Ohio-ACC Poster Competition

Fellow in Training Research Abstracts Oral Competition

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Title: (Do not enter author details)

T-Wave Abnormalities as ECG Signature of Myocardial Edema in NST-Elevation Acute Coronary Syndromes

Andrea Cardona[1], Giuseppe Ambrosio[2], Stephen Schaal[1], Karolina Zareba[1], Subha Raman[1], [1]*Ohio State University, Columbus, USA,* [2]*University of Perugia, Perugia, Italy*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction: Persistent T-wave abnormalities (TWAs) are often seen during the acute phase of non ST-segment elevation acute coronary syndromes (NSTE-ACS), but their pathophysiological significance has not been established.

Hypothesis: We hypothesized that persistent TWAs in NSTE-ACS correspond to the presence of myocardial edema by T2 cardiac magnetic resonance (CMR).

Methods: In 82 prospectively-enrolled patients with NSTE-ACS, 12-lead electrocardiography (ECG) and CMR with T2-weighted imaging (T2W) and late gadolinium enhancement were acquired before invasive coronary angiography. TWAs were defined as presence of inverted or biphasic T-wave in ≥ 2 leads on the ECG acquired closest in time to CMR examination. Myocardial edema by CMR was defined as ≥ 2 T2-positive left ventricular segments.

Results: Patients were studied at a median 24 (IQ 17 – 50) hours after admission. Of 79 patients with adequate T2W-CMR, 36 (46%) showed TWAs on ECG. The prevalence of myocardial edema was higher in those with vs. without TWAs (32/36 [89%] vs. 20/43 [47%], p<0.001). TIMI risk score, hemodynamics, ejection fraction, major cardiovascular risk factors, time-to-CMR, and troponin plasma concentrations, were similar in the 2 groups.

By univariable logistic regression analysis, wall motion score index, and myocardial edema at CMR were the only variables significantly associated with TWA presence. By multivariable logistic regression analysis (adjusted for age and sex) edema at CMR was the only independent predictor of T-wave abnormalities (OR=14.6; 95% CI 2.9-73.2; p<0.001). TWA yielded 89% positive predictive value, with a specificity of 85%, and a sensitivity of 61% to predict edema at CMR.

Conclusions: This is the first demonstration that standard 12-lead ECG T-wave abnormalities seen during the acute phase of NSTE-ACS are related to the presence of myocardial edema. Presence of TWAs is a simple, highly specific, and moderately sensitive marker of myocardial edema. The electrophysiological mechanisms and treatment implications of this underlying pathophysiology warrant further investigation.

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Title: (Do not enter author details)

Three Dimensional (3D) Printing and Functional Assessment of Aortic Stenosis Using a Flow Circuit: Feasibility and Reproducibility

Serge Harb[1], Ryan Klatte[1], Brian Griffin[1], Leonardo Rodriguez[1], [1]*Cleveland Clinic, Cleveland, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Background: Recent reports have created 3D printed models of cardiac structures and various defects, mainly congenital, from echocardiographic (echo) or computed tomography (CT) data. We sought to functionally assess the 3D printed model of severe aortic stenosis (AS) using a flow circuit. The case of an 83 year old man with severe calcific AS was studied. On echo, at a heart rate of 60 b.p.m. and a blood pressure of 122/70 mmHg, the calculated stroke volume (SV) was 67 mL (indexed 29mL/m2), the peak/mean gradients were 68/45 mmHg, the aortic valve area 0.82 cm2 and the ejection fraction 60%.
Methods: Based on a high resolution, contrast enhanced CT at the 70% phase, a 3D reconstruction (Mimics, Materialise) and modeling (Rhino 3D, McNeel and Associates) software were used to prepare the model for printing, which was done with a Stratasys Connex 350 printer (figure 1). Tango+ material was used for soft tissue and VeroWhite material for calcifications. The 3D printed model was connected into a flow circuit containing a pulsatile pump (Harvard Apparatus Model 1423), a pressurized and an unpressurized tank (figure 2). Flow was measured using a transonic flow probe (model ME 13 PXN, Transonic Systems, Inc.) and meter (model T410, Transonic Systems, Inc). Pressures proximal and distal to the aortic model were obtained via fluid filled sensors (model DTX Plus DT 4812D, Becton Dickinson) and signal amplifiers (model 11-4123-09, Gould). The data was acquired using LabVIEW.
Results: In order to simulate the patient's HR and SV, the pump was set at a rate of 60 strokes per minute, with a stroke output of 70 ml. Figure 3 depicts the measurements obtained. The gradients obtained were 67/51 mmHg, very close to those obtained by echo. Altering the flow settings generated different gradients.
Conclusion: Functional assessment of a 3D printed model of severe AS using a flow circuit is feasible and yielded gradients similar to those obtained by echo. 3D printing may become a helpful tool in the understanding of valvular pathophysiology.

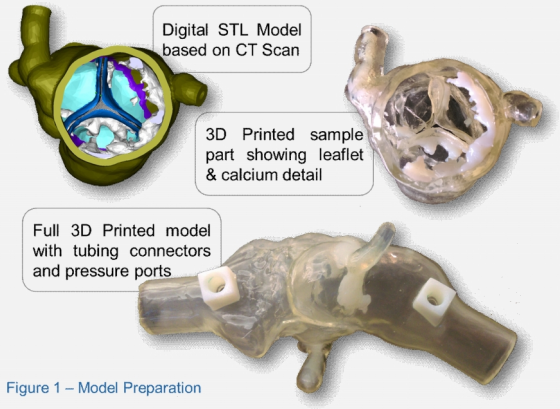

Figure 1 – Model Preparation

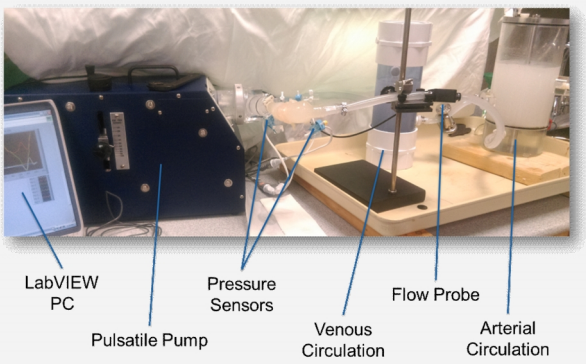

Figure 2 – Test Loop Setup

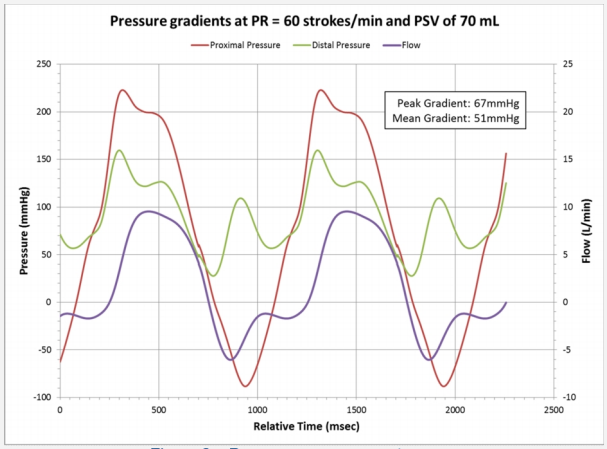

Figure 3 – Pressure measurements

CV TEAM

Case Abstracts

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Title: (Do not enter author details)

Cangrelor Use in a Variety of Patient Populations: Medication Utilization Evaluation and Case Reports at an Academic Medical Center

Robert Barcelona[0], [1]*University Hospitals Cleveland Medical Center, Cleveland, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction:

Antiplatelet therapy is necessary in treating acute coronary syndromes. Prasugrel and ticagrelor are oral $P2Y_{12}$ inhibitors that have differences in pharmacokinetics that lead to faster onset and more potency compared to clopidogrel. The newest approved agent, cangrelor, is an ultra-fast acting intravenous $P2Y_{12}$ inhibitor, which may be useful in conditions where reversibility of antiplatelet activity may be necessary. This report describes the use of cangrelor, an ultra-fast acting intravenous $P2Y_{12}$ inhibitor, in a variety of patient cases and the 1-year experience at an academic medical center.

Objectives and Case Reports:

Cangrelor was approved to the formulary at University Hospitals and restricted to the following patient populations: 1) ad-hoc complex percutaneous coronary intervention (PCI) who had not received a loading dose of a $P2Y_{12}$ inhibitor, 2) as a bridging agent for patients with a coronary stent who require their oral $P2Y_{12}$ inhibitor to be discontinued, and 3) in patients with a left ventricular assist device with thrombosis that is worsening despite antiplatelet and anticoagulation therapy, and a short acting antiplatelet is required. Usage of cangrelor from August 2015 – July 2016 was obtained using data from the computerized order entry system and cardiac catheterization data. Data collected included patient demographics, medical history, indication for usage, concomitant antiplatelet therapy, dose of cangrelor used, duration of cangrelor infusion, and bleeding complications. Objectives of the medication utilization evaluation were to determine if the cangrelor restriction guidelines were followed and to describe the patient population cangrelor was utilized in. Examples of patients where cangrelor was utilized will be described.

Discussion/Conclusion:

Pharmacotherapy of acute coronary syndromes consists of dual antiplatelet therapy. Bleeding complications and need for urgent surgery is challenging with current oral agents that have a long duration of action. These case reports describes the cangrelor in the treatment patients who require dual antiplatelet therapy in settings where an ultra-short acting agent was preferred. The results of our current reports and 1-year experience may require reinforcement of our current guidelines and will help ensure that utilization of this agent is used in the appropriate patients.

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Title: (Do not enter author details)

Nonbacterial Thrombotic Endocarditis - A Diagnosis of Exclusion

Shirlien Metersky[0], [1]*OhioHealth Heart & Vascular Physicians, Columbus, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction – Nonbacterial endocarditis (NTBE), also known as marantic endocarditis or Libman-Sacks endocarditis is a rare disorder involving noninfectious vegetation consisting of sterile platelet rich thrombi on the heart valves in the absence of bacterial infection. It is most commonly seen in the 4th- 8th decades of life and there is no distinction between sexes. Left sided heart valves are primarily affected with prevalence in the aortic valve over the mitral valve. Rarely are right sided heart valves affected. The most common population of patients affected by NTBE are patients with advanced malignancies comprising 80% of cases. Other less common etiologies include inflammatory states such as systemic lupus erythematosus and antiphospholipid syndrome. Patients are typically asymptomatic until they present with systemic embolization signs and symptoms rather than valvular heart disease symptoms. The instigating factor in the pathogenesis of NTBE is uncertain, but felt to be due to endothelial injury in the setting of a hypercoagulable state. NTBE vegetation is more easily dislodged that vegetation from infective endocarditis. NTBE is a diagnosis of exclusion since there are no laboratory tests that confirm the diagnosis. A definitive diagnosis can only be made with pathological review of platelet thrombi on autopsy or surgical specimens.

Case Presentation

#1 - 45 yo obese white female with PMH of poorly controlled NIDDM, active tobacco abuse, mixed HLD, and CVA 2009 presents with acute right MCA stroke. CT angio neck without significant stenosis in carotids or vertebral arteries. TEE revealed rounded, shaggy, nonmobile echodensity measuring 9mm in diameter attached to left atrial surface of the middle (P2 scallop) of the posterior MV leaflet. There were tiny filamentous mobile echodensities extruding from the mass. Echodensity was felt to be nonspecific and most consistent with NBTE or papillary fibroelastoma. Normal blood cultures (BC) x 2 and sedimentation rate argued against infectious endocarditis. Hematology consult was placed to rule out hypercoagulable state as an etiology that was negative including antiphospholipid antibodies. Although empiric anticoagulation (AC) with LMWH is preferred over warfarin for NTBE, neurology felt that patient was at high risk for hemorrhagic transformation and patient discharge on lovenox to warfarin bridge. Plan was for repeat TEE in 2-3 months to reassess the presence and size of MV echodensity. If there was resolution of the MV echodensity with AC, then this would favor the diagnosis of NTBE. Whereas, growth of echodensity would favour papillary fibroelastoma. Patient lost to follow-up since was noncompliant with outpatient neurology and cardiology follow-up.

 # 2 – 45 yo Asian female with PMH of TB and stage IV nonsmall cell lung cancer diagnosed 12/2011 with bone and brain metastasis s/p XRT and chemotherapy who presents with acute large right MCA stroke. Just discharged from large tertiary hospital one week prior with symptoms of progressive dyspnea. TTE performed revealed AV mass prompting TEE. TEE showed mobile AV mass located on noncoronary cusp (primarily along ventricular surface) with associated moderate to severe AI and moderate circumferential pericardial effusion. Unable to determine if endocarditis or thrombus. Seen in consult by infectious disease and cardiology who and felt to be NTBE. Discharged on full strength lovenox. Had been on ATB therapy prior to admission with negative BC x 2 at OLH. Full strength lovenox was stopped on admission due to large stroke. Repeat TTE did not visualize the AV well and no mass was seen. TEE not repeated since would not alter plan of care. Negative BC x 1 on admission. Transferred back to her primary hospital for continuity of oncology care since in a research clinical trial.

Discussion

Since NTBE is a diagnosis of exclusion, a definitive diagnosis could not be made with either patient. Presentation of embolic stroke for both patients prompted a TEE revealing vegetation. With negative BC and lack of evidence of systemic infection the diagnosis of NTBE was entertained although fastidious organisms should be considered. Three sets of BC should be performed prior to ATB therapy which was not done on either patient. NTBE may be missed when embolization is to common sites such as spleen, kidney, skin, and extremities due to lack of devastating symptoms compared to stroke presentation. Although patient # 1 had other stroke risk factors including active tobacco abuse, poorly controlled DM, and mixed HLD, her vegetation found on TEE was felt to be the prompting factor for her embolic stroke. Etiology for NTBE on this patient was not determined since her hypercoagulable workup was negative. Further workup would include malignancy workup.

A diagnosis of NTBE is more compelling in patient # 2 due to her advanced malignancy. Her prognosis is poor due to high risk of recurrent embolization and her underlying malignancy. Although AC with LMWH indefinitely is the treatment of choice due to the friable nature of NTBE vegetation, this patient's risk of hemorrhagic conversion prohibited the continuation of AC. Treatment for NTBE is systemic AC. Although there has not been any formal studies comparing heparin to warfarin in NTBE patients, older studies have suggested warfarin as inferior to heparin in reducing incidence of recurrent embolization. There have been no AC studies with direct thrombin or factor Xa inhibitors.

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Case Abstracts

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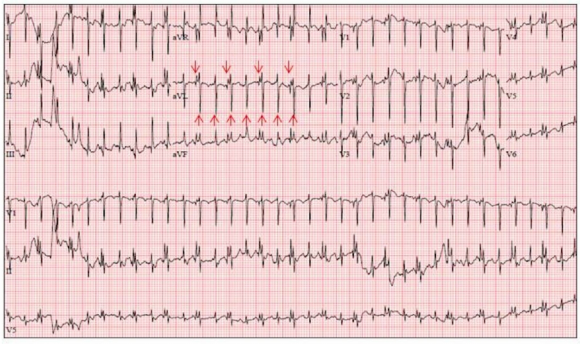
Title: (Do not enter author details)

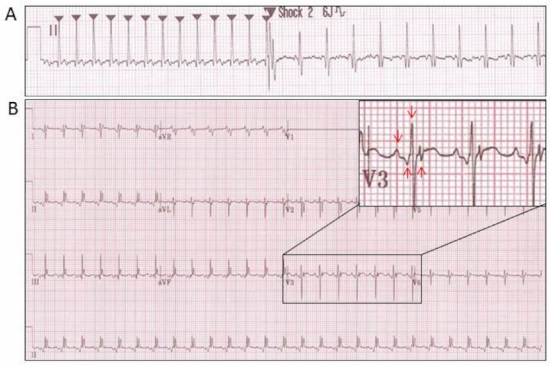
Successful external cardioversion of supraventricular tachycardia in omphalo-thoracopagus conjoined twins.

M. Rizwan Afzal[1], [1]*Ohio States University, Columbus, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

A pair of omphalo-thoracopagus twins was diagnosed with supraventricular tachycardia (SVT) on the first day of life. A 12 lead electrocardiogram (ECG) was performed with leads placed on twin A (Upper Panel). The 2:1 ratio of the two QRS populations was thought to be the result of SVT in twin A, with 1:1 conduction between the two atria and 2:1 conduction at the level of atrioventricular node of twin B. Intravenous adenosine (0.1 mg/kg and 0.2 mg/kg) was given without success. External cardioversion was performed with defibrillation patches placed on the back of each neonate. The thoracopagus anatomy precluded an antero-posterior placement of defibrillation patches. A synchronized shock of 6 joules successfully restored normal sinus rhythm (Lower Panel A). Repeat 12 lead ECG after cardioversion showed two different populations of P and QRS waves with identical heart rates (Lower Panel B). We concluded that the twin with faster sinus rate was driving the rate of the second twin through a connection at the atrial level. The imaging studies including contrast-enhanced computerized tomography revealed a complex relationship of the two hearts.





