SAN ANTONIO UNIFORMED SERVICES HEALTH EDUCATION CONSORTIUM (SAUSHEC) PRESENTS...

SOUTHWEST TEXAS ACP CONFERENCE

FRIDAY, SEPTEMBER 17, 2021

POSTER COMPETITION | ABSTRACT BOOKLET

Clinical Vignettes Breakout 1

Breakout Room #1 Agenda

Time	Presenter	Program	Presentation Number
13:15-13:20	Moderator Remarks		
13:20- 13:26	Alexis Bejcek	Baylor Scott and White-Temple	1.1
13:26- 13:32	Shirley Reed	Baylor Scott and White-Temple	1.2
13:32-13:38	Hannah Shine	Baylor Scott and White Temple	1.3
13:38-13:44	Lina Pedraza	UTRGV- DHR	1.4
13:44-13:50	Dina Hammad	UTRGV- DHR	1.5
13:50-13:56	Sonya Montes	UTRGV- DHR	1.6
13:56-14:02	Olga Lavrynenko	Laredo Medical Center	1.7
14:02-14:08	Eugene Stolow	UTHSCSA	1.8
14:08-14:14	Dennys Jimenez	UTHSCSA	1.9
14:14-14:20	Ariadna Perez-Sanchez	UTHSCSA	1.10
14:20-14:26	Keerthana Pakanati	Baylor Scott and White Round Rock	1.11
14:26-14:32	Rahul Thakkar	Baylor Scott and White Round Rock	1.12

Presentation Number: 1.1

Category: Clinical Vignettes

Title: A Mysterious Case of Elevated Hemoglobin A1c

First Author: Alexis Bejcek

Additional Authors: David Wenkert, MD

Institution: Scott and White Hospital

Abstract: Hemoglobin A1c (HbA1c) is an important tool for diagnosis and management of diabetes mellitus. However, multiple factors can interfere with laboratory assays and lead to inaccurate results. We describe the case of a 50-year-old Hispanic female with hypothyroidism, hyperlipidemia, and hypertension who was referred to the endocrinology clinic for management of type 2 diabetes mellitus. She was initially diagnosed with type 2 diabetes mellitus by her primary care provider based on an A1c reading of 10.3% on the Biorad Variant II High Performance Liquid Chromatography system. Fasting glucose readings at that time were within normal range from 84-91. She was started on metformin 500 mg twice daily, attended diabetes education classes, began training for a 5K race, and started checking blood sugars at home. Repeat laboratory determinations approximately 4 months later demonstrated fasting glucose 91 and HbA1c 10.3% on the same testing platform. The normal fasting readings and lack of improvement in A1c with multiple interventions prompted further evaluation. Additional investigation was completed by fructosamine analysis, chromatography, electrophoresis cascade (Mayo Clinical Laboratories), and mass spectrometry (Mayo Clinical Laboratories). HbA1c results with the Siemens DCA point-of-care analyzer provided results of 5-6% and fasting blood glucose readings were 80-95. Fructosamine was within the normal range at 233-251. Hemoglobin electrophoresis cascade and mass spectrometry confirmed the presence of hemoglobin Wayne variant. Although hemoglobin Wayne is often clinically silent, falsely elevated HbA1c results could lead to unnecessary medical interventions that could cause patient harm. This variation in results highlights the importance of utilizing additional measurements such as glucose readings and evaluating for hemoglobin variants when results are discordant.

References:

1. American Diabetes A. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2020. Diabetes Care. 2020;43(Suppl 1):S14-S31.

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8. Sharma P, Das R. Cation-exchange high-performance liquid chromatography for variant hemoglobins and HbF/A2: What must hematopathologists know about methodology? World J Methodol. 2016;6(1):20-24.

Presentation Number: 1.2

Category: Clinical Vignettes

Title: Late Presenting Complex Femoral Pseudoaneurysm

First Author: Shirley Reed

Additional Authors: Clint Jones DO, Yissela Escobedo MD, Steven Manuel Costa MD

Institution: Scott and White Hospital

Abstract: Background:

Iatrogenic pseudoaneurysms are a rare complication associated with femoral artery access for invasive cardiac and vascular procedures. Early detection and treatment can reduce the risk of a complicated pseudoaneurysm. Signs of this complication include regional discomfort, access site swelling, ecchymosis, bleeding, evidence of a pulsatile mass, or auscultation of bruit or thrill.

