.

Where did the name come from?

The word origin is self-explanatory: The prefix a- or an- means no or absence of. The suffix -emia means blood. The meaning of the word altogether is missing blood.

How do doctors treat anemia?

Doctors treat acute anemia with blood transfusion as appropriate and work to stop causes of loss. That's why, in mass casualties, it is critical to keep the blood bank stocked and ready to supply hospitals. In chronic cases, transfusion is used as the last resort as the treatment of the underlying condition that caused anemia is the target of medical care.

How can I help management of anemia?

If you have some of the symptoms of anemia (as outlined above) you need to present for medical attention early. Average balanced diet with moderation is helpful but will not correct most of the anemias. So, avoid excess calorie intake since it may cause harmful obesity.

Dr. Cameron Hodges, Surgeon,
died September 25, 2017 at the age of 46.
He was a member of the Potter-Randall
County Medical Society for 5 years.

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Happy Holidays

from the Staff at Potter-Randall County Medical Society

Dr. Constantine Saadeh Introduces

DR. NICOLE DAVEY

Board-Certified Rheumatologist

We are so pleased to have Dr. Nicole Davey-Ranasinghe (ΑΩΑ, University of Nevada School of Medicine) aboard Allergy A.R.T.S.

Dr. Davey did her internal medicine residency at the University of Nevada School of Medicine where she served as chief resident. Following residency, Dr. Davey completed her clinical training with a **fellowship in rheumatology** at Oregon Health and Science University. She has spent the last three years with Centura Health Physician Group in Durango, Colorado.

Board Certified in rheumatology and internal medicine, she brings experience and passion for the management of both common and complex rheumatologic conditions, such as **rheumatoid arthritis, lupus, osteoarthritis, spondyloarthritis and osteoporosis**.

I know Dr. Davey will be a great asset to the patients of Allergy A.R.T.S. and to the Amarillo medical community. **Welcome!**

To make an appointment with Dr. Davey, please call (806) 353-7000

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