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Title: (Do not enter author details)

***Retrieve to Relieve:* Novel percutaneous thrombectomy device in select patients with Pulmonary Embolism.**

Abiodun Ishola[1], Umair Ahmad[1], Raymond Magorien[1], Scott Lilly[1], [1]*The Ohio State University, Columbus, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Although improving, in-hospital mortality among those admitted with pulmonary embolism remains virtually 10%. In addition to anticoagulation, catheter-based therapies for pulmonary embolism have been established, in an effort to mitigate the complications observed with systemic fibrinolysis. Among these, ultrasound assisted thrombolysis (EKOS Corp) has emerged as an acceptable alternative among patients with pulmonary embolism and evidence of associated right heart strain (references). Other studies have suggested that thrombus fragmentation and aspiration may help stabilize patients with massive PE, especially when fibrinolysis is contraindicated or ineffective (references). Although there are no commercially available and approved devices for thrombus aspiration in the setting of PE, a novel thrombectomy system, may enable thrombus extraction in patients with large pulmonary emboli.

Herein we describe two cases of submassive pulmonary embolism managed with percutaneous thrombectomy with the novel FlowTriever Retrieval/Aspiration system (Inari Medical, Ivrvine CA.). These cases illustrate an additional catheter-based therapy for pulmonary embolism, and may have safety advantages over protocols that require thrombolytic agents.

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Title: (Do not enter author details)

Iatrogenic Saphenous Vein Graft Dissection

Sajid Ali[1], Justin Ugwu[1], Ma'en Al-Dabbas[1], Ali Chaudhry[1], Sohail Ali[1], Mohummad Taleb[1], [1]*Mercy Health, Toledo Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction

Complications seen during percutaneous coronary intervention include those related to cardiac catheterization and those that occur as a consequence of the specific equipment. Dissection and abrupt closure after PTCA is one of the complications. However the dissection of venous graft is a rare complication. We know of only two reported cases in literature. We present a 72 year old male who was transferred from outside facility with ST-elevation MI after diagnostic percutaneous catheterization and was found to have diagnostic catheter induced dissection of functional saphenous venous graft leading to type D dissection.

Case

A 72 year old male with multiple comorbidities and past medical history of coronary artery disease status post coronary artery bypass graft including a saphenous venous graft to right coronary artery (SVG-RCA), and right internal mammary artery to left anterior descending artery (RIMA-LAD). He was scheduled to undergo a diagnostic coronary angiography in an outlying facility for evaluation of Ventricular tachycardia. Angiography revealed severe disease of the native vessels and graft injection initially showed SVG-PDA with 70% ostial calcified plaque with patent RIMA to LAD. Towards the end of angiography, contrast was seen to be staining the ostium of the SVG-PDA graft. A further injection demonstrated 100% occlusion from the ostium of the PDA graft. Inferior ST Elevation was demonstrated on his EKG immediately and patient was subsequently air lifted to our facility. After arriving to our catheterization laboratory the SVG-PDA ostial lesion was eventually able to be crossed and when moving to the caudal RAO position showed a type D spiral dissection which was also confirmed with intravascular ultrasound.

Discussion

Veins have thin walls which result in its tortuosity and collapsibility. As a result of their thin wall and low flow pressure, dissection is a rare pathology of native veins. When grafted to arterial circuits, exposure to increased stress of high pressure flow may be the predominant factor that increase predisposition to dissection formation when compared to native functioning veins. It is suggested that plaque rupture may be a part of vein graft disease, and this may correlate with graft aging. Vein graft atheroma have also been shown to contain foam cells and inflammatory cells with a poorly developed or absent fibrous cap, making the grafts more prone to plaque rupture and dissection. This risk may be increased with older calcified tortuous vein grafts. Saphenous venous graft dissection is one of the rare differential diagnoses on post PTCA complications that should be recognized and kept in mind.

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Title: (Do not enter author details)

Transient Myocardial Ischemia: A Complicated Etiology Of A Serious Disease

Muhanad Al-Zubaidi[1], George Broderick[0], *[1]Wright State University, Dayton, USA, [2]Good Samaritan Hospital, Dayton, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Case Presentation:

A 76 year-old male with CAD. 2 months before presentation, he underwent 4 vessels coronary bypass. He presented with chest pain, normal initial troponin and non-specific ST changes on EKG. He was admitted and placed on ACS protocol with IV heparin and subsequently chest pain improved. Next day early morning, he suddenly developed worsening chest pain and non-specific ST changes in spite of being on IV heparin and high dose IV nitroglycerine drip. Suddenly, he became unresponsive and developed sustained ventricular arrhythmia. CPR was performed and he was electrical-shocked to normal sinus rhythm and survived the acute event. Troponin peaked to 4 ng/mL. Coronary angiogram showed occluded LIMA-LAD graft, native LAD has 50% lesion and SVG to RCA had a 99% ulcerative looking stenosis. The other 2 venous grafts were occluded as well with estimated Ejection fraction (EF) of 30%. Considering his new HFrEF and severe native and grafts disease, we planned for Impella support SVG-RCA PCI next day. Surprisingly, the 2nd left heart catheterization showed normal SVG-RCA Graft. Patient was treated medically and his EF improved to 50% after 3 months.

Discussion:

Transient myocardial ischemia has been described before with poorly defined pathophysiology. Patient may present with any type of the ACS. Some studies show that 64% of all unstable angina and NSTEMI episode occurred between 10 pm and 8 pm with or without a preceding increase in heart rate. Usually, it is more frequent and prolonged in NSTEMI than unstable angina. Transient ischemia may persist even on optimal medical therapy and it carries a poor prognosis.

Different mechanisms have been considered as an isolated culprit or in multi-mechanical fashion. These mechanisms includes myocardial oxygen demand mismatch, acute rupture of a plaque, circadian variations of platelet aggregability and nocturnal increased coronary tone which may lower the ischemic threshold, as well as, the lower endogenous fibrinolytic activity and higher free radical generation at night and early morning.

Management includes standard ACS therapy and continuous ST-segment monitoring for early diagnosis, prognosis and to evaluate medical therapy. Nocturnal peak of symptoms, ST changes and unresponsiveness to treatments can be clinical clues.

Conclusion:

Transient ischemia has low ischemic threshold and occurs mainly at night. It may lack increase in heart rate and respond to standard medical therapy. Further studies are needed to understand its pathophysiology and best management.

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Title: (Do not enter author details)

Aborted Sudden Cardiac Death with Extracardiac Sarcoidosis

Timtohy Byrnes[1], Ottorino Costantini[1], Jessica Kline[1], [1]*Summa Health System, Northeast Ohio Medical University, Akron, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction: Extracardiac sarcoidosis has an estimated prevalence of 330/100,000 as of 2010. To date, we have a limited understanding of the risk of developing cardiac sarcoidosis in these patients, as well as their risk of sudden cardiac death.

Case: 60 year old female with a past medical history of extracardiac sarcoidosis, hypertension, and moderate mitral regurgitation presents with the chief complaint of chest pain to the Emergency Department. She was discharged from the hospital less than one week prior after presenting with similar symptoms and a troponin of 18.8 ng/mL. Electrocardiogram showed sinus rhythm with premature ventricular beats. Her coronary angiogram and transthoracic echocardiogram found no obstructive coronary artery disease and a left ventricular ejection fraction of 60%, respectively. She was diagnosed with pericarditis and discharged with anti-inflammatory therapy. After arrival to the Emergency Department, the patient developed ventricular fibrillation. She returned to normal sinus rhythm with frequent ventricular ectopy after a single 200 J shock. The patient was transferred to our hospital. A cardiac MRI found a left ventricular ejection fraction of 60%, edema of the apical and lateral left ventricular walls, and delayed transmural gadolinium enhancement in the apical segments of the left ventricle anterior and posterior walls, consistent with cardiac sarcoidosis and active inflammation. The patient received a dual-chamber ICD and discharged the following day with steroid therapy.

Conclusion: This patient highlights the need for improved sudden cardiac death risk stratification in patients with extracardiac sarcoidosis. We propose routine cardiac MRI to assess for cardiac involvement in patients with extracardiac sarcoidosis and a left ventricular ejection fraction greater than 35%, particularly if additional signs of cardiac involvement are present, such as pericarditis or frequent ventricular ectopy.

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Title: (Do not enter author details)

Antipsychotic Therapy Causing The Heart To Go Mad: A Rare Sequelae Of Bupropion Use

Janish Kothari[2], Jered Cook[1], Megha Kothari[3], Amer Syed[4], *[1]University of Toledo Medical Center, Toledo, Ohio, USA, [2]St George's University, St. George's, Grenada, [3]New York Methodist Hospital, Brooklyn, New York, USA, [4]Jersey City Medical Center, Jersey City, NJ, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

A 45 year old Caucasian male with a past medical history of depression and anxiety presents to the emergency department complaining of a long-standing dry cough, palpitations, and dyspnea on exertion. Upon physical examination and echocardiogram it was revealed that the patient was in heart failure withan ejection fraction of 10% without regional wall motion abnormalities. Upon further investigation it was found that the patient had persistent tachycardia (HR of 120 bpm) secondary to bupropion use that lead to myocardial changes, consistent with proposed mechanisms in current literature. This case report explores our patient's medical history and concludes with a discussion on the topic of bupropion causing tachycardia-induced cardiomyopathy, a rare sequelae of the medication.

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Title: (Do not enter author details)

A Rare Case of Right Coronary Sinus of Valsalva Aneurysm in an Elderly Woman

Katherine Dodd[1], Laxmi Mehta[1], [1]*Ohio State University Medical Center, Columbus, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction/Objective:
Sinus of Valsalva aneurysms (SVAs) are rare and typically asymptomatic, with the diagnosis usually being incidental. Among patients undergoing cardiac surgery, a SVA is found in 0.1 - 3% of patients[1]. The most common origin of SVAs is the right coronary sinus (70% of patients), compared to origin from the non-coronary sinus (25% of patients) and the left coronary sinus (5% of patients)[1]. Men are also more likely to have SVAs than women (male to female ratio is 4:1)[1].

Case Presentation:
A 72 year old Caucasian woman with paroxysmal atrial fibrillation on Tikosyn and Coumadin, prior ischemic stroke with residual left hemiparesis, multiple sclerosis, hypothyroidism, sick sinus syndrome status post dual-chamber pacemaker, presented to the hospital on July 20, 2016 with palpitations after she was found to be in atrial fibrillation with a rapid ventricular response during a doctors appointment. Electrophysiology was consulted and she was initiated on Amiodarone. A routine echocardiogram was performed the next day which showed a large aneurysmal dilation of the right coronary cusp (5.3 cm x 3.6 cm in diameter). Cardiac surgery saw the patient during her admission. A CT angiogram of the chest to better delineate the aneurysm was obtained. The patient was taken to the operating room on day six of admission for a planned aortic root repair of the of the right coronary sinus of Valsalva aneurysm with resuspension of the aortic valve and bovine pericardial reconstruction of the aorta. Unfortunately, post operatively, the patient experienced multiple episodes of cardiac arrest (both pulseless electrical activity and ventricular fibrillation). Despite re-exploration for the cause of cardiac arrest which was not identified, the patient required escalating doses of pressor support and eventually developed systolic arrest with no return of spontaneous circulation.

Discussion/Conclusion:
Aneurysm of the sinus of Valsalva are uncommon, usually affect the right coronary sinus, and diagnosis is typically incidental[1]. Though rare, when they do occur, the management of a SVA is quite difficult and surgical intervention is required. The embryologic origin of a SVA is thought to be from distraction of the aortic media from the annulus, which is also why the majority of patients have associated anomalies (aortic regurgitation and ventricular septal defects are the most common)[3,4]. Despite the fact that our patients aneurysm was quite large, she miraculously made it into the seventh decade of life with no significant symptoms and had no associated structural anomalies. When symptoms do arise, they are typically related to either compression of adjacent structures, intracardiac shunting, or aneurysmal rupture[6]. SVAs were originally identified with transthoracic echocardiography, but transesophageal and CT angiography are also valuable imaging entities to better determine the origin and involvement of surrounding structures. It is important to determine whether there is aortic valve leaflet involvement, because if this is the case, repair or replacement of the valve is necessary. Our patient did not have aortic insufficiency or leaflet involvement, and therefore was able to undergo aortic root repair with valve sparing surgery[5]. However, our patient did have involvement of the right coronary artery, necessitating re-implantation during aneurysmal repair. Surgical repair is the mainstay of treatment for patients with SVAs, but technique (direct closure, patch repair, endovascular interventions) depends on the origin and size of the aneurysm and whether any surrounding structures are involved[7]. Given complexity of the surgery, however, complications can occur, as was the case for our patient.

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Isolated Troponin Elevation in an Unclear Episode of Loss of Consciousness

Federico Garcia Trobo[1], Ahmad Abdin[1], Marc Penn[1], [1]*Summa Health Systems, Akron/Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction: The role of cardiac biomarkers in risk stratification of syncope is unclear. Troponins are not included in most accepted syncope risk scores, although it is common to use Troponin as part of the workup for syncope.

Case: We report the case of a 78 year-old male with no significant past medical history presenting to the emergency department via EMS after found unresponsive by his wife. Per EMS report, patient was found down with emesis next him. No focal deficits were appreciated. Upon arrival, patient denied any chest pain, shortness of breath, headache, and difficulty with speech, weakness, or changes in vision. Brain CT was negative for acute pathology and after being evaluated by Stroke team the patient was admitted to medicine ward with the diagnosis of TIA versus seizure. On follow up labs noted a significant increase in Troponin levels (from 0.059 to 2.49), even though the patient was asymptomatic and his EKG showed a normal sinus rhythm with no signs of acute or chronic ischemic pathology. It was decided to transfer the patient to CCU and treat him as a NSTEMI. Overnight the patient presented asymptomatic EKG dynamic changes T wave inversions from V2-V6.
In light of evidence of active ischemic disease, cardiac catheterization was preformed showing a severe proximal Left Anterior Descendent artery lesion, also known as "Widow Maker", that was stented with no major complications. Retrospective analysis of this case suggests the etiology of the syncope episode was most likely a cardiac arrhythmia trigger by ischemia.

Conclusion: This case is an example of the game changer roll of a cardiac marker in a syncope evaluation. More studies need to be done to evaluate the impact of cardiac markers at the time of risk stratification of patients with syncope.

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Henoch-Schonlein Purpura (HSP) Presenting as a Large Pericardial Effusion in an Adult

Atif Hassan[1], Romesa Sajjad[0], *[1]university of cincinnati, cincinnati, ohio, USA, [2]Dow university, karachi, sindh, Pakistan*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

INTRODUCTION:
Henoch-Schonlein Purpura (HSP) is the most common form of systemic vasculitis in children [1]. It is characterized by a tetrad of palpable purpura, arthralgia, abdominal pain and renal disease in children [2]. It rarely presents with significant pericardial disease, especially in adults and is a rare entity with only a few reported cases.
CASE DESCRIPTION:
A 49 year old Male presented with two month history of progressive dyspnea, polyarthralgia, fatigue, skin rash and was found to have a large pericardial effusion on an echocardiogram with respiratory variation requiring drainage. Physical exam was consistent with cardiac dullness beyond the apical point of maximal impulse, elbow joints with limited range of motion and swelling, periungal erhythema and palpable purpura over extremities. Laboratory evaluation revealed markedly elevated inflammatory marker, CRP of 225. Biopsy of the skin purpura was consistent with IgA leukocytoclastic vasculitis with intense deposition of IgA within superficial dermal blood vessel walls. Pericardial fluid was consistent with acute inflammation with several neutrophils but no growth on culture. Patient was started on high dose steroids with marked improvement in symptoms including decreased pericardial effusion. A repeat echocardiogram was obtained which showed only trivial pericardial effusion with no signs of tamponande.
DISCUSSION:
Henoch-Schonlein Purpura is rather a common disease in children with systemic hypersensitivity vasculitis in the skin and other organs including kidneys, but rarely involves myocardium or pericardium and is even rarer to present as a large pericardial effusion as a manifestation of the disease, especially in an adult. Henoch-Schonlein Purpura presenting as pericardial disease is a rare entity and only a few cases have been reported. Although the pericardial involvement of Henoch-Schonlein Purpura seen in this patient is rare, any collagen vascular disease can present with pericardial effusion and might respond to appropriate anti-inflammatory therapies including steroids if recognized promptly.

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Title: (Do not enter author details)

Tachycardia born from a Bradycardic Substrate

Sammy Hayes[1], Dilesh Patel[1], [1]*The Ohio State University, Columbus, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction:
A wide complex tachycardia (WCT) is uncommon in young healthy patients with structurally normal hearts. When present, it often represents a diagnostic challenge and a learning opportunity. We present one such case of a young man presenting to our emergency department with a WCT and a rare diagnosis.