Case Description:

This case report describes an eighty-two-year-old male with a history of chronic atrial fibrillation on Xarelto, hypertension, and Parkinson's disease presenting with bleeding from right groin region. Of note, he had severe aortic insufficiency status-post surgical repair in 2009 with recent trans-catheter aortic valve replacement (TAVR) with MANTA vascular closure 9 months before presentation due to bioprosthetic valve degeneration.

Decision Making:

Ultrasound of the lesion was performed which showed a 5.1 cm right common femoral artery (CFA) pseudoaneurysm. CTA Runoff revealed a bilobed 5.1cm right CFA superior aneurysm with surrounding inflammatory fat stranding concerning for a secondary infection. Serial exams noted lesion to be enlarging with worsened surrounding necrosis. Patient was taken emergently for open repair of right common femoral pseudoaneurysm and sartorius myoplasty. Wound cultures from initial operation grew Pseudomonas aeruginosa for which he received ceftazidime IV for six weeks.

Conclusion:

Although iatrogenic pseudoaneurysms have been well defined in the literature, they have often been detected shortly after the procedure, typically within 24 hours or up to 10 days. This case is unusual given a large-size complicated pseudoaneurysm presented months after a TAVR and thorough inpatient evaluation for said complication after the procedure. Another compelling aspect is MANTA vascular closure devices are rarely associated with pseudoaneurysm

formation as seen in this patient. The present case suggests evaluation for iatrogenic pseudoaneurysm would be warranted even months after the procedure if a patient presents with concerning symptoms related to this complication.

References:

1. Webb, Sherrie. Postcatheterization Femoral PSEUDOANEURYSMS. 2019, www.acc.org/latest-in-cardiology/ten-points-to-remember/2019/06/04/10/26/postcatheterization-femoral-pseudoaneurysms#:~:text=Management%20options%20include%20observation%2C%20ultrasound,should%20avoid%20lifting%20or%20bending.

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Presentation Number: 1.3

Category: Clinical Vignettes

Title: A Precarious Case of Pregnancy and PH

First Author: Hannah Shine

Additional Authors: Tasnim Lat, DO

Institution: Scott and White Hospital

Abstract: Pulmonary arterial hypertension (PAH) remains a disease with high morbidity and mortality in pregnant women, despite advances in therapeutics and improved understanding of the disease. Retrospective studies worldwide have reported the current maternal mortality anywhere from 10-30%. Although markedly high considering these are 20-40 year-old women, these statistics are reduced from those of the 1990s which approached 38%-60%. Women with mild pulmonary hypertension or classes outside of Class I tended to have more favorable outcomes. The greatest risk of mortality is in the one-month post-partum state.

This case explores a successfully treated young woman who delivered a viable infant at 28 weeks gestation and the management of her exacerbated PAH during her final weeks of pregnancy. She presented 26 weeks pregnant with no prenatal care, and intermittent use of teratogenic medications including macitentan, and lisinopril, in addition to non-teratogens sildenafil and furosemide. She was started on IV epoprostenol and steroids for fetal development and fetus delivered at 28 weeks via cesarean. She was diuresed and kept on epoprostenol during two-week hospitalization. Transthoracic echo revealed severe right ventricular dilation, right atrial enlargement, severe TR, and RVSP of 120mmHg. After a cesarean and prolonged ICU course, she was transitioned from epoprostenol to sildenafil and discharged in stable condition.

Physicians should strongly counsel regarding the risks of pregnancy in women with PAH, as not only is mortality a concern but also maternal morbidity and the concern of teratogenic effects on the fetus due to the number of drugs classified as teratogens commonly used in the treatment of PAH.

References:

Martin SR, Edwards A. Pulmonary Hypertension and Pregnancy. Obstet Gynecol. 2019 Nov;134(5):974-987. doi: 10.1097/AOG.0000000000003549. Erratum in: Obstet Gynecol. 2020 Apr;135(4):978. PMID: 31599832.

Olsson KM, Channick R. Pregnancy in pulmonary arterial hypertension. Eur Respir Rev. 2016 Dec;25(142):431-437. doi: 10.1183/16000617.0079-2016. PMID: 27903665.

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Presentation Number: 1.4

Category: Clinical Vignettes

Title: Where is all the potassium going? A case report of a Gitelman syndrome in an asymptomatic adult with severe hypokalemia

First Author: Lina Pedraza

Additional Authors: Victor Zamora MD PGY3, Nevin Varghese MD Assistant professor

Institution: UTRGV-DHR

Abstract: Introduction

Gitelman syndrome (GS) is a rare inherited tubulopathy that arises from mutations on a cotransporter gene that encodes the thiazide-sensitive sodium chloride channel (1) It manifests with metabolic alkalosis, hypokalemia, hypomagnesemia, hypocalciuria and a normal blood pressure. The diagnosis is challenging due to the complex presentation and unfamiliarity for most clinicians.(2)

Case report

We present the case of a 37-year-old gentleman with no past medical history brought by the police to the emergency department after suspected overdose of cocaine, amphetamines, and marihuana the day before. Initial workup showed severe hypokalemia of 2.6 mEq/dl, metabolic acidosis, and acute kidney injury, with unknown baseline creatinine. After 12 hours of continued potassium replacement and more than 120 mEq given orally and IV, recheck levels showed a potassium of 2.0 mEq/dl. Refractory hypokalemia raised concerns for potential tubulopathies. Workup evidenced potassium wasting based on potassium/creatinine ratio of >37mmol/g and hypocalciuria with calcium/creatine ratio of 0.086 mg/mg. Plasma renin activity was high. On the third day, nephrology was consulted, and the patient was started on indomethacin and amiloride with the intention to block the distal tubular sodium-potassium exchange pump. On the fifth day of admission recheck potassium was 3.8mEq/dl and magnesium 2.2 mEq/dl.

Discussion

GS can be diagnosed once common causes of hypokalemia and metabolic alkalosis have been excluded. (3) GS continues to be a clinical challenge despite the KDIGO consensus from 2017 regarding this entity. Suspicion of GS must remain high with the evidence of metabolic alkalosis, hypocalciuria and potassium wasting in a clinical scenario with refractory hypokalemia. In our case the use of amiloride improved the potassium wasting(4), the concomitant use of NSAID

was limited due acute kidney injury and no evidence of prostaglandins in urine. (5). Patient was discharged with outpatient follow up.

References:

1. Blanchard, Anne, et al. “Gitelman Syndrome: Consensus and Guidance from a Disease: Improving Global Outcomes (KDIGO) Controversies Conference.” Kidney International, vol. 91, no. 1, 2017, pp. 24–33., doi:10.1016/j.kint.2016.09.046.

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Presentation Number: 1.5

Category: Clinical Vignettes

Title: Looking Beyond the Skin: A Case of Systemic Sclerosis Sine Scleroderma in a Hispanic Woman

First Author: Dina Hammad

Additional Authors: Christine Loftis, Rosa Guedez, Emilia Dulgheru

Institution: UTRGV-DHR

Abstract: Systemic sclerosis (SSc) is an autoimmune multisystemic disease that is characterized by fibrotic and vascular manifestations affecting the skin and visceral organs. Systemic sclerosis sine scleroderma (ssSSc) is a rare entity without obvious skin thickening and accounts for about 10% of the SSc subsets (1).

Case Description: 67-year-old lady with history of interstitial lung disease and Peutz-Jeghers syndrome was referred to the hospital for evaluation of index finger ischemia. She presented with worsening pain and ulceration of the index fingertip that was preceded by exposure to cold temperature.

The patient denied induration or thickening of skin, dysphagia, myalgias or joint pain. Physical exam demonstrated small ulceration of the right index finger, multiple areas of brown macules at the radial aspect of the index fingers bilaterally, no synovitis, and no sclerodermatous changes of skin. Pulses were intact in upper extremities. Laboratory studies showed sedimentation rate of 42 u/mL, a positive ANA with an ELISA titer of 16 (Ref range less than 1) and SS-A titer 206, with negative serology for centromere B antibody, double stranded DNA antibody, smith antibody, Scl-70 antibody, SS-B antibody, U1RNP and rheumatoid factor. Arterial Doppler ultrasound of upper extremities was negative. Right subclavian angiography reported no circulation to the distal phalanx of the right index finger. Limb changes were consistent with a diagnosis of secondary Raynaud's phenomenon. Diagnosis was assessed as systemic sclerosis sine scleroderma based on positive ANA, severe Raynaud's phenomenon with fingertip necrosis and history of interstitial lung disease.