Case Presentation:
A 22 year old male with pectus excavatum and history of palpitations who presented to our emergency room with a HR of 157 bpm. ECG showed a WCT with RBBB pattern with superior axis and atrioventricular (AV) dissociation. RBBB pattern was that of a typical RBBB with a QRS duration of 104 ms. Given concern for SVT with aberrancy, adenosine was administered and tachycardia terminated. Baseline ECG in sinus bradycardia showed no pre-excitation, incomplete RBBB and right axis deviation. Careful review of the WCT ECG was consistent with a ventricular tachycardia (VT) based in the His-Purkinje system (HPS). Our patient was previously on metoprolol for his palpitations with progression of his symptoms despite dose escalation so we proceeded with an electrophysiology study (EPS) after an echocardiogram revealed completely normal LV, RV, atria and no signs of valvular dysfunction. EPS surprisingly revealed signs of severe conduction disease during sinus rhythm with prolonged HV interval consistent with infra-nodal conduction disease. We also proved the right bundle to be part of the retrograde limb of his VT circuit confirming our clinical suspicion based on his initial 12-lead ECG. To map the antegrade part of his VT circuit, LV mapping was attempted but patient developed infra-hisian AV block after a spontaneous premature ventricular contraction (PVC) terminated VT. Patient recovered AV conduction and ablation was deferred with plans for a cardiac MRI (CMR) to help understand the etiology behind his conduction system disease. CMR was completely normal with no signs of fibrosis or infiltrative process. Rheumatologic and neurologic evaluations were also initiated to assess for inflammatory processes or myotonic dystrophy. Meanwhile, verapamil was started in an attempt to quiet his VT over the weekend. On Monday, he developed recurrent VT, despite verapamil use, during a treadmill stress ECG study. A second EPS was performed and the patient had two different inducible VT morphologies (one was the original right bundle with superior axis and the other was a right bundle with inferior axis) involving the HPS. Mapping during VT identified purkinje potentials at the earliest activation sites and successful ablation was performed at the distal anterior fascicle with no inducible VT, despite aggressive testing for 30 minutes post-ablation. Unfortunately, patient had recurrent VT later that evening and required another EPS. During the third EPS, inducing VT was extremely challenging and the posterior fascicle was mapped with empiric ablation transecting the posterior fascicle. After this study, our patient did quite well with no recurrence of VT and no signs of AV block. Furthermore, neurologic evaluation revealed no signs of muscular dystrophy and rheumatologic evaluation displayed no peripheral signs of inflammatory process to explain the conduction system disease.

Discussion:
This case of WCT brought up several uncommon, interesting findings which continually changed our clinical direction and care. The termination of the presenting tachycardia with adenosine initially suggested an AV node dependent tachycardia yet the patient turned out to have VT. Once the diagnosis of HPS-based VT was made, the structurally normal heart suggested idiopathic fascicular VT (Belhassen VT) as the diagnosis. However, the lack of response to verapamil and findings of infra nodal conduction disease are atypical of idiopathic fascicular VT and the working diagnosis changed to bundle branch re-entrant VT (BBRT). Then, recurrence of VT despite ablation of the anterior fascicle pushed us to ablate along the posterior fascicle. This decision was especially difficult given the risk of permanent AV block because of the underlying conduction system disease. The clinical situation called for the ablation of a diseased conduction system with the goal of abolishing tachycardia without worsening the existing conduction disease and creating bradycardia with pacemaker dependence. Following this challenging journey, our patient is free of his WCT but will continue to deal with a diseased conduction system. He will be closely monitored for any signs of worsening conduction disease, which may require pacemaker implantation in the future.

Conclusion:
Diagnosis of VT in a structurally normal heart can be clinically challenging. Our patient presentation highlights a rare type of fascicular re-entrant ventricular tachycardia that differs from the classic idiopathic fascicular (Belhassen) VT. Our patient's diseased conduction system was the underlying substrate for his VT circuit, making it difficult to effectively ablate his VT while also minimizing his risk for future pacemaker reliance.

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A rare presentation of adenosine induced coronary vasospasm following FFR study.

Iliana S. Hurtado Rendon[1], Justin Dunn[0], [1]*Summa Health System, Akron, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction

Adenosine has several important cardiac properties including regulation of coronary blood flow and electrophysiological effects on the conduction system. Exogenous use of adenosine is common in multiple clinical settings such as acute assessment and termination of some supraventricular arrhythmias, myocardial perfusion imaging and for physiological evaluation of atherosclerotic coronary lesions in the cardiac catheterization suite. We report a case of adenosine-induced coronary vasospasm following measurement of FFR (fractional flow reserve) during coronary angiography.

Case presentation

A health conscious 52 year old woman is seen for a 2 year history of chest pain and exertional dyspnea. She was an avid runner until her symptoms began to limit her daily exercise routine. She did not initially seek medical attention but made a valiant effort to continue running until the symptoms forced her to quit. The pattern remained stable until recently when the symptoms became noticeable with low levels of activity but never at rest. She underwent an exercise nuclear stress test that provoked 1mm ST segment depressions on the lateral leads during the treadmill portion and an area of apical ischemia was seen on the myocardial perfusion scan. The patient was referred for a diagnostic cardiac catheterization. Coronary angiography revealed essentially normal coronary anatomy with the exception of a stenotic appearing segment of the mid-LAD suspicious for myocardial bridging. To better characterize the degree of flow limitation, intracoronary adenosine was given while measuring the FFR (fractional flow reserve). The flow reserve ratio was abnormal at 0,75. During this study the patient complained of retrosternal chest pain. An intravascular ultrasound catheter was then introduced to better visualize the mid-LAD segment and confirm the absence of a stenotic atherosclerotic plaque. The acoustic image showed no atherosclerosis but did reveal profound vasospasm in two segments of the mid-LAD. After receiving multiple boluses of nitrates - a total of 2700 mcg - the vasospasm resolved and the patient's symptoms abated.
The patient was discharged home on a low dose of long acting nitrate. She was followed up two weeks later in the outpatient clinic with improvement of her symptoms.

Discussion

This patient had myocardial bridging with severe coronary vasospasm induced by adenosine during FFR. Adenosine is a potassium channel agonist that acts on specialized receptors in the coronary arteries to decrease vascular tone. The vasodilatory effect transiently eliminates the higher pressure environment of the distal epicardial vascular bed thereby creating a moment of balanced perfusion that allows for increased accuracy in measuring the flow limiting characteristics of an atherosclerotic plaque. Coronary vasospasm has been reported during myocardial perfusion testing in few cases. However, this agent has not been described as a vasospastic culprit during cardiac catheterization in such a low dose as is used for FFR.
The paradoxical response of coronary vasospasm after adenosine is thought to be a result of the abrupt withdrawal of this short acting vasodilator. However, the role of the autonomic nervous system, baroreceptor physiology, catecholamine and neurohormonal influences are also a consideration. The exact mechanism remains unclear.

References.

1. Weissman G Scandrett RM, Howes CJ III et al. Coronary vasospasm during adenosis stress test. J Nucl Cardiol 2004; 11: 747 – 50
2. Golzar J, Mustafa SJ Mvahed A. Chest pain and ST segment elevation 3 minutes after completion of adenosis pharmacological stress testing. J Nucl Cardiol. 2004; 11: 747 - 750

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Recurrent Takotsubo Cardiomyopathy with Varying Wall Motion Abnormalities

Brendan Korpanty[1], Stephanie Ruddy[1], Ross Brubaker[1], Vijai Tivakaran[1], Amy Jiang[2], Timothy Petrie[2], *[1]Grandview Medical Center, Dayton, Ohio, USA, [2]Wright State University, Dayton, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction

Takotsubo cardiomyopathy, also known as apical ballooning syndrome or stress-induced cardiomyopathy is characterized by transient left ventricular dysfunction with regional wall motion abnormalities. It is usually precipitated by stress and typically resolves completely over a period of weeks to months. Patients with takotsubo cardiomyopathy often present with findings consistent with acute coronary syndrome (ACS). In fact, approximately 1-2% of all patients admitted to the hospital for suspected ACS will be found to have takotsubo cardiomyopathy, with the notable difference being normal coronary angiography in the case of takotsubo. Recurrence of takotsubo is rare and typically involves similar wall motion abnormalities. We present here a case of recurrent takotsubo cardiomyopathy with varying wall motion abnormalities.

Case Presentation

A 59 year old female with a history of cirrhosis and portal hypertension secondary to Hepatitis C presented to the emergency department with hematemesis secondary to variceal bleeding. During her admission, she developed respiratory failure that required intubation. An electrocardiogram (EKG) done at the time showed non-specific ST depression, and she had an elevated troponin which ultimately peaked at 18.21ng/ml. Echocardiogram showed severe left ventricular dysfunction with an ejection fraction (EF) of 30%, akinesis of the basal segments and severe mitral regurgitation. She went for emergent cardiac catheterization. Coronary angiography revealed no significant coronary artery disease. Left ventriculogram again showed the akinetic basal segments with a hyperdynamic apex and apical segments, consistent with reverse takotsubo cardiomyopathy. Her heart failure was treated medically, and repeat echo revealed her EF had improved to 45-50% with resolution of the wall motion abnormalities and mitral regurgitation. Six weeks later, the patient presented to the emergency department for acute cholecystitis. During her admission, she developed significant hypotension and was poorly responsive. An EKG showed diffuse T-wave inversion, and labs revealed an elevated troponin, which ultimately peaked at 4.840 ng/ml. She was started on vasopressors for her hypotension and intravenous heparin for presumed acute coronary syndrome. Echocardiogram revealed severely reduced EF of 20-25% with akinesis of the left ventricular apex and mid-ventricular segments with hyperkinetic basal segments. Cardiac catheterization again revealed normal coronary arteries. Her clinical presentation was suggestive of typical takotsubo cardiomyopathy. Her hypotension was treated with IV fluids and intra-aortic balloon pump. Her clinical status gradually improved and she was ultimately discharged home without further intervention.

Discussion

Takotsubo cardiomyopathy is characterized by left ventricular wall motion abnormalities in the absence of coronary artery disease. It is usually associated with significant stress or acute illness. The mechanism for takotsubo cardiomyopathy remains unknown, but most evidence suggests the involvement of stress induced catecholamine release which cause myocardial stunning. Recurrent takotsubo cardiomyopathy is uncommon, estimated to be 2.7-11.4% of all takotsubo cases. Recurrence typically will present with similar regional involvement, and case reports of recurrent takotsubo with different morphologies are very rare. The case presented here is a unique presentation of recurrent takotsubo cardiomyopathy, which was characterized by two distinctly different morphologies.

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Successful Temporary Support of the Right Ventricle After HeartMate II Placement Using a Percutaneous Right Ventricular Assist Device

Bryan Lee[1], Montoya Taylor[1], Bryan Whitson[1], Scott Lilly[1], [1]*The Ohio State University, Columbus, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction: With the advent of left ventricular assist devices (LVAD), patients with end-stage heart failure can be offered an improved quality of life and functional capacity if cardiac transplantation is not an immediate option. Whether used as a bridge to transplantation (BTT) or destination therapy (DT), the survival benefit is superior when compared to continuous inotrope therapy. The hemodynamic and structural changes created by left ventricular assist devices (LVADs) may precipitate right ventricular failure (RVF) which can lead to decreased end-organ perfusion, poor LVAD flow, and higher morality in the early post-operative period. With the advent of percutaneous right ventricular assist devices (RVAD), improvements in short-term outcomes in this population may be attainable. We present a case that demonstrates the successful use of a percutaneous RVAD for temporary support in a patient who developed severe RVF after LVAD implantation.

Case Presentation: A 52-year-old female with a past medical history of chronic nonischemic cardiomyopathy, uncontrolled type 2 diabetes mellitus, chronic kidney disease, presented with acute on chronic NYHA IV heart failure. A multi-disciplinary discussion ensued, and advanced mechanical circulatory support was deemed appropriate and a HeartMate II LVAD was placed as intended destination therapy. Although vasoactive support was initially weaned, she then developed progressive shortness of breath and malaise, with serologies indicative of venous congestion and poor cardiac output (Table 1). In addition, her LVAD had repeated alarms for low-flow. Transthoracic echocardiogram and hemodynamic indices were consistent with RVF. Due to her tenuous status an Impella RP (Abiomed Inc., Danvers, MA) was placed. Over 6 days her right ventricular function improved and she was extubated (Table 2). She was later discharged, and at this time is NYHA II with transplant workup.

Discussion: RVF requiring mechanical support immediately following LVAD placement is a well-established predictor of one-year mortality. Moreover, there have been numerous efforts to develop pre-operative risk tools to quantify the risk of RVF in LVAD candidates. The inherent issue with most of these scores is that they were developed in predominantly BTT patients with pulsatile devices, not reflecting the current LVAD population. Without an effective method to predict RVF, cases like the one we present will likely continue to occur. Instead of relying on traditional RVAD support, which requires an invasive surgical approach, the advent of the percutaneous RVAD allows practitioners to give temporary support in a safer fashion. We demonstrate that a percutaneous RVAD can stabilize those who suffer from severe RVF after LVAD implantation and enable de-escalation of life-support measures. The hope is this trend will continue and improve short-term mortality in the LVAD population.

Table 1: Laboratory Data Pre and Post Impella RP Implantation

	Day -2	Day -1	Day of Implant	Day +1	Day +2	Day +3	Day +4	Day +5	Day +6
Creatinine	0.76	1.1	1.34	1.61	1.7	2.34	2.27	2.02	1.67
Total bilirubin	0.5			1.1	1.5	2.1	2.2	3.9	2.4
AST	24			376	250	164	98	112	64
ALT	13			244	216	173	121	85	67
INR	1.6	1.4	3.5	2.4	2.4	2.6	3.1	4.3	1.5

Table 2: Measurement of Right Ventricular Function

	Pre-Impella RP	Post-Impella RP
RA (mmHg)	27	18
PA (mmHg)	39/26/31	41/30/32
PCWP (mmHg)	26	28
RA/PCWP or LAP	1.04	0.64
PAPi	0.52	0.61
RV S' (cm/sec)	5	Unable to attain

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A Case of Refractory New Onset Arial Fibrillation in a Patient with Iron Overload Treated with Iron Chelation Therapy

Mark Marinescu[1], Sharon Roble[1], Naga Garikipati[1], Mahmoud Houmsse[1], [1]*The Ohio State Univesrity, Columbus, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

A 29 year old male with a history of sickle cell disease requiring multiple transfusions, iron overload, and restrictive cardiomyopathy, who presents from outpatient clinic to the hospital with new onset atrial fibrillation. A recent cardiac MRI showed severe burden of cardiac and hepatic iron deposition, moderate to severe LV and RV dysfunction, as well as severe biatrial enlargement.

The patient was initially treated with rate controlling agents. DC cardioversion was performed before and after amiodarone loading. However, following both attempts, the patient converted back to atrial fibrillation.

The patient was then started on an aggressive iron chelation therapy consisting of IV desferrioxamine 40-50mg/kg/24hr and oral deferiprone 25mg/kg every eight hours. The patient continued to receive amiodarone for a total of 5 grams prior to it being discontinued given concerns for worsening hepatic dysfunction.

Two days after initiating aggressive iron chelation therapy the patient spontaneously converted to sinus rhythm and maintained sinus rhythm to discharge. Repeat cardiac MRI prior to discharge showed decreased cardiac and hepatic iron burden.

Several case reports have suggested that aggressive iron chelation therapy may improve cardiac function and reduce the burden of atrial fibrillation in patients with increased cardiac iron deposition. Here we present a case of a patient with severe cardiac iron overload due to multiple blood transfusions with new onset atrial fibrillation who spontaneously converted to and maintained sinus rhythm after treatment with aggressive iron chelation therapy.

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Hemopericardium and Hemothorax Secondary to Spontaneous Coronary Sinus Lead Perforation 13 Years After CRT-D Implantation

James Monaco[1], Subha Raman[1], Patricia Allenby[1], [1]*Ohio State University Medical Center, Columbus, Ohio, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Patient RC was a 90 year old man who presented to our emergency department with acute syncope and hypotension. The patient had a known history of dilated cardiomyopathy with EF as low as 20%, which had experienced excellent recovery to >50% after placement of a Boston Scientific biventricular pacing system in 2003. Since initial placement, he had undergone two generator changes in 2007 and 2013; however, all original leads remained in place. These consisted of a Pacesetter 1488T Tendril SDX in the right atrial position, a St Jude 1581 Riata dual-coil defibrillation lead in the right ventricular position, and a Guidant / CPI Del Caribe 4813 Easytrak LV-1 in the lateral cardiac vein. Although the device had continued to pace normally throughout its service life, increased noise had been noticed on his RV lead two weeks prior to his presentation and concern had been raised that this could represent insulation failure or fracture of the recalled Riata RV lead.

The patient's blood pressure initially stabilized with fluid resuscitation. An initial CXR showed a small left sided pleural effusion. A non-gated CT pulmonary angiogram was performed and appeared to show hemopericardium and hemothorax with possible perforation of the RV lead through the RV apex. An emergent transthoracic echocardiogram was performed and confirmed the presence of a large volume of complex fluid in the left thorax and clot in the pericardium, but was unable to clearly confirm or rule out lead perforation. The thoracic aorta was well visualized on the echo and the CT without evidence of dissection. Initial labwork showed acutely low hemoglobin. The patient developed recurrent severe hypotension at this point, and he received 4 units of unmatched pRBCs which again achieved temporary hemodynamic stabilization. A follow-up CXR showed significant enlargement of the left sided effusion. Pacemaker interrogation at this time revealed normal, stable impedance, sensing, and capture of all leads.