Conclusion: Being that systemic scleroderma sine scleroderma is a rare entity, it is important to have a high index of suspicion and understand that cutaneous manifestations and serological markers are not always manifest on initial presentation.

References:

1) Kaur G, Banka S, Meena B, Kulkarni A, Prajapat R, Dhaka J. Sine Scleroderma. J Assoc Physicians India. 2020 Apr;68(4):68-70. PMID: 32610852.

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Presentation Number: 1.6

Category: Clinical Vignettes

Title: A case report of the atypical presentation of Guillen Barre Syndrome with Facial diplegia

First Author: Sonya Montes

Additional Authors: Rosa Guedez Baute, Arleen Delgado

Institution: UTRGV-DHR

Abstract: Introduction: Guillain Barre syndrome (GBS) is an inflammatory disease of the peripheral nervous system and is the most common cause of acute flaccid paralysis, with an annual global incidence of approximately 1–2 per 100,000 person-years1.This uncommon variant of bilateral facial palsy with paresthesias manifests between 1 to 28 days after initiation of symptoms and occurs in less than 1% of all GBS patients2. In this case the patient exhibited a rare presentation of the disease that is worth discussing.

Case description: 29-year-old man with no significant past medical history presented to the emergency department (ED) complaining of worsening gait instability, bilateral weakness of upper and lower extremities, paresthesia on palms and soles, bilateral facial diplegia and shortness of breath. Two weeks prior to the admission the patient had developed flu-like symptoms accompanied by watery diarrhea of unknown etiology. Soon after, he started developing neurological symptoms requiring multiple ED visits, and was treated as Bell's palsy with Acyclovir and steroids. Examination revealed facial diplegia with bilateral ptosis, unable to tighten eyes, decreased overall facial expressions and bilateral symmetrical weakness of upper > lower extremities and areflexia to lower extremities.

Discussion: A lumbar puncture was performed; it revealed albumin-cytological dissociation of cerebrospinal fluid (CSF), dissociation consistent with GBS syndrome, for which plasmapheresis was prescribed and he subsequently improved. Facial diplegia is a rare variant of GBS that presents a very challenging diagnosis to make in the ED due to its wide differential diagnosis. When patients present with bilateral facial palsy it might mimic different diseases, and diverse etiologies should be considered.

Conclusion: We aim to raise awareness of the atypical presentation of GBS as it can be potentially fatal and therefore, early identification is important.

References:

1. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain–

Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671-683. doi:10.1038/s41582-019-0250-9

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Presentation Number: 1.7

Category: Clinical Vignettes

Title: Case-report Euglycemic Diabetic Ketoacidosis in a patient with dehydration on SGLT2 inhibitor

First Author: Olga Lavrynenko

Additional Authors: Hector Santos, MD, Amando Garza, MD, Rayan Qazi, MD, Leopoldo Cobos, MD

Institution: Laredo Medical Center~TX~USA

Abstract: Diabetic ketoacidosis (DKA) is a life - threatening complication of diabetes mellitus (DM) and needs to be diagnosed and treated promptly and aggressively. The classic triad of DKA is: (1) hyperglycemia (blood glucose (BG) more than 250 mg/dl; (2) anion gap metabolic acidosis (pH less than 7.3 and bicarbonate less than 18 mEq/L); and (3) ketonemia. With the advent of novel treatments for DM, specifically the sodium - glucose transporter 2 (SGLT2) inhibitors, DKA can occur with BG levels below 200 mg/dl and has been defined as euglycemic DKA (EuDKA). Due to the absence of hyperglycemia, the diagnosis of EuDKA is more challenging and often is delayed.

This case of EuDKA, in a patient taking a SGLT-2 inhibitor (empagliflozin, 25 mg/daily) and pioglitazone (30 mg/daily), was precipitated by dehydration. This 60-year-old Hispanic male with diabetes presented to the emergency room with gastrointestinal problems: diarrhea, bloating, abdominal pain and cramps, which started 20 days ago. He was admitted with a presumed diagnosis of colitis and dehydration. On admission laboratory evaluation revealed a metabolic acidosis with an elevated anion gap of 18, bicarbonate of 19 mEq/L, and a minimally elevated serum glucose of 146 mg/dL. There was no history of ingestion of alcohol, salicylates, methanol, ethylene glycol and nothing to suggest lactic acidosis. The patient was treated with metronidazole, levofloxacin and lactate ringer. On the following day, he developed a marked increase in the anion gap to 22 mEq/l, with a further decrease in the bicarbonate to 13 mEq/L, and ketones were detected in the serum. The patient was treated for EuDKA in ICU with intravenous insulin and dextrose (to prevent hypoglycemia), with resolution of his symptoms and EuDKA in 3 days.

References:

1.Peters AL et al. Euglycemic Diabetic Ketoacidosis: A Potential Complication of Treatment with Sodium-Glucose Cotransporter 2 Inhibition. Diabetes Care 2015; 38 (9): 1687 – 93. PMID: 26078479

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Presentation Number: 1.8

Category: Clinical Vignettes

Title: Cutaneous T-Cell Lymphoma in an Immunocompromised Female

First Author: Eugene Stolow

Additional Authors: McKenna Boyd, MD, Sadie Trammell Velásquez, MD, FACP

Institution: Univ of Texas Hlth Sci Ctr Prog

Abstract: Introduction

Cutaneous T-cell lymphoma (CTCL) is a lymphoid neoplasm typically diagnosed in the sixth decade of life with an incidence of 0.05 cases per 100,000 people in the United States. We present an 81-year-old immunocompromised female with CTCL to demonstrate diagnostic and management strategies.

Case Description

An 81-year-old female with a history of stage III nodal marginal zone lymphoma in remission and pulmonary sarcoidosis on systemic glucocorticoids, presented with 1 month of progressive dyspnea. She reported a rash for 6 days coinciding with recent termination of her prednisone taper. She denied new medications, sick contacts, fevers, or weight changes.

Physical exam demonstrated diffuse violaceous macules and patches with fine overlying scale on her bilateral arms and legs (Image 1a,b). No lymphadenopathy was present.

Initial WBC count was 13.0 x 10^3/µL. Chest computed tomography noted diffuse multifocal airspace consolidations. She was started on broad-spectrum antibiotics, and steroids were re-initiated per pulmonology.

Dermatology initially suspected leukocytoclastic vasculitis with an infectious trigger (pneumonia) versus cutaneous sarcoidosis. Sputum bacterial and fungal cultures and fungal serologies were unremarkable. Autoimmune work-up was normal. Shave biopsy of a shoulder lesion revealed atypical lymphocytic infiltration consistent with CTCL. Outpatient hematology follow-up was arranged.

Conclusion

CTCL has a broad differential and pathognomonic clinical features are lacking. Diagnosis may be obscured by underlying chronic diseases with mimicking cutaneous manifestations. The lymphomatous variant accounts for 20 percent of cases and excisional biopsy of an involved lymph node is often necessary for histopathologic diagnosis. Most patients have antibodies to

human T-lymphotropic virus, type I. CTCL progresses rapidly without treatment with a median survival time often measured in months. Systemic chemotherapy, LSG15, when combined with intrathecal chemotherapy, is associated with increased survival time. This regimen can lead to significant toxicity, so CHOP is recommended in patients over 70 years old.

References:

1. Bagherani N, Smoller BR. An overview of cutaneous T cell lymphomas. F1000Res. 2016;5:F1000 Faculty Rev-1882. Published 2016 Jul 28. doi:10.12688/f1000research.8829.1

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Presentation Number: 1.9

Category: Clinical Vignettes

Title: Austrian's Syndrome: A Rare Triad of a Common Infection During Uncommon Times.

First Author: Dennys Jimenez

Additional Authors: Francisco Brito-Aleman MD MBA, Lynn Horvath MD, FACP, FIDSA

Institution: Univ of Texas Hlth Sci Ctr Prog

Abstract: In early April 2020, an unidentified 52 y.o male presented with altered mental status and respiratory distress. He was intubated and treated with broad spectrum antibiotics. Chest CT showed multifocal infiltrates with ground glass opacities. He was thought to have COVID-19, however, PCR was ultimately negative in the next 8 hours.