Although it seemed unlikely for a lead perforation with pericardial laceration to occur spontaneously so many years after placement, we had no alternate diagnosis to explain the patient's rapidly developing hemothorax. This was explained to the patient and family. Given the patient's age and co-morbidities, his expected mortality with thoracic surgery was felt to be prohibitive. The patient and family did not wish to pursue surgery, and elected instead to receive inpatient hospice care. The patient passed away within 48 hours.

Given the possibility of an unusual complication from a recalled pacemaker lead, we requested the family allow an autopsy, to which they consented. Surprisingly, this revealed that while lead perforation and laceration of the pericardium had occurred, it was the coronary sinus lead at fault, and not the recalled and possibly malfunctioning Riata RV lead that appeared to be the culprit on CT. Gross pathology showed LV lead perforation of the lateral cardiac vein, overlying laceration of the pericardium, and hemopericardium and hemothorax.

Lead perforations most frequently occur as a complication of device placement. To our knowledge, this is the first case described of a coronary sinus lead spontaneously perforating after so many years of normal operation, and demonstrated the difficulty of diagnosing a coronary sinus lead perforation in the emergency setting.

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Incorrect ventricular lead placement into the systemic right ventricle of a patient with D-transposition of the great vessels after Mustard procedure

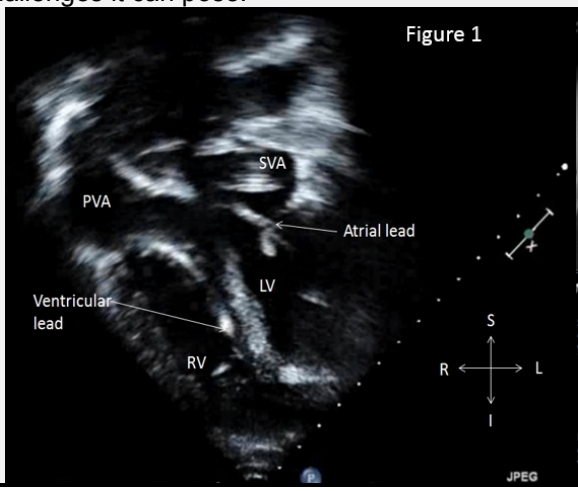
Adam Powell[1], Thomas Kimball[0], [1]*Cincinnati Children's Hospital, Cincinnati, OH, USA*

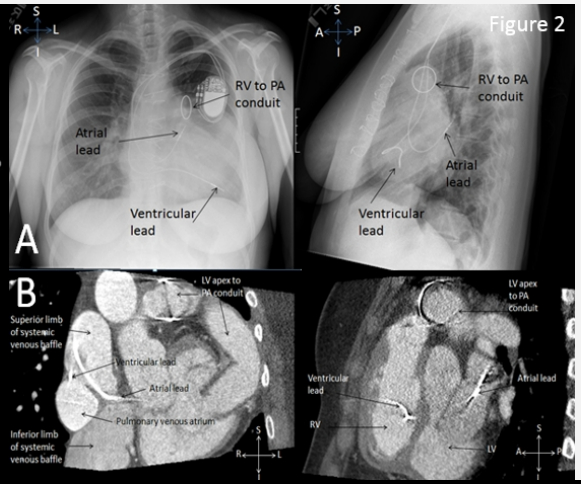
Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction: Incorrect pacemaker lead placement into the systemic ventricle is a complication that has rarely been described in patients with d-transposition status-post atrial baffle palliation. We present a case of ventricular lead misplacement in the systemic right ventricle of a patient with D-transposition of the great arteries after Mustard procedure. This case demonstrates the challenges with proper imaging of lead placement in patients with atrial baffles and long-term management of a lead in the systemic ventricle.

Case Presentation: A 39-year-old female with history of D-transposition of the great arteries presented to the clinic in 2010 to establish care after previously being treated at a different institution. Her past medical history was remarkable for D-transposition of the great arteries which initially required a Blalock-Hanlon septectomy in 1971 followed by a Mustard procedure in 1972. In 2005, she had a dual chamber pacemaker placed at another institution secondary to sinus node dysfunction. At this visit, her routine echocardiogram demonstrated mild obstruction of the superior limb of the systemic venous baffle and a small pulmonary venous baffle leak. Additionally, the echocardiogram demonstrated a previously unseen finding of the ventricular lead coursing across the atrial baffle, from the systemic venous atrium entering the pulmonary venous portion of the atria, then coursing through the tricuspid valve with its tip in the systemic right ventricular apex (Figure 1). A chest x-ray (Figure 2A) and cardiac CT scan (Figure 2B) performed after the echocardiogram confirmed lead placement in the more anterior systemic right ventricle. The abnormal placement of her ventricular pacing lead in the systemic right ventricle raised concern for thrombus formation and future thromboembolic events. It was felt that lead removal would be safest with surgery but carried significant operative risk compared to the current risk of thromboembolism. We elected to continue long-term anticoagulation with aspirin following closely for thromboembolic phenomenon. She has been followed yearly in the clinic over the past five years during which she has not had any significant thromboembolic phenomenon.

Discussion: While there are multiple reports of incorrect ventricular lead placement in patients with structurally normal hearts, there are limited data on the management and intervention strategies of incorrectly placed transvenous pacing leads in the systemic ventricle of patients with transposition of the great arteries after atrial switch operation. There is only one report of improper lead placement in the setting of an atrial switch. This was an adult patient with a Senning repair of D-transposition of the great arteries who had a misplaced ventricular lead diagnosed 3 years after implantation. While this patient was asymptomatic from thromboembolic complications, he did have his misplaced lead extracted secondary to concerns for future thromboembolic events (Kuppahally SS, et al, Circulation 2010). That case, like ours, was associated with a delay in diagnosing a misplaced lead emphasizing the need for thorough, compulsive imaging. We recommend diagnosis with either fluoroscopy during lead implantation or post-implantation chest x-ray. This patient's misplaced leads were diagnosed by echocardiogram which should not be the initial test of choice however the astute interpretation by the echocardiographer allowed for this unexpected problem to be diagnosed. As for the thromboembolic management of these patients, there is no consensus in the management of inadvertent lead placement into the left ventricle of patients with structurally normal hearts. Some advocate lead removal and others recommend life-long anticoagulation with either Coumadin or aspirin. In summary, this report hopes to increase consciousness about proper lead positioning in patients with transposition of the great arteries who have been treated with an atrial switch operation and to demonstrate the diagnostic challenges it can pose.





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Title: (Do not enter author details)

Early calcification of a bioprosthetic mitral valve

Juan Pablo Rodriguez-Escudero[1], Diego Alcivar Franco[1], Roger Chaffee[1], Kevin Silver[1], [1]*Summa Cardiovascular Institute, Akron, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction

Calcification is a major cause of long-term bioprosthetic heart valve failure. Clinically important bioprosthetic valve calcification in the mitral position usually occurs approximately 10 years or longer after the implantation. We report a case of unusually early degenerative mitral bioprosthetic calcification resulting in severe mitral valve stenosis and resultant heart failure.

Case presentation

A 57 year old woman with a 2 year old bioprosthetic mitral valve was seen in the outpatient clinic prior to a possible renal transplant. She had a history of hypertension, type 2 diabetes mellitus, dyslipidemia, and renal failure requiring hemodialysis. The hemodialysis pre-dated the mitral valve surgery. She had symptoms of New York Heart Association class III heart failure at the time of that visit. Due to symptomatic degenerative mitral valve insufficiency she had received a 27 mm Carpentier-Edwards PERIMOUNT mitral valve prosthesis. She also underwent a percutaneous coronary intervention about the same time. This was done at another institution. Her symptoms of heart failure were progressive and in retrospect had begun as early as 6 months after valve implantation. A transthoracic echocardiogram revealed a left ventricular ejection fraction of 45%, a mean mitral valve gradient of 18 mmHg, a calculated mitral valve area of 0.6 cm2. There were no other valvular abnormalities. A transesophageal echocardiogram with three dimensional imaging confirmed the presence of severe calcific mitral stenosis revealing a calculated mean gradient of 22 mmHg and valve area of 0.8 cm2. There was mild central mitral insufficiency and no left atrial appendage thrombus. A diagnostic right and left cardiac catheterization revealed nonobstructive coronary artery disease, patent left anterior descending and right coronary stents, a systolic pulmonary artery pressure of 54 mmHg, a pulmonary artery wedge pressure of 36 mmHg and a left ventricular end diastolic pressure of 12 mmHg. These findings also confirmed severe mitral stenosis. Her case was presented at our multidisciplinary valve conference. The traditional approach would be to replace the stenotic bioprosthetic mitral valve with a mechanical valve and commit to long-term warfarin therapy. Due to her high-risk of operative mortality (STS score > 8), the recommendation was to offer a novel approach for mitral valve replacement, which is a transcatheter valve in valve implantation placed upside down in mitral position. This decision was chosen by the Heart Team Approach (Cardiologist, Cardiothoracic Surgery and Interventionist) as recommended by the ACC/AHA guidelines. It was believed that a second bioprosthetic valve would survive longer after renal transplantation.

Conclusion

Early bioprosthetic valve calcification and clinically important degeneration has been reported as early as 3 years in certain patients. Our patient demonstrates that this can occur as early as 2 years. We believe her relatively young age and hemodialysis accelerated the degeneration. This case illustrates this problem, the potential opportunity for a novel approach to valve replacement, and the complicating issues of organ transplantation.

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Title: (Do not enter author details)

How Many Patent Ductus Arteriosus Did You Say?

Tamika Rozema[1], Ravi Ashwath[1], [1]*Rainbow Babies and Children's Hospital, Cleveland, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Case Presentation

A 22 year old woman was referred for abnormal obstetrical ultrasound at 31 weeks gestation. Fetal echocardiogram demonstrated double outlet right ventricle, pulmonary atresia with malposed great arteries, and a large ventricular septal defect. The baby was born at 38 weeks. Prostaglandin infusion was started after delivery. Transthoracic echocardiogram demonstrated situs solitus, double outlet right ventricle with malposed great arteries, and pulmonary atresia with discontinuous pulmonary arteries supplied by bilateral patent ductus ateriosus. In particular, the left pulmonary artery arose from the reverse angle patent ductus ateriosus from the undersurface of the transverse aorta (Figure 1A). The right pulmonary artery arose from the patent ductus ateriosus that inserted at the base of the right innominate artery (Figure1B). Cardiac CTA confirmed this anatomy.

Due to family's desire for a bloodless surgery, he underwent cardiac catheterization at 6 days of life for palliation with bilateral ductal arteriosus stenting via a femoral approach with 3 Integrity© coronary artery stents placed in the right patent ductus arteriosus and 2 Integrity© coronary artery stents placed in the left patent ductus arteriosus (Figure 2). The patient was discharged to home after 6 days post-procedure. Subsequent pulmonary over circulation was pharmacologically managed with furosemide and enalapril. Complete repair is anticipated by 5 months of age.

Discussion/Conclusion

Bilateral patent ductus arteriosus are an uncommon abnormality, usually associated with pulmonary atresia and discontinuous branch pulmonary arteries, with approximately 29 cases reported in the current literature. Previously described approaches to management include palliation with transcatheter ductal stenting to complete repair. This is the first reported case of stenting of the bilateral ductus arteriosus via the femoral artery approach. Baspinar et al reported a case of bilateral ductal stenting with the left duct approached from the right femoral artery and the right duct from the right carotid artery. Complimentary imaging modalities including echocardiography, CTA or MRA are beneficial for pre-procedural planning.

This case underscores the need for accurate diagnosis by complimentary imaging modalities and also illustrates the feasibility of initial transcatheter palliation as an alternative to cardiac surgery requiring cardiopulmonary bypass.

Images:

B. CT 3D reconstruction

Image 1: Left patent ductus arteriosus (1A), Right patent ductus arteriosus (1B)

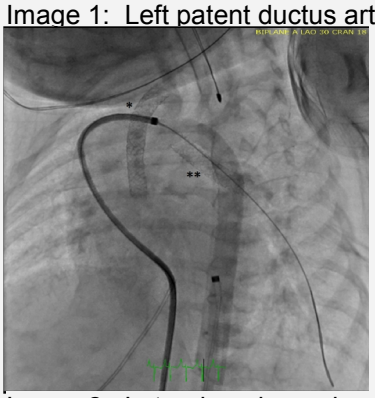

Image 2: Lateral angiography showing the stents in the right (*) and left (**) patent ductus arteriosus

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Title: (Do not enter author details)

Atrioesophageal fistula after radiofrequency ablation for atrial fibrillation.

Pargol Samani[1], Jordan Bohin[1], Pankaj Sharma[2], [1]*Kettering Medical Center, Kettering, Ohio, USA,* [2]*Dayton VA Medical Center, Dayton, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction:
Radiofrequency ablation (RFA) for atrial fibrillation has been shown to be more effective at maintaining sinus rhythm in selective patients than antiarrhythmic therapy[1]. Changes to the most recent AHA/ACC/HRS guidelines have moved RAF to first-line therapy for symptomatic paroxysmal AF refractory or intolerant to at least one antiarrhythmic drug[2]. Of the known side effects, atrioesophageal fistula is rare occurring in about 0.04% of cases[3]. When it occurs, however, the mortality is as high as 80% [4] . Here we are presenting a case whose RFA procedure convoluted with this rare complication.

Case presentation: Ohio
A 47 year old man with drug refractory persistent atrial fibrillation (AF) who failed multiple electrical cardioversions underwent Radiofrequency ablation (RFA) of wide circumferential areas in the left atrium (LA) to isolate the pulmonary veins. Intracardiac echo showed LA dimension of 53x43 mm with ejection fraction of 55%. The patient tolerated the procedure well with no immediate perioperative complications. One month after the procedure, the patient presented with weakness and numbness of his extremities and speech disturbance. He also complained of persistent cough and vomiting and hoarseness since RFA procedure. On initial presentation, the patient was febrile, but no neurological deficit was noted at that time. On the second day of admission, patient developed right side weakness associated with facial palsy, aphasia and dysarthria. He then developed septic shock. MRI of the brain showed extensive bilateral cerebral, cerebellar, and brainstem infractions (Fig.1). Broad-spectrum IV antibiotics were initiated. Blood cultures grew Streptococcus viridans as well as Candida Glabrata. Transesophageal Echocardiography (TEE) showed 1 cm mobile lesion, with small stock, in the posterior wall of the left atrium near the pulmonary vein and left atrial appendage (Fig2-3). CT scan of the chest also revealed esophageal perforation near the pulmonary vein with small amount of adjacent soft tissue gas directly posterior to the left atrium (Fig.4). Esophagram with contrast demonstrated Atrio-esophageal Fistula (Fig.5). Patient was recommended for surgical repair of the LA and esophagus, however, died before surgical correction could be attempted.

Discussion:
Atrioesophageal fistula remains a rare and uncommon complication of RFA for AF however one that carries an alarming mortality risk. Our case highlights several of the important features when considering an atrioesophageal fistula following RFA. With recent guidelines emphasizing the role of catheter ablation, it is likely the incidence will increase. A high index of suspicion is required to ensure adequate and timely diagnosis and optimal treatment. Diagnosis is usually confirmed by CT with IV contrast or MRI. EGD should be avoided to prevent air embolism. Treatment consists of broad spectrum antibiotics and, in almost all cases, surgical repair of the left atrium and resection of the damaged portion of the esophagus is required.

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Title: (Do not enter author details)

Massive Pulmonary Embolism Masquerading As Acute Coronary Syndrome

Aldo Schenone[1], Michael Hoosien[1], Julie Rosenthal[1], Nael Hawwa[1], Leslie Cho[1], [1]*Cleveland Clinic, Cleveland, Ohio, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Background

Acute extrinsic compression of the left main trunk (LMT) by the pulmonary artery (PA) is a very unusual entity (1,2,3,4). Its prevalence in the setting of pulmonary hypertension ranges from 5-44% (1). PCI is an effective treatment strategy in cases of LMT compression due to severe pulmonary hypertension. (1,5)

Case Report

A 53-year-old male with a history of chronic thromboembolic pulmonary hypertension (CTEPH) presented with severe, right-sided pleuritic chest pain, non-productive cough and progressive shortness of breath. He was initially admitted to the medical intensive care unit the setting of evolving shock state. Chest X-ray revealed right lower/mid lobe opacity with pleural effusion. A 12-lead electrocardiogram revealed sinus tachycardia with a complete right bundle branch block, 2mm ST-segment elevation in lead AVR, and 2-3mm ST-segment depression in the anterolateral precordial and inferior limb leads. Additional pertinent laboratory included a leukocyte count of 41,000/uL, serum creatinine of 1.7mg/dL, serum lactate of 3.1 mmol/L and elevated cardiac biomarkers (CK-MB 395 and Troponin T 12). Given the finding of global ischemia on the ECG in conjunction with rising cardiac biomarkers, the patient was transferred to the coronary intensive care unit.