The following day, blood cultures grew pan-susceptible Streptococcus pneumoniae. Further evaluation led to a negative head CT followed by lumbar puncture, both initially delayed due to COVID-19 concerns. CSF analysis confirmed meningitis and cultures grew S. pneumoniae. Therapy was then narrowed.

On day 3, patient was identified as a 52y.o with well controlled HIV and alcohol use. Family reported he delayed seeking medical care due to COVID-19 concerns. Of note, he was vaccinated with PCV13 (2014) and PPSV23 (2015).

An echocardiogram was done since he persisted febrile. No vegetations were seen, but a new mild tricuspid regurgitation was noted.

On day 4, he had a generalized tonic-clonic seizure confirmed on EEG. MRI brain showed multiple new left cerebral hemisphere and internal capsule infarcts from suspected septic emboli. TEE was not performed due to COVID-19 delays because test was thought to be a false negative given CT findings. Patient was treated empirically for 6 weeks for high suspicion of endocarditis.

Patient was extubated but unfortunately, he remained nonverbal with limited probability of neurological recovery.

Invasive S. pneumoniae is common in HIV patients. Austrian's Syndrome is a rare triad of pneumonia, meningitis, and endocarditis due to S. pneumoniae. Was first documented in 1957 and is associated with a high mortality. Our patient had well controlled HIV, was vaccinated but was a drinker.

This case is important for two reasons. First, it highlights a rare and severe form of a common infection. Second, it illustrates COVID-19 related delays in patient care.

References:

Austrian syndrome: A rare manifestation of invasive pneumococcal disease. A case report and bibliographic review. Rodríguez Nogué M, Gómez Arraiz I, Ara Martín G, Fraj Valle MM, et al. Rev Esp Quimioter. 2019 Apr;32(2):98-113.

Austrian's syndrome (endocarditis, meningitis and pneumonia caused by Streptococcus pneumoniae). Apropos of a rare case. Siles Rubio JR, Anguita Sánchez M, Castillo Domínguez JC, et al. Rev Esp Cardiol. 1998 Dec;51(12):1006-8.

Austrian syndrome: The deadly triad. Shin YI, Papyan N, Cedeño H, Stratidis J. IDCases. 2020;22:e00948. doi: 10.1016/j.idcr.2020.e00948.

Severe Austrian Syndrome in an Immunocompromised Adult Patient - A Case Report. Chirteș IR, Florea D, Chiriac C, et al. J Crit Care Med (Targu Mures). 2018 Feb 9;4(1):17-22

Presentation Number: 1.10

Category: Clinical Vignettes

Title: Bladder Tuberculosis: A rare presentation of a common disease

First Author: Ariadna Perez-Sanchez

Additional Authors: Daniel Rosas, Stephanie Ibrahim, Matthew Brigmon

Institution: Univ of Texas Hlth Sci Ctr Prog

Abstract: Tuberculosis is an infection primarily known to cause pulmonary disease. However, extrapulmonary tuberculosis has become increasingly recognized but can be difficult to diagnose due to its spectrum of manifestations. We present a case of longstanding hematuria, which is an atypical presentation for genitourinary tuberculosis. This is a diagnostic challenge, becoming critical to suspect and treat in a timely manner.

Case

A 37-year-old male from Mexico, with history of nephrolithiasis, presents with gross hematuria and dysuria for four months. Urology performed a cystoscopy with transurethral resection of a bladder lesion, laser lithotripsy, and placement of a right ureteral stent. After discharge, he returned with fever, rigors, and lower back pain. Given worsening clinical status he was transferred to the ICU. On arrival, he was intubated and started on broad-spectrum antibiotics. Nevertheless, fever persisted, with shock requiring pressor support. Urine cultures, blood cultures, and imaging were unrevealing for a source of infection. The urinalysis showed RBC casts concerning for glomerulonephritis and methylprednisolone was started. On urine microscopy, no dysmorphic RBCs were seen, and steroids were discontinued. After two weeks, there was no improvement in clinical status. At this time, bladder biopsy reported focal non-necrotizing granulomatous inflammation that prompted the ICU to perform a bronchoscopy. The latter showed acid-fast bacilli and PCR positive for Mycobacterium tuberculosis in the bronchoalveolar lavage. He was started on RIPE therapy.

Discussion

This patient with long-standing hematuria, coming from an endemic country, with persistent fevers, sterile pyuria, and shock had an elusive diagnosis of genitourinary TB. Administration of high-dose steroids could have precipitated worsening disseminated disease. Tuberculosis is a major infectious killer globally, with genitourinary TB representing the third most common form of extrapulmonary cases. This case outlines the relevance of early recognition of urogenital tuberculosis to institute the appropriate treatment and prevention of unnecessary therapy and morbidity.

References:

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2. World Health Organization. Global Tuberculosis Report 2020. World Health Organization; 2020.

Presentation Number: 1.11

Category: Clinical Vignettes

Title: Profound Hypokalemia leading to Ventricular Arrhythmias

First Author: Keerthana Pakanati, MD

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Abstract: This case describes an 83-year-old-male who presented to the emergency department (ED) with weakness, fatigue, and worsening confusion for the past one month. His chief complaints were decreased appetite, weight loss, occasional dizziness, nausea, vomiting, sore throat, headache, and intermittent low-grade fevers at 99 degrees Fahrenheit. Initial labs included potassium of 1.6 and it was normal 4 months ago. During the initial evaluation in the ED, the patient had 4 episodes of life threatening arrythmias which included pulseless ventricular tachycardia (VT) which converted to atrial fibrillation, pulseless VT that converted to Torsades, and pulseless VT that converted back to sinus rhythm – all of which required cardiopulmonary resuscitation, electrical defibrillation and IV medications to achieve return of spontaneous circulation. During the hospitalization, the patient received total of 434.3mEq of potassium to maintain a potassium level above four and the patient had no further episodes of ventricular tachycardia. His cardiac workup did not reveal structural causes of his arrest. On hospital day five, the patient demonstrated significant clinical improvement and met criteria for discharge. This case demonstrates that profound hypokalemia, especially a potassium less than 2, can increase risk for ventricular arrhythmias – in particular, pulseless ventricular tachycardia.

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Category: Clinical Vignettes

Title: Left ventricular thrombosis and venous thromboembolism in the setting of acute cocaine use and underlying protein C deficiency

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Abstract: While hereditary thrombophilias are an important differential in patients with unexplained thrombotic events, a confirmed diagnosis should not limit the consideration of additional factors contributing to these events.

A 31-year-old previously healthy, African American male presented to the emergency department with a 2-day history of left-sided pleuritic chest pain and dyspnea. He was hemodynamically stable upon admission and physical exam was unremarkable. He denied any recent provoking factors or any significant past medical history. Although he denied any significant family history, he later noted that his father passed away from a "blood clot". He described current, daily cigarette smoking (18 pack years) with frequent marijuana and alcohol use. Urine drug screen was positive for cocaine. CT pulmonary angiography revealed large, bilateral segmental and subsegmental pulmonary emboli. Bilateral lower extremity doppler ultrasound revealed extensive right-sided deep vein thromboses extending from mid-femoral vein to the posterior tibial vein. Transthoracic echocardiogram demonstrated mildly reduced left ventricular (LV) systolic function (ejection fraction 48%) with a large 2.2cm x 1.4cm protruding LV mass consistent with thrombus. Coagulability workup indicated a functional protein C deficiency, with activity of 64% (normal range 83-168%). Patient was started on therapeutic enoxaparin and discharged with plans to bridge to warfarin in an outpatient setting.

Reported cases of LV thrombi due to protein C deficiency are exceptionally rare; moreover, the protein C levels observed in these cases are generally lower than that detected in this patient, thereby suggesting additional etiologies contributing to this patient’s hypercoagulable state. This patient’s notable cocaine and tobacco use likely bolstered the prothrombotic effects spurred by his underlying protein C deficiency, resulting in extensive systemic thromboses. This case not only demonstrates the importance of considering hereditary thrombophilias in patients with unexplained thrombotic events, but also recognizing multiple etiologies rather than settling on a single unifying diagnosis.

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