Decision-making

A transthoracic echocardiogram reveled a significant reduction in the left ventricular ejection fraction from prior studies (37% from a baseline of 60%), with severe global hypokinesis. Additionally, right ventricular systolic function was felt to be moderately decreased. Interestingly, it was discovered that the patient had developed worsening dilatation of the main PA (7.6cm in diameter compared to 6.5cm on a prior study) with spontaneous echo-contrast in both the main and right PA. Based on ECG, cardiac biomarker and echocardiographic findings, it was felt at this time that the patient had suffered an acute pulmonary embolus with worsened acute dilatation of the PA, and that this was causing compression of the left main coronary artery. A pulmonary artery catheter was inserted to better delineate shock state. Initial hemodynamics favored cardiogenic shock with markedly elevated pulmonary artery (PA) pressures (80/65mmHg, mean 73mmHg) and a calculated cardiac index of 1.7 L/min/m2 (Fick method). A pulmonary capillary wedge pressure was not obtained. An intraaortic balloon pump was placed emergently, and the patient was taken to the cardiac catheterization laboratory. Coronary angiography revealed a severe 95% stenosis of the proximal left main trunk with no other obstructive coronary disease. Urgent coronary artery bypass grafting was discussed, but given the high operative risk, percutaneous coronary intervention (PCI) was performed instead. Soon thereafter the IABP was discontinued, and on hospital day 8 the patient was weaned from vasopressors and transferred to the regular nursing floor. Repeat echocardiography demonstrated preserved left ventricular systolic function with an ejection fraction of 55%.

Conclusion

LMT compression can provoke or worsen ischemia in the context of PH, and should be strongly considered in cases of worsening angina or acute coronary syndrome since it represents a treatable cause of ischemia and left ventricular dysfunction (1). The optimal management strategy remains uncertain, and includes coronary artery bypass grafting, percutaneous coronary intervention, pulmonary artery thromboembolectomy, and transplantation (1). While bypass grafting remains the gold standard for left main stenosis secondary to atherosclerotic disease, individuals with compressive disease and severe pulmonary hypertension represent a very high-risk surgical group, and PCI can generally be performed with acceptable risk and low restenosis rates (1,5).

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Double Trouble: Renal artery stenosis due to fibromuscular dysplasia, atherosclerosis or both?

Hemindermeet Singh[1], Faraz Khan Luni[1], Owais Khawaja[1], Hafeezuddin Ahmed[1], Syed Sohail Ali[1], Mohammed Taleb[1],
[1]*Mercy St. Vincent Medical Center, Toledo, Ohio, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction

Atherosclerosis and fibromuscular dysplasia (FMD) are two important causes of renal artery stenosis (RAS) which can lead to resistant hypertension. However, these are anatomically and physiologically different entities. They differ in terms of patient characteristics, diagnosis and management. We present a unique case of a 64-year-old female who presented with resistant hypertension and found to have both atherosclerotic RAS (ARAS) and FMD at same time.

Case Presentation

A 64-year-old woman with history of coronary artery bypass in past was referred for uncontrolled hypertension. She was treated with chlorthalidone, carvedilol, losartan and was intolerant to many antihypertensive including amlodipine, hydralazine and clonidine. Renal artery ultrasound done as a work up for secondary hypertension revealed greater than 60% stenosis in left renal artery with no significant stenosis in right renal artery. Bilateral renal artery angiogram was performed which showed two different etiologies of RAS. The left renal arteriogram demonstrated 80-90% atherosclerotic ostial stenosis [Fig.1]. The lesion caused significant pressure dampening. Intravascular ultrasound (IVUS) confirmed the plaque and it was successfully treated with stent placement reducing stenosis to 0%. The right renal arteriogram however, showed beaded appearance of the artery suggestive of FMD [Fig. 2]. This was also confirmed with IVUS which showed intraluminal fibrous webs consistent with FMD. FFR (Fractional Flow Reserve) was performed to check for functional narrowing of artery and it was 0.62 suggesting significant obstruction. It was hence treated with balloon angioplasty. The repeat FFR was 1.0 suggesting restoration of normal flow. Patient tolerated the procedure well and her BP was noted to improve the subsequent day. She was discharged home in stable condition with uneventful follow up.

Discussion

ARAS and FMD are two different diseases. ARAS is usually seen in elderly patients with risk factors for cardiovascular disease and is treated with medical therapy, risk factor modification and revascularization with stents in refractory cases. FMD on the other hand is seen in younger patients and is treated with balloon angioplasty. To our knowledge, this is first reported case of RAS due to both atherosclerosis and FMD in the same patient.

Conclusion

Renal artery stenosis secondary to atherosclerosis and FMD can simultaneously occur and may present as resistant hypertension.

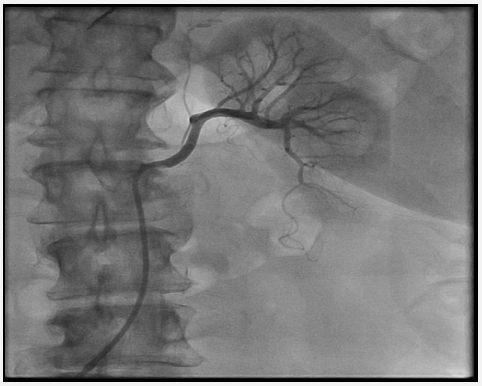

Fig.1: Left renal arteriogram showing 90% atherosclerotic ostial stenosis of left renal artery

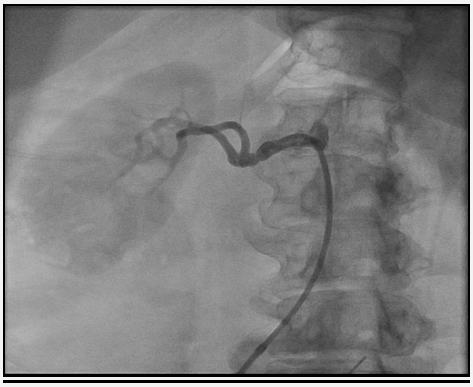

Fig.2: Right renal arteriogram showing beaded appearance of right renal artery suggesting fibromuscular dysplasia

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Use of SSRI and Midodrine in a patient with Auotonomic Instability 2/2 Compressive Squamous Cell Carcinoma and Pain

Thomas Vacek[1], Kyle Ball[0], [1]*Wright state University, Dayton, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

A rare cause of reflex syncope is metastatic cancers involving the head and neck. These can irritate the glossopharyngeal nerve and lead to glossopharyngeal neuralgia (GPN) with associated syncope. This type of syncope is difficult to treat, since it commonly involves both a vasodepressor and cardioinhibitory response, and typically requires removal of the irritative focus. A 52-year old male was admitted after a syncopal episode at home. He endorsed a five-week history of progressively worsening headaches prior to arrival. These headaches seemed to worsen when he turned his neck in certain directions, particularly to the left. He also admitted to over a 40-pound weight loss and occasional drenching night sweats over the past six months. In the emergency department, his heart rate was noted to drop into the 20's, which was associated with a blood pressure as low as 60/31mmHg, which responded to IV atropine. Physical examination revealed a large ulcerative lesion in the left tonsillar area. After biopsy of the lesion, a diagnosis of stage IV squamous cell carcinoma of the neck was made; CT angiogram and PET CT confirmed involvement in the posterior tongue extending to the left palatine tonsil in addition to the left jugular chain. The patient was started on cisplatin and radiation therapy, but continued to have episodes of syncope associated with bradycardia and hypotension. He required repeated doses of atropine, and ultimately was started on a dopamine drip to maintain adequate blood pressure and heart rate. After a failed trial of benztropine, the patient was started on sertraline and midodrine with resolution of syncope. In this case, the patient experienced resolution of symptoms after being started on midodrine and sertraline. Midodrine works by stimulating alpha-adrenergic receptors leading to an increase in vascular tone. Sertraline is thought to work centrally via down-regulation of 5-HT1a receptors in the brain, which blunts the response to baroreceptors yielding a steady blood pressure. For patients not candidates for surgery or chemotherapy, or those awaiting definitive treatment, this could be a potential option that is associated with relatively few unwanted side effects.

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Understanding Fetal Cardiac Output and Pulsatility Indices in Different Congenital Heart Defects. The Effect of Fetal Hemodynamics on Fetal Growth

Tarek Alsaied[1], Stephanie Tseng[1], Eileen King[1], Eunice Hahn[1], Allison Divanovic[1], Mounira Habli[1], James Cnota[1], *[1]Cincinnati Children's Hospital, Cincinnati, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background: Combined cardiac output (CCO) and cerebral autoregulation impact on fetal growth may vary in different congenital heart defects. This study compared serial measures of fetal growth; CCO, middle cerebral artery pulsatility index (MCA-PI) as an indicator for cerebral vascular resistance in four groups: hypoplastic left heart (HLHS), non-HLHS single ventricle (SV), transposition of great arteries (TGA) and normal controls.

Methods and results: Fetal echocardiograms from 109 fetuses were reviewed: HLHS (n=30), SV (n=20), TGA (n=17) and controls (n= 42). CCO was calculated using valvar area, velocity time integral and heart rate. MCA-PI was calculated using systolic, diastolic and mean velocities. Anthropometric measures were recorded. Regression models were used to study CCO, PIs and fetal anthropometric trends over gestational age. Multivariate analysis was used to determine the association of CCO and MCA-PI at 30 weeks with birth weight, length and head circumference z scores.
As expected CCO increased in all 4 groups through gestation but plateaued in HLHS and SV at the end of gestation. MCA-PI values were lower in HLHS and higher in SV through gestation compared to TGA and controls suggesting a different cerebral blood distribution. At the end of gestation fetal weight plateaued in HLHS and SV (similar to CCO curves) and head circumference plateaued in all groups but controls. CCO positively correlated with birth weight z scores ($p=0.003$) and birth length Z scores ($p=0.04$). Birth head circumference z scores differed by group with no significant correlation with CCO or MCA-PI.

Conclusions: CCO positively correlates with birth weight and may provide one mechanism to understand differences in fetal growth in congenital heart defects. A brain sparing mechanism in HLHS is supported by lower cerebral vascular resistance.

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Title: (Do not enter author details)

Long Term Mortality Rates of Premature Coronary Artery Disease in Young U.S. Veterans

Thein Tun Aung[1], Samuel Roberto[1], Ajay Agarwal[2], Jonathan Pollock[3], Ronald Markert[1], Amish Patel[1], *[1]Wright State University, Dayton, OH, USA, [2]Dayton VA Medical Center, Dayton, OH, USA, [3]Wright-Patterson Air Force Base, Dayton, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background

The mortality trends from premature coronary artery disease (CAD) in young US Veterans are not well defined.

Methods

We retrospectively analyzed 261 consecutive US Veterans who were younger than 55-years old from a cohort of 1193 Veterans who underwent cardiac catheterization from October 2001 to January 2005. Primary outcome was all-cause mortality. This data was also examined for prevalent risk factors and demographics.

Table 1.

Variables	
Number of patients	261
Age in years (mean ± SD)	49.2 ± 4.7
BMI (mean ± SD)	30.9 ± 6.2
Hypertension (%)	79.2
Smoking (%)	59.4
Mental Disorder (%)	41.8
Alcoholism (%)	19.5
Family history of premature CAD (%)	41.5
Diabetes (%)	37.5
Hepatitis C infection (%)	9.2
Obstructive CAD (%)	61.7
Left Ventricular Ejection Fraction % (mean ± SD)	48 ± 13

Results

Baseline characteristics are summarized in Table 1. The mean follow up duration was 124±43 months. Indication for cardiac catheterization was acute coronary syndrome 16.8%, positive stress test 76.2%, and cardiomyopathies 7%. There was high prevalence of hypertension, smoking, alcoholism, mental disorder, and concomitant Hepatitis C virus (HCV) infection. All-cause mortality rate was 29.1%, which increased to 44.4% in the subgroup with combined history of diabetes mellitus, smoking, and low left ventricular ejection fraction.

Discussion

Data concerning the mortality rates of CAD in younger populations is sparse. Preliminary evidence in the general population suggests the mortality trend of young CAD is worsening in association with increased prevalence of metabolic syndrome and hypertension. However, there is no well-defined mortality data for young US Veterans. This single center retrospective study revealed high long-term mortality rates in young US Veteran population with premature CAD. The study also showed high prevalence of hypertension, smoking, alcoholism, mental disorder and HCV infection. This observation calls for a more aggressive focus on primary, secondary preventive measures, and mental health support to young US Veteran population.

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Title: (Do not enter author details)

Prevalence of Cognitive Impairment in Patients admitted to the Cardiac Units and its impact on the health outcomes: A Pilot Study

Bishnu Dhakal[1], Shalini Ratnagiri[1], Sadeer Al-Kindi[1], Tamra McDevitt[1], Chantal ElAmm[1], Michael Zacharias[1], Monique Robinson[1], Guilherme Oliveira[1], Mahazarin Ginwalla[1], *[1]Division of Cardiovascular Medicine, University Hospitals Harrington Heart and Vascular Institute, University Hospitals, Case Medical Center, Case Western Reserve University, Cleveland, Ohio, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background:
The Mini-Cog test is an easy and effective tool to screen for cognitive impairment (CI). Patients with cardiovascular diseases often have complex medication regimens and cognitive impairment may significantly impact their medication compliance and can lead to worse health outcomes.

Objectives:
1) To determine the prevalence of cognitive impairment in patients with cardiac diagnoses in the acute care setting.
2) To determine if an abnormal Mini-Cog test correlates with a higher re-hospitalization rate and a longer length of stay.

Methods:
A pilot study was conducted in 59 consecutive patients admitted to a cardiac telemetry step-down unit in a tertiary care teaching hospital with cardiac diagnoses including myocardial infarction, heart failure, valvular heart disease, and post-cardiac surgery. The Mini-Cog test consisting of a 3-item recall and a clock-drawing test was administered during their stay either during admission or discharge. Test results were scored on a 5-point scale (1 point for each word recalled, 2 points for clock drawing). A score of ≤2 was suggestive of CI. Patients were followed over a 30-day period after discharge for any readmission. The outcomes that we measured were prevalence of CI, length of stay (LOS) and 30-day re-admission rate.

Results:
Our patient cohort consisted of mostly elderly population (mean± SD = 67 ± 13 yrs) and the incidence of CI was 35.6% in those patients admitted with cardiac diagnoses. Most of those patients with CI≤2 were impaired in the clock drawing test. The patients with CI were older than those without CI (73±14 vs 64± 12 yrs, p= 0.02). Interestingly, a larger percentage of females compared to males had CI (62% female in CI≤2 vs 47% female in CI≥3). The 30-day readmission rate and hospital length of stay was not statistically different in patients with CI compared to those without CI but some patients were lost to follow up. Patients with CI were discharged on a more complex medication regimen, 14.3 ± 4.1 medications in patients CI vs 9.0 ± 5.8 in those without CI.

Conclusions:
More than a third of patients admitted to the tertiary care hospital cardiac unit have CI but paradoxically the same patients were discharged in a higher number of medications compared to those without CI. The Mini-Cog test is a simple and effective tool to identify those patients with CI and guide the implementation of measures to improve compliance and post-hospitalization care. Future studies with larger number of patients are required to replicate these findings and to identify appropriate interventions in those patients with CI to improve both short-term and long-term outcomes.

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Title: (Do not enter author details)

Non-ST Elevation Myocardial Infarction in ARIC - Mortality Beyond Comorbidities

Avirup Guha[1], Henry Chang[1], Emily C O'Brien[2], Julie K Bower[1], Elliot D Crouser[1], Wayne D Rosamond[3], Subha V Raman[1], Randi Foraker[1], *[1]The Ohio State University, Columbus, OH, USA, [2]Duke University, Durham, NC, USA, [3]University of North Carolina, Chapel Hill, NC, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background: Non-ST segment elevation myocardial infarction (NSTEMI) comprises the majority of acute coronary syndromes worldwide, yet management remains heterogeneous compared to those with ST-elevation myocardial infarction (STEMI). It is not known to what degree post-MI mortality is driven by comorbid conditions.

Objectives/Hypothesis: To quantify mortality after incident NSTEMI and STEMI compared to matched controls not experiencing MI.

Methods: Survival from MI event to death, loss of follow-up, or end of 2011 was assessed in 422 (NSTEMI) and 189 (STEMI) Atherosclerosis Risk In Communities cohort participants versus 18386 and 8690 matched participants who did not experience NSTEMI/STEMI, respectively. Patients were matched for baseline age group, sex, and race/study community. Cox proportional hazards regression (frailty models) was performed to estimate hazard ratios and 95% confidence intervals for all-cause mortality. The influence of covariates was tested in a full multivariable model.

Results: Controlling for age, sex and race, the mortality hazard ratio (HR) for participants with vs. without NSTEMI was 2.44 (2.15, 2.78); similar HR for participants with vs. without STEMI was 2.25 (1.86, 2.72). Controlling additionally for all other covariates (figure 1), the mortality HR was 1.78 (1.55, 2.04) for NSTEMI and 1.79 (1.47, 2.19) for STEMI. The Kaplan-Meier survival in NSTEMI/STEMI showed worse survival with both NSTEMI and STEMI vs. MI-free participants (Figure 2a and 2b).

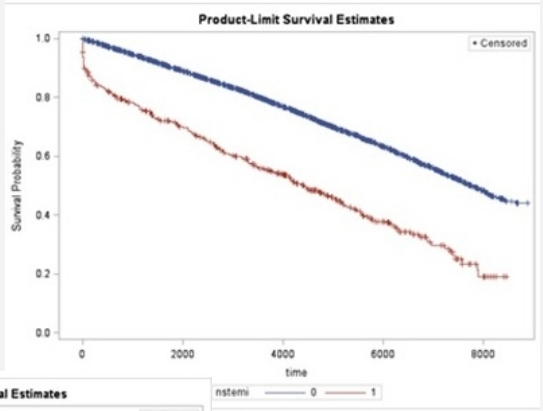
Conclusions: Both NSTEMI and STEMI events themselves incur significantly higher mortality risk beyond what can be attributed to comorbid conditions. Consideration of more aggressive measures to limit myocardial damage in patients with NSTEMI warrant consideration.

Figure 1 – Hazard ratios and 95% confidence intervals for mortality among participants with NSTEMI and STEMI in a multivariate

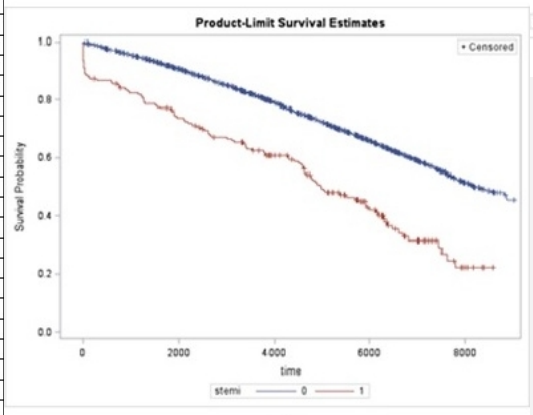
	NSTEMI (p<0.05)	STEMI (p<0.05)
Age, years		
60-64	3.04 (2.74, 3.38)	3.58 (2.88, 4.46)
55-59	1.96 (1.75, 2.18)	2.43 (2.03, 2.91)
50-54	1.44 (1.28, 1.63)	1.71 (1.43, 2.04)
45-49	Ref	Ref
Sex		
Male	1.28 (1.18, 1.38)	1.41 (1.20, 1.65)
Female	Ref	Ref
Race/study community	Not significant	Not significant
ACS event itself		
Yes	1.78 (1.55, 2.04)	1.79 (1.47, 2.19)
No	Ref	Ref
Body mass index		
Obese	1.12 (1.04, 1.21)	1.20 (1.07, 1.34)
Overweight	Not significant	Not significant
Normal	Ref	Ref
Creatinine		
High	Not significant	3.58 (2.06, 6.23)
Elevated	Not significant	3.89 (2.24, 6.72)
Normal	Ref	Ref
Education		
Less than high school	1.17 (1.10, 1.25)	1.22 (1.12, 1.34)
High school or greater	Ref	Ref
Smoking status		
Current	2.22 (2.07, 2.38)	2.34 (2.12, 2.59)
Former	Not significant	2.44 (2.21, 2.69)
Never	Ref	Ref
Cancer		
Yes	1.21 (1.08, 1.35)	1.25 (1.07, 1.46)
No	Ref	Ref
Diabetes		
Yes	1.84 (1.70, 1.99)	1.83 (1.64, 2.05)
No	Ref	Ref
Left ventricular hypertrophy		
Yes	1.26 (1.08, 1.48)	Not significant
No	Ref	Ref
Lung disease		
Yes	1.59 (1.42, 1.77)	1.82 (1.56, 2.15)
No	Ref	Ref
Aspirin use	Not significant	Not significant
Blood pressure-lowering medication use		
Yes	1.46 (1.37, 1.56)	1.59 (1.45, 1.74)
No	Ref	Ref
Cholesterol-lowering medication		
Yes	0.71 (0.57, 0.90)	0.52 (0.34, 0.79)
No	Ref	Ref

Figure 2 – Survival in ARIC Participants who Experienced NSTEMI vs. MI-Free Controls (a) and STEMI vs. MI-Free Controls (b) Kaplan-Meier curves demonstrating a difference in survival between NSTEMI (a) or STEMI (b) event (red) and MI-free (blue) participants from the ARIC study. Time is shown in days.

(a)NSTEMI



a



(b)STEMI

b

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Outcomes of Immediate versus Delayed Intervention in patients presenting 24 -72 hours after Myocardial infarction.

CHINEDU ANGELA IGWE[1], KEVIN SILVER[1], OTTORINO COSTANTINI[1], MARC PENN[1], *[1]SUMMA HEALTH CENTER, AKRON, OHIO, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Background: Guidelines state that Percutaneous Coronary Intervention (PCI) is a class I indication for STEMI within 12 hours of symptom onset, and a class IIa recommendation for patients presenting between 12 and 24 hours after STEMI onset. The OAT trial demonstrated that in patients with total occlusion of the infarct-related artery presenting 72 hrs-30 days after MI, PCI compared with conservative therapy did not decrease death, re-infarction, or heart failure during 4 year follow up. There is no clear recommendation on mode of therapy for patients presenting between 24-72 hours after a myocardial infarction.

Objective: We sought to determine whether immediate vs delayed PCI would improve length of stay (LOS) and other outcomes in this patient population.

Methods: We retrospectively reviewed 969 consecutive STEMI patient charts from Jan 2013 - Dec 2015 for the time of symptom onset. Patients who presented < 24 hours, > 72 hours or who did not have clear documentation of time of symptom onset were excluded. Timing of PCI, death, heart failure, and readmission were abstracted.

Results: 45 patients presented 24-72 hours after STEMI onset. The mean age was 65 years (range 41- 83). There were 20 women and 25 men. Of the 45 patients, 43 (96%) were revascularized with PCI. Two patients were treated medically (due to severe triple vessel disease and takotsubo cardiomyopathy). Delayed intervention (>6hrs door to balloon time; mean 24 hours) occurred in 8 (17.7%) patients. Immediate intervention (<6hrs door to balloon time; mean 30mins) occurred in 35 (77.8%) patients

Of the 43 patients, 12 (26.7%) had hospital LOS >3 days. Among the patients with immediate intervention versus delayed intervention, LOS > 3 days was 7 (15%) vs 5 (62.5%) days respectively ($p<0.05$). The composite outcome of in-hospital death, NYHA III-IV heart failure and 30-day readmission occurred in 17 patients (37.8%); 9 patients had $\leq$30 day readmission; 3 died and 4 patients developed NYHAIII-IV. The composite outcome occurred in 13 (37.1%) patients with immediate intervention versus 3 (37.5%) patients with delayed intervention ($p>0.05$).

Conclusions: Immediate revascularization in patients presenting with MI 24-72 hours after symptom onset significantly decreases the LOS. There is no significant difference in the composite of death, NYHA III-IV heart failure and 30 day readmission. A larger study is needed to determine if the reduction in LOS with immediate PCI may result in cost savings while maintaining quality outcomes.

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Higher Trimethylamine-N-Oxide Levels in Diabetic vs. Non-Diabetic Women: A Potential Mechanism for Attenuation of the Female Advantage

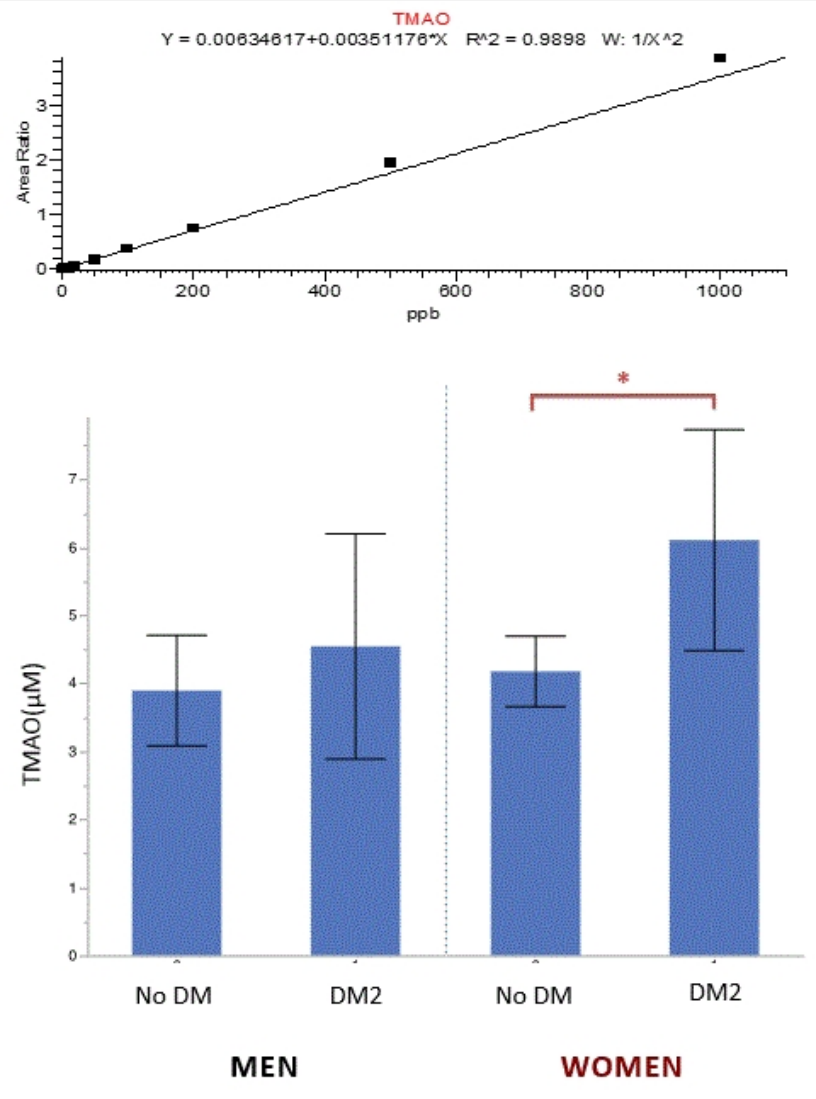
M. Wesley Milks[1], Yu Cao[1], Travis Sharkey-Toppen[1], Suzanne Smart[1], Beth McCarthy[1], Arpad Somogyi[1], Subha Raman[1],
[1]*Ohio State University Wexner Medical Center, Columbus, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

BACKGROUND: Over decades of advances in atherosclerotic cardiovascular disease (ASCVD), women have typically been older in age at incident ASCVD event vs. men; however, diabetes mellitus erases this advantage. Circulating levels of trimethylamine-N-oxide (TMAO), a potent atherogenic metabolite with dietary and genetic influences, have been associated with cardiovascular risk.

OBJECTIVE: We tested the hypothesis that women show higher levels of TMAO in the setting of type 2 diabetes mellitus (DM2).

METHODS: Women without evident ASCVD but with two or more of the following were prospectively identified: total cholesterol ≥240 mg/dL, systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, current smoking, or diabetes mellitus. A control group of men of similar age and risk factor distribution was then enrolled. Plasma TMAO was measured with liquid chromatography-tandem mass spectrometry (LC-MS/MS).



RESULTS: After ensuring the accuracy of the TMAO LC-MS/MS assay across a broad range of standards (R^2=0.9898), we analyzed 101 plasma samples from women aged 49.8 ± 3.8 and 46 men aged 49.5 ± 3.6 years; prevalence of DM2 was similar in both groups (27.7 vs. 28.2%, p=0.95), and distributions of low/intermediate/high 10-year ASCVD risk were similar in men and women (37/52/11% vs. 36/57/7%, p=0.98). Levels of TMAO were significantly higher in women with vs. without diabetes (lnTMAO 1.54 ± 0.68 vs. 1.29 ± 0.52 mg/dL, p=0.02), while this difference was not apparent in the smaller group of men (lnTMAO 1.46 ± 0.54 vs. 1.22 ± 0.67 mg/dL, p=0.23).

CONCLUSIONS: The presence of DM2 is associated with higher levels of TMAO in perimenopausal women at risk of ASCVD. Further investigation into the influence of diabetes on this atherogenic pathway may help identify novel approaches to mitigate ASCVD risk in diabetic women.

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Risks and Outcomes of Direct Current Cardioversion in Pediatric and Adult Congenital Heart Disease

Melissa Morello[1], Gruschen Veldtman[1], Philip R. Khoury[1], Richard J. Czosek[1], [1]*Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
Arrhythmias in adult congenital heart disease (CHD) often signify worsening hemodynamics, resulting in greater morbidity and mortality. Direct current cardioversion (DCCV), though effective, can post significant risk.

Objectives:
1) Describe efficacy of DCCV in patients with and without CHD with arrhythmias treated in a tertiary pediatric cardiac center, and 2) Describe risks for adverse events associated with DCCV.

Methods:
In this single-center retrospective series, patients undergoing DCCV Jan 2010 - May 2015 were categorized as pediatric (<18 years) and adult (>18 years). Electronic medical records were reviewed for demographic, arrhythmic and CHD-specific characteristics; acute efficacy; and arrhythmia recurrence within 3 months of DCCV. Univariate and multivariate analyses were performed to determine risks for complications of DCCV.

Results:
We identified 104 patients with a total of 152 discrete DCCV events, median age 17.4 years (0.15-62.2 years). There were 24 patients (23%) with ≥1 complication, median age 17.7 years (0.15-49 years). Non-life-threatening complications were hypotension and/or bradycardia requiring pacing or medical therapy (12,) worsened/other arrhythmia (16) and myocardial dysfunction (8). Life-threatening complications were CPR (3), ECMO (1) and 30 day all-cause mortality (9). Patient age, duration of arrhythmia, arrhythmic medication, anti-coagulation status and use of echocardiography were not associated with complications. Independent determinants of complications following DCCV were CHD (trend, not statistically significant; $p = 0.07$), moderate – severe systolic dysfunction ($p = 0.004$) and >1 shock administered at the time of DCCV ($p = 0.001$). Complications are associated with death at end of study ($p = 0.0002$). Adults had earlier time to arrhythmia recurrence within 3 months following DCCV ($p = 0.0007$) compared to those < 18 years, but not a greater complication risk.

Conclusions:
DCCV is an effective treatment for arrhythmias. Potentially life-threatening complications may arise. Those with CHD, moderate-severe systolic dysfunction and >1 shock at time of therapy are at greatest risk. Great caution, including back-up hemodynamic support strategies, should be rapidly accessible for those at greatest risk.

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Long Term Mortality Rates of U.S. Veterans with Bundle Branch Blocks Presenting for Coronary Angiography

Amish Patel[1], Jonathan Pollock[2], Edward Sam Roberto[2], Thein Tun Aung[2], Ronald Markert[2], Ajay Agarwal[1], *[1]Dayton VA Medical Center, Dayton, OH, USA, [2]Wright State University School of Medicine, Dayton, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Background:
The relationship between bundle branch blocks (BBB) and poor outcome in heart failure patients is well known. However, the evidence for left bundle branch block (LBBB) or right bundle branch block (RBBB) being a better predictor of mortality is inconsistent. We conducted a retrospective study to answer this question and to determine if BBB is an independent risk factor for mortality in a cohort of U.S. Veterans with atherothrombotic risk factors.

Methods:
Data from a Veterans Affairs (VA) health care facility were retrospectively collected for n=1193 consecutive patients (October 2001 to January 2005) to determine mortality rates in patients with and without BBB who presented for coronary angiography. The primary outcome was all-cause mortality rate and the mean follow up duration was 124±43 months.

Results:
Patients with BBB accounted for 11.4% (136/1193) of the VA cohort. Mean age was 68.9±9.6 years with 95% male. Mean body mass index (BMI) was 29.2±4.9, and mean hemoglobin (Hb) was 13.7±1.6. The prevalence of selected risk factors was: hypertension (HTN) (95%), coronary artery disease (CAD) (94%), hyperlipidemia (77%), diabetes (DM) (55%), left ventricular hypertrophy (LVH) (49%), history of peripheral vascular disease (PVD) (19%), and history of cerebrovascular accident (CVA) (9%). The baseline serum creatinine was 1.9±1.3 mg/dL, with 18% patients having chronic kidney disease (CKD). Mean left ventricular ejection fraction (LVEF) was 40±16, and mean QRS duration was 143.7±20.9. LBBB and RBBB were reported in 54 (39.7%) and 82 (60.3%) of the 136 patients with BBB, respectively. The mortality rate was higher in patients with BBB vs. no BBB (74.3% vs. 45.4%, $p<0.001$) and those with LBBB vs. RBBB (85.2% vs. 67.1%, $p=0.018$). The odds of mortality were higher for those with BBB compared to those without BBB (unadjusted odds ratio [OR] = 3.47 [95% CI=2.32 to 5.20]). The odds of mortality remained higher for the BBB group after adjusting for the 13 significant univariate risk factors - age, DM, LVEF, hyperlipidemia, CKD, CAD, CVA, PVD, QRS duration, HTN, LVH, BMI and Hb. The adjusted OR = 3.15 [95% CI=1.24 to 8.02], $p=0.01$.

Conclusion:
In this study of US Veterans with atherothrombotic risk factors, the all-cause mortality rate was higher with BBB and even greater for LBBB than RBBB. We concluded BBB was an independent risk factor for mortality within this specific cohort.

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Ambulatory Arrhythmia Detection with ZIO® XT Patch in Pediatric Patients

Jeffrey Robinson[1], Jill Shivapour[1], Christopher Snyder[1], [1]*The Congenital Heart Collaborative, Rainbow Babies and Children's Hospital, Case Western Reserve University School of Medicine, Cleveland, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction: Ambulatory electrocardiogram (ECG) monitoring devices are used for 24-72 hours to detect arrhythmias. A novel device, ZIO® XT Patch (ZIO), can be utilized for ECG monitoring for 14 days. The purpose of this study is to describe the duration of ZIO use by age, as well as time to arrhythmia detection in the pediatric population.
Methods: A single-center, retrospective review of patients ≤ 17 yrs of age prescribed a ZIO from 10/14 to 2/16. Demographic and diagnostic data were analyzed, along with duration of ZIO use and time to first arrhythmia. Arrhythmia was defined as supraventricular tachycardia (SVT), advanced atrioventricular block (AV block), or ventricular tachycardia (VT). All ZIO reports were reviewed by a blinded pediatric electrophysiologist. Comparisons were made to 24-hour Holter monitors from the same time period.
Results: A total of 406 ZIO monitors were prescribed for 363 patients; median age 12.7 yrs (0.01-17 yrs), 50% male. The median duration of monitoring significantly increased with age: 1 day (1-14) for 41 ZIO in age < 3 yrs, 2 days (1-13) for 57 ZIO in age 3-6 yrs, 3 days (1-14) for 109 ZIO in age 7-12 yrs, and 4 days (1-14) for 199 ZIO in age 13-17 yrs ($p < 0.001$). A total of 499 Holter monitors were performed; median age 4.9 yrs (0.03-17 yrs), 51% male. Median age differed between the ZIO and Holter groups ($p < 0.0001$), but rates of detection were similar: 11% by ZIO vs 10% by Holter ($p = 0.51$). Arrhythmia was identified on: 45 ZIO (35 SVT, 3 AV block, 7 VT) and 48 Holter (34 SVT, 3 AV block, 11 VT). The first arrhythmia on ZIO occurred in < 24 hrs of monitoring in 42% patients, 24-71 hrs in 24%, and > 72 hrs in 33%, while arrhythmias detected by Holter monitor all occurred within < 24 hrs. A significant number were detected with ZIO after 24 hrs ($p < 0.0001$). Indications for monitoring in patients with first documented arrhythmia at > 72 hrs were palpitations (11) and previous arrhythmia (4).
Conclusions: The ZIO® XT Patch should be considered to detect arrhythmias in pediatric patients of all ages with palpitations or previous arrhythmia. Duration of ZIO monitoring significantly increased with increasing patient age. The majority of arrhythmias occurred after > 24 hours, with a significant number requiring greater than 3 days of continuous monitoring.

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A Unique Case of Myocarditis Causing Acute Isolated Right Ventricular Dysfunction.

Ahmad Abdin[1], Federico Trobo[1], Joseph Pietrolungo[1], Roger Chaffee[1], [1]*SUMMA Health System, Ohio, USA,* [2]*Alpert Medical School of Brown University, Rhode Island, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
Left ventricular (LV) or biventricular dysfunction secondary to acute myocarditis is not an infrequent complication. We present a unique case of myocarditis causing acute isolated right ventricular (RV) dilatation and dysfunction where no other identifiable causes were found.

Case Presentation:
This is a 45-year-old man with past medical history of rectal adenocarcinoma status post abdominoperineal resection with colostomy and urostomy, recent admission for polymicrobial bacteremia that is being treated with intravenous antibiotics, presented to the emergency department (ED) with acute chest pain and shortness of breath that started couple of hours prior to his presentation. His review of systems was otherwise unremarkable. In the ED, he was hypoxic, tachypnic and tachycardiac. The blood pressure and the remainder of the physical examination were normal. Initial labs were remarkable for leucocytosis, positive Troponin and elevated BNP. EKG showed a sinus tachycardia with new T wave inversion in septal leads. Transthoracic Echocardiogram (TTE) exhibited a normal LV function and a severely dilated hypokinetic RV that was a new finding comparing with recent studies. Transoesophageal Echocardiogram (TEE) confirmed the severe RV dysfunction and ruled out endocarditis or any valve pathology. Chest CT angiogram and pulmonary angiography showed no evidence of pulmonary embolism or any parenchymal process. Left and Right heart catheterization showed no coronary artery disease with normal pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output. However, mean RA pressure and RV pressure were 15 mmHg and 37/16 mmHg respectively, consistent with RV dysfunction. In view of these findings, the most likely diagnosis was acute RV dysfunction secondary to acute myocarditis.

Discussion:
The presentation of acute right ventricular dilatation varies between patients based on the severity of ventricular dysfunction and associated comorbidities. Right ventricular dysfunction is usually secondary to acute pulmonary embolism, acute inferior myocardial infarction or acute left ventricular dysfunction. Myocarditis is known to cause left ventricular dysfunction but is not reported to cause isolated right ventricular dysfunction. Most cases of myocarditis are usually triggered by viruses and augmented by autoimmunity. The myocyte damage is believed to be mediated both by direct invasion of the myocardium and by immune insult.

Conclusion:
Our case proves that myocarditis can cause isolated right ventricular dilatation and dysfunction. Currently, we are not aware about any similar case of right ventricular dysfunction that is reported in the adult medical literature.

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"The advantages and limitations of echocardiography in a unique presentation of double-sided endocarditis from intravenous drug use"

Ben Alencherry[1], Claire Sullivan[2], Brian Hoit[2], [1]*Department of Medicine, University Hospitals Case Medical Center, Cleveland, Ohio, USA,* [2]*Harrington Heart and Vascular Institue, University Hospitals Case Medical Center, Cleveland, Ohio, USA,* [3]*Case Western Reserve University, Cleveland, Ohio, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

A 25-year-old female intravenous drug user presented with a three-week history of fever and myalgias and was found to have infective endocarditis affecting the mitral and tricuspid valves. Blood cultures grew methicillin sensitive *Staphylococcus aureus*. Computed tomography showed emboli in multiple organs. Transthoracic echocardiogram revealed large vegetations on the mitral valve with moderate valvular regurgitation and a transesophageal echocardiogram identified a posterior mitral leaflet perforation. The patient underwent urgent surgical intervention after developing respiratory distress from worsening congestive heart failure. Intraoperative findings in addition to those found on echocardiography included a large abscess in the posterior wall of the left atrium. The patient underwent mitral and tricuspid valve replacement due to valve damage. This case shows a unique presentation of valvular involvement of infective endocarditis and the capabilities and limitations of echocardiography in assessing the extent of disease in this condition.

Key Words: Endocarditis, Valve Perforation, Echocardiography, Mural Abscess, Intravenous Drug Use

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Case Report of Junctional Tachycardia with Exit Block in an Adolescent

Nicholas Cundiff[1], Jeffrey Robinson, MD[2], Bryan Cannon, MD[3], Christopher Snyder, MD[3], *[1]Centers for Osteopathic Research and Education, Heritage College of Osteopathic Medicine, Athens, Ohio, USA, [2]The Congenital Heart Collaborative, Rainbow Babies and Children's Hospital, Case Western Reserve University School of Medicine, Cleveland, Ohio, USA, [3]Mayo Eugenio Litta Children's Hospital, Rochester, Minnesota, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
This is a case report of an otherwise healthy adolescent female who presented with symptomatic vasovagal syncope. During her workup an ambulatory ECG monitor revealed junctional tachycardia with exit block. She was treated with flecainide with resolution of the arrhythmia.

Case Presentation:
A 17-year old, healthy Caucasian female presented with a 2-year history of exercise-related palpitations, shortness of breath and chest tightness that lasted up to 45 minutes and resolved with rest. Ambulatory ECG monitoring demonstrated sinus rhythm correlating with complaints of her palpitations and asymptomatic episodes of Mobitz type I and advanced, second-degree atrioventricular (AV) block (Figure 1).

The patient was admitted for evaluation and management, which revealed normal imaging and laboratory studies. 30-minute head-up tilt test resulted in symptoms consistent with the chief complaint while maintaining normal sinus rhythm and 1:1 AV conduction throughout. Based on the intermittent character of the advanced, second-degree AV block and her symptoms during tilt test brought out during sinus rhythm, the diagnosis of junctional tachycardia with exit block with concurrent vasovagal syncope was entertained. To prove this, the patient was started on flecainide at 130mg/m^2/day by mouth divided twice daily. Her telemetry and ECG monitoring demonstrated normal rhythm without episodes of junctional tachycardia with exit block. The patient was discharged home with a repeat Holter monitor without episodes.

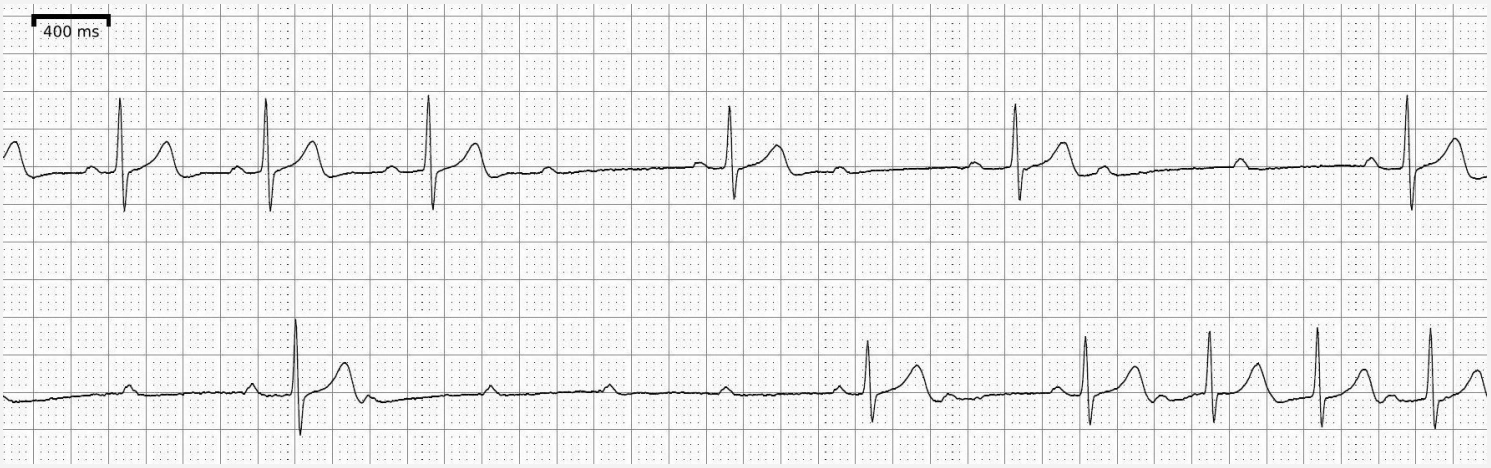

Figure 1: This ambulatory ECG monitor is showing junctional tachycardia with exit block. The rhythm has atrial P-waves at equal intervals and blocked QRS complexes.

Discussion:
Junctional tachycardia with exit block in the pediatric population most commonly presents in the postoperative setting. Non-postoperative junctional tachycardia with exit block has not been previously described in a pediatric patient. Historically, this arrhythmia has been documented in the elderly population, commonly associated with digoxin toxicity or myocardial infarction. Some case reports describe successful management with propranolol or flecainide.

The case presented herein includes both junctional tachycardia with exit block and vasovagal syncope. The result of the head-up tilt test helped rule-out AV block secondary to increased vagal tone and was consistent with the diagnosis of vasovagal syncope. The intermittent character of the asymptomatic arrhythmia is incongruent with advanced, second-degree AV block. Additionally, flecainide is contraindicated in AV block and the patient's response to this therapy further suggests the diagnosis of junctional tachycardia with exit block.

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Curious case of patient prosthesis mismatch presenting with endocarditis

Xu Gao[1], Avirup Guha[1], Subha Raman[1], David Orsinelli[1], [1]*The Ohio State Wexnar Medical Center, Columbus, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:

Prosthesis-patient mismatch (PPM) is defined on the basis of small valve size indexed to body surface area, excessive gradients across the prosthesis and by other measurements and their combination. We present a peculiar case of bio-prosthetic aortic valve (BAVR) with PPM as well as infective endocarditis of the prosthesis.

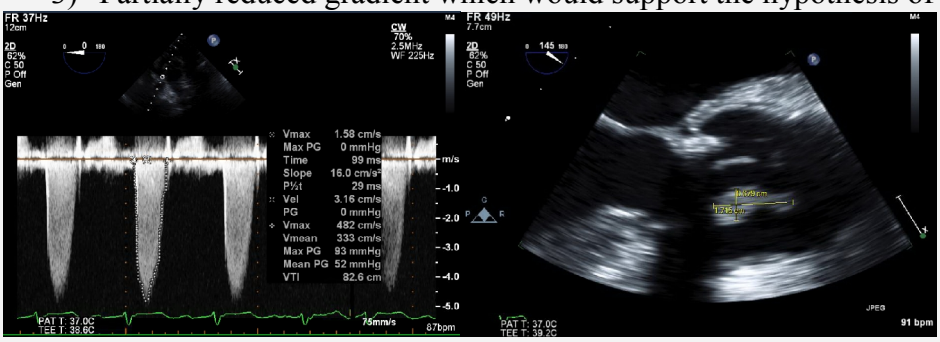
Case:

Fifty-two year old female presented to the hospital with pressure and sharp chest pain with a fever of 100.3. Medical history was significant for congenital bicuspid aortic valve stenosis with BAVR in 2013 (initial mean gradient of 14 mm Hg and Doppler velocity index (DVI) of 0.5) with a 21inch prosthesis, hypertension and had underwent colonoscopy a week ago. Transthoracic echocardiogram from one year prior to this presentation showed elevated prosthetic mean gradient of 36 mmHg and DVI of 0.3. Transesophageal echocardiogram demonstrated a large mass on one of the prosthesis leaflet with normal leaflet opening. The peak instantaneous gradient of the aortic valve was 92 mmHg with the mean AV gradient of 52 mmHg, DVI of 0.25 and no aortic regurgitation. Blood cultures were positive for methicillin resistant Staphylococcus epidermidis. The patient's hospital course was complicated by high degree atrioventricular block and she underwent a valve replacement with 21 inch prosthesis. She received appropriate antibiotic and pacemaker therapy.

Discussion:

Although most cases of aortic valve endocarditis present with acute aortic valve regurgitation, this was not seen in our case. Instead, we noted worsening stenosis of the aortic valve when comparing echo results to those of the previous year (mean gradient of 14 mm Hg -> 36 mmHg -> 52 mmHg). Doppler velocity index (DVI) or dimensionless index is the ratio of velocity proximal to the valve, to the velocity through the valve. Acceleration time (AT) is the time from the onset of flow to maximal velocity through the valve which is another way of objectively studying a BAVR. The American Society of Echocardiography algorithm for evaluating BAVR states that DVI of 0.25-0.29 and AT of <100 ms with conical shape of the envelope likely represents valve PPM or high flow instead of a stenotic valve. Our patient had DVI of 0.25 and AT of 99 ms which was most consistent with PPM as there the left ventricle was not hyperdynamic which rules out high flow state. As the envelope was not typically conical and the AT was of borderline value of 99 ms, there is a likelihood that there was some additional aortic stenosis which developed either due to natural deterioration of BAVR or from the vegetation. The patient underwent aortic valve replacement with the same 21 mm valve due to lack of fit of a bigger valve and we are awaiting evaluation with transthoracic echo which would prove one of the 3 theories:

1) Gradient of ~ 14 mm Hg which would mean that this was simple case of aortic stenosis of BAVR with subsequent endocarditis with no PPM
2) Mean gradient exactly as there was pre-op which would prove that this is 100% PPM.
3) Partially reduced gradient which would support the hypothesis of combination of PPM and BAVR stenosis.



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Sometimes an ACS is not just an ACS A case report

Vittal Hejjaji[1], Jun Li[1], Sahil A Parikh[1], [1]*Case Western Reserve University, Cleveland, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background/Objective

Coronary artery vasospasm occurs frequently with a prevalence of up to 4% among all cases of chest pain [1]. Prinzmetal or variant angina presents as resting chest pain with ST segment elevation attributed to spontaneous coronary artery vasospasm [2]. Vasospasm frequently co-exists with non-obstructive coronary atherosclerosis and may be provoked by environmental or pharmacologic stimuli. While frequently benign, here we are describing a patient with documented plaque rupture as well as refractory and debilitating vasospasm despite optimal medical therapy.

Case

A 41-year-old woman with hypertension and chronic marijuana abuse presented to our hospital in March of 2015 with angina. Her heart rate was 59 and blood pressure was 179/98 mmHg. Physical exam was unremarkable. Her troponin was elevated to 1.63 ng/mL and her electrocardiogram (ECG) showed ST segment elevation in leads V1 and V2. After administration of nitroglycerin, her symptoms improved. She emergently underwent coronary angiography which demonstrated a hazy 40% lesion in the proximal left anterior descending (LAD) artery with TIMI 3 flow. The remaining vessels were free of angiographically visible atherosclerosis. Optical coherence tomography (OCT) of the LAD identified plaque rupture within a fibrous plaque without significant thrombus. Given the resolution of chest pain, normal coronary perfusion and resolved ST changes, the patient was treated medically with a low threshold for repeat angiography and intervention. The patient was maintained on aspirin and ticagrelor, and the intravenous (IV) nitroglycerin which had been started in the Emergency Department (ED) was weaned. Recurrent angina with dynamic ECG changes prompted repeat angiography and stenting of the proximal LAD with a drug eluting stent(DES). The patient was discharged home on aspirin, ticagrelor, atorvastatin, and a long acting nitrate. However, in the ensuing 6 months, she presented to the ED twice for similar anginal symptoms. On the first admission, repeat angiography showed a widely patent stent with severe vasospasm at the distal stent edge relieved by intracoronary nitroglycerin. Her second episode was complicated by ventricular fibrillation and cardiac arrest, from which she was resuscitated. Angiography was unremarkable with a patent stent. She underwent placement of an implantable cardioverter defibrillator for secondary prevention [3] and was discharged on isosorbide mononitrate and amlodipine. However, due to hypotension, she was unable to tolerate maximal doses of either agent.
The patient continues to have episodic vasospasm despite abstinence from smoking (tobacco and marijuana), good medical compliance and participation in cardiac rehabilitation. She returned to the ED 8 months after her cardiac arrest with severe chest pain and ST elevations in leads I and aVL. Repeat coronary angiography again showed a widely patent stent with diffuse vasospasm in the LAD relieved with intracoronary nitroglycerin. Her amlodipine was switched to nifedipine.

Discussion/conclusion

Vasospastic angina is more common in females under the age of 50 [4]. Luminal irregularities, intimal erosion, fibrous plaque formation and rupture have been documented at vasospasm sites [5]. Observational data suggest that plaque rupture with thrombus formation is frequently co-existent with vasospasm. [1] Histological evaluation of vasospastic sites has shown the presence of neo-intimal hyperplasia which developed from recurrent spasms and contribute to the development and progression of a plaque [6]. Moreover, diffuse intimal hyperplasia is common in patients who suffer from vasospasm [7] [8]. Calcium channel blockers are the first line of treatment while nitrates are the second line [9] [10] but better outcome is derived from combined therapy. [11]

In conclusion, this a unique case where vasospastic angina was masked by intravenous nitroglycerin use for what was believed to be a *de novo* acute coronary syndrome (ACS) with an atherosclerotic plaque rupture. It is well-described that occult atherosclerotic lesions can be present at vasospastic foci which may be angiographically normal. In this case, a plaque rupture diverted us from the initial diagnosis of vasospasm, which has proven difficult to control for this patient.

1. Ergonovine maleate testing during cardiac catheterization: a 10-year perspective in 3,447 patients without significant coronary artery disease or Prinzmetal's variant angina.*J Am Coll Cardiol*. 1992 Jul. 20(1):107-11.
2. Prinzmetal M Kennamer R MERLISS R, et al. **Angina pectoris. A variant form of angina pectoris; preliminary report** Am J Med 1959; 27:375
3. **Clinical Implications of an Implantable Cardioverter-Defibrillator in Patients With Vasospastic Angina and Lethal Ventricular Arrhythmia**
Yuya Matsue, MD,* Makoto Suzuki, MD, PHD,* Mitsuhiro Nishizaki, MD, PHD,† Rintaro Hojo, MD,‡ Yuji Hashimoto, MD, PHD,* Harumizu Sakurada, MD, PHD‡
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5. **OCT-Defined Morphological Characteristics of Coronary Artery Spasm Sites in Vasospastic Angina.**
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6. **Histological evaluation of coronary plaque in patients with variant angina: relationship between vasospasm and neointimal hyperplasia in primary coronary lesions.**
Suzuki H[1], Kawai S, Aizawa T, Kato K, Sunayama S, Okada R, Yamaguchi H.
7. **Coronary plaque component in patients with vasospastic angina: a virtual histology intravascular ultrasound study.**
Tsujita K[1], Sakamoto K, Kojima S, Kojima S, Takaoka N, Nagayoshi Y, Sakamoto T, Tayama S, Kaikita K, Hokimoto S, Sumida H, Sugiyama S,Nakamura
8. Miyao Y, Kugiyama K, Kawano H, et al. Diffuse intimal thickening of coronary arteries in patients with coronary spastic angina. J Am Coll Cardiol 2000;36:432
9. **Pharmacotherapy of Vasospastic Angina**
Justin R. Harris, PharmD, BCPS1; Genevieve M. Hale, PharmD, BCPS2; Tarun W. Dasari, MD, MPH, FACC3; Nicholas C. Schwier, PharmD4
10. **Prognostic Effects of Calcium Channel Blockers in Patients With Vasospastic Angina – A Meta-Analysis –**
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Conti CR, Hill JA, Feldman RL, Conti JB, Pepine CJ.

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Septic Pulmonary Emboli from Mitral Valve Endocarditis in a Patient with Repaired Tetralogy of Fallot

Aniket Rali[1], Arun Iyer[1], Claire Sullivan[2], James Strainic[3], Brian Hoit[2], *[1]Department of Medicine, University Hospitals Case Medical Center, Case Western Reserve University, Cleveland, OH, USA, [2]Harrington Heart and Vascular Institute, University Hospitals Case Medical Center, Case Western Reserve University, Cleveland, OH, USA, [3]Rainbow Babies and Children's Hospital, Case Western Reserve University, Cleveland, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
The annual incidence of infective endocarditis (IE) is estimated to be about 3 to 9 cases per 100,000 persons in industrialized countries. Individuals at greatest risk are those with prosthetic valves, intracardiac devices, unrepaired cyanotic congenital heart diseases or a prior history of IE. Other risk factors include chronic rheumatic heart disease (< 10% of cases in industrialized countries), hemodialysis, age-related degenerative valvular lesions and other co-morbidities such as HIV infection, diabetes and intravenous drug use. Infectious endocarditis in native as well as repaired ventricular septal defects (VSD) is a known and well documented phenomenon.

This case report will discuss a young woman with repaired tetralogy of Fallot who presented with septic emboli to the lungs resulting from mitral valve endocarditis. Patient's post-surgical cardiac anatomy, extent of septic emboli to various organs and mechanism of pulmonary emboli from left sided endocarditis make this case unique.

Case Presentation:
A 37 year old woman with a past medical history significant for congenital deafness and surgically repaired Tetralogy of Fallot presented with three day history of nausea, vomiting, fever, chills, dyspnea, and lower extremity weakness and physical examination notable for Janeway lesions. Peripheral blood and urine cultures were positive for methicillin sensitive *Staphlococcus aureus.* Transesophageal echocardiogram was consistent with mitral valve endocarditis. Computed tomography images of the chest, abdomen and pelvis demonstrated septic emboli to multiple organs including lungs, liver, spleen and kidneys. Saline contrast study was negative for a patent foramen ovale, or residual ventricular septal defect. Thus, effectively ruling out left to right intracardiac shunt as the cause of pulmonary septic emboli from mitral valve endocarditis. Moreover, cardiac MRI did not show any evidence of right sided endocarditis. Therefore, we believe the source of septic pulmonary emboli from mitral valve endocarditis to be through the bronchial arteries.

Discussion / Conclusion:
The patient presented with signs and symptoms of severe sepsis and was found to have infective endocarditis. Several aspects of this case make it unique namely the patient's post-surgical cardiac anatomy, potential sources of initial infection with MSSA, extent of septic emboli to various organs and most importantly pulmonary emboli from mitral valve endocarditis. Our patient had a dental procedure performed two weeks prior to her initial presentation that may have been the source of bacteremia. Although she took prophylactic antibiotics, MSSA would be an atypical organism.

Pulmonary valve regurgitation is a known complication of prior tetralogy of Fallot repair Our patient had her native pulmonary valve excised during her surgical repair with residual severe pulmonary regurgitation. Such valve insufficiency can also occur in the setting of endocarditis. In our patient with obvious evidence of septic pulmonary emboli, the concern for right-sided endocarditis is high but was unable to be visualized TEE or cardiac MRI. Further, no left to right shunt such as residual VSD or PFO were visualized.

Lungs receive dual blood supply from pulmonary as well as bronchial circulation. While this makes lung tissue more resistant to infarction, it also puts it at risk for emboli from both right and left sides of the heart. In our patient with known mitral valve endocarditis and extensive showering of emboli to liver, spleen, kidneys, brain and shoulder joint, we believe the mechanism of septic pulmonary emboli to be through the bronchial arteries. To the best of our knowledge, this is the first reported case of septic pulmonary emboli from left sided endocarditis in a patient with surgically repaired TOF. Patient did not require surgical repair of her mitral valve as no mitral valve vegetation was noted on TEE performed after seven weeks of intravenous antibiotics.

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A RARE CASE OF COCAINE INDUCED LEFT ATRIAL THROMBUS LEADING TO EMBOLIC STROKE

Sravani Kamatam[1], Raktim Ghosh[1], Ramyashree Tummala[1], Kevyan Ravakhah[1], Anjan Gupta[1], [1]*St. Vincent Charity Medical Centre, Cleveland, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

BACKGROUND: Cocaine use is a major public health problem contributing to over 500,000 emergency room (ER) visits per year in US population. Cocaine stimulates sympathetic nervous system by preventing the reuptake of noradrenaline, serotonin and dopamine at presynaptic nerve terminals causing vasoconstriction. Cocaine-induced stroke pathophysiology includes cerebral vasospasm, vasculitis, enhanced platelet aggregation, cardioembolism, and hypertensive emergencies leading to intracranial hemorrhage. Of all the possible mechanisms, cardioembolism is a rare cause of cocaine-induced stroke particularly in a patient who does not have atrial fibrillation. We are presenting a rare case of cocaine-induced LA thrombus, where the patient had normal sinus rhythm and subsequently developed stroke from an embolism.

CASE PRESENTATION: A 60-year-old male with no past medical history was admitted for chest pain to rule out acute coronary event. The patient admitted to consumption of marijuana and cocaine for few years with the most recent use of cocaine just before the onset of his current symptoms. On admission, chest x-ray, Electrocardiogram (EKG) and troponin were normal. Three hours on admission, he developed sudden onset of slurred speech, left sided weakness and numbness, and the telemetry showed new onset atrial flutter. The patient was never diagnosed with atrial fibrillation or any abnormal cardiac rhythm. The primary care office visits in last one year always recorded sinus rhythm. On examination his left arm and left leg had grade 2 power and decreased sensation on the left side of the body. There was an extensor response in the left toe, with increased tone and brisk reflexes on the left side of the body. NIH stroke scale was 6. Tissue plasminogen activator (t-PA) was administered and stroke workup was pursued. His CBC and CMP were normal, EKG showed new onset atrial fibrillation. CT scan of the brain did not show any intracranial hemorrhage. Brain MRI/MRA showed subacute infarcts in the territory of the bilateral middle cerebral arteries. Carotid ultrasound and hypercoagulability workup Hypercoagulability workup including platelet count, prothrombin time, partial thromboplastin time, blood cultures, lupus anticoagulant, free protein S activity, antithrombin III activity, plasminogen activity, protein C activity, and lupus antigen workup were negative. Transesophageal echo (TEE) revealed a large LA thrombus. This patient had multiple emboli believed to be cardioembolism secondary to LA thrombus induced by cocaine abuse. He was started on aspirin and statin 24 hrs. after the initial tPA administration.

DISCUSSION: Previous studies have documented cocaine induced platelet aggregation and thrombosis in various arteries. However, there is paucity of published literature on occurrence of LA thrombus, induced by cocaine presenting as embolic stroke. Cocaine contributes to ischemic events by promoting platelet aggregation on endovascular surfaces, in addition to its sympathomimetic effects. Platelets get activated after cocaine administration and they release constituents of their alpha granules, promoting thrombosis. Another presumed mechanism of cocaine induced thrombosis involves, endothelial injury occurring with vasospasm and subsequent platelet aggregation. In vitro studies showed that cocaine caused increased response of platelets to arachidonic acid and increased thromboxane production and platelet aggregation. Along with vasoconstriction, platelet activation, and endothelial dysfunction, impaired fibrinolysis is also a recognized mechanism of cocaine-induced thrombosis. Some of the possible mechanisms of cocaine induced left atrial thrombus formation include low plasma tissue plasminogen activator activity, factor XII deficiency, high tissue plasminogen activator inhibitor activity, leading to hypercoagulability. Deficiency of protein C and S were found to be normalized after withdrawal of cocaine, generating another possible explanation of hypercoagulable state, making them prone for thrombus formation. Our patient never had any baseline abnormal heart rhythm, and no valvular abnormality. The multiple emboli which were cardioembolism secondary to LA thrombus were believed to be induced by cocaine abuse. In patients with cocaine-induced ischemic stroke, a cardiac source of embolus should be investigated.

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Hole in the Heart: Ventricular Septal Defects Due to Delayed Presentations for Myocardial Infarction – A Case Series

Amber Makani[1], John Hornick[1], Jun Li[1], Claire Sullivan[1], Richard Josephson[1], [1]*Case Western Reserve University Hospitals, Cleveland, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction/Objective:
Acquired ventricular septal defect (VSD) is a complication of approximately 1-2% of acute myocardial infarctions (AMI) in the prethrombolytic era and <1% since the initiation of thrombolysis.[1] However, while the incidence is decreasing, it remains associated with high morbidity and mortality as 60-70% patients die within 2 weeks if treated with medical management alone.[2] Current guidelines recommend emergency surgical repair to prevent expansion of the rupture site and hemodynamic collapse; however, in hemodynamically unstable patients, percutaneous closure has been trialed as a temporizing measure. We present several cases of VSD complicating AMI and a review of current recommendations regarding surgical and percutaneous closure.

Case presentation:
In this study, we evaluated six patients who developed ventricular septal defect in the setting of acute myocardial infarction, prior to transfer or while at our tertiary care facility. Using this data, we were better able to understand the presentation of VSD as well as the epidemiology of this complication. In our case series, we found that VSD was complication of delayed presentation in patient's with risk factors such as coronary artery disease, smoking, and diabetes. Of the six patients we identified with this complication, there was an equal proportion of males to females, and 5/6 patients were noted to be older than 65 years of age. VSD was diagnosed by ventriculogram or transthoracic echocardiogram in our patients. Unfortunately, several of our patients suffered hemodynamic instability, precluding surgery as an immediate treatment option. However, delayed surgical intervention was successfully performed on 1 of our 6 patients, who is doing well to date. Of the remaining 5 patients, 3 underwent urgent surgical closure but only 1 has survived to date.

Discussion/Conclusion:
Acquired VSD in the setting of AMI was once associated with 100% mortality when management was limited to medical therapy alone.[3] Since the advent of thrombolytic therapy and percutaneous coronary intervention, the incidence of VSD has dramatically decreased. However, patients with delayed STEMI presentations with acquired VSD continue to pose a complex challenge for immediate hemodynamic stabilization and long term survival.
Urgent surgical repair remains a class I indication according to the American Heart Association/American College of Cardiology practice guidelines.[4] Nonetheless, studies have shown that early surgery (between 3 days to 4 weeks after an AMI) carries an overall in-hospital mortality of 52.4%[5], while intervention delayed for 2 weeks has been associated with decreased mortality[6]. This is likely due to a significant selection bias of patients who have survived the prolonged waiting period, as well as improved surgical success by allowing a complete infarct to occur with decreased myocardial friability. In adition, surgical intervention has been associated with residual shunting in 10-37% of patients due to development of new VSD or unnoted additional defect, with approximately 11% of patients requiring additional surgeries.[7] Transcatheter closure was initially introduced for closure of residual leak after surgical repair as well as those deemed too high risk for surgery. Subsequently transcatheter device closure has emerged as an acceptable alternative to primary closure of post-AMI VSD in selective patients. In a 51 patient study using Amplatzer device closure for VSD <15mm and surgical intervention for VSDs >15mm at a fixed time interval in patients who were otherwise randomized, there was no difference in overall mortality.[8] In a select patient population with small defects, sub-acute, or in those who are too unstable to withstand surgical intervention, transcatheter closure and repair may have a role as standard therapy for post-MI VSD therapy.

References:

[1] Serpytis P, Karvelyte N, et al. Post-Infarction Ventricular Septal Defect: Risk Factors and Early Outcomes. *Hellenic J Cardiol.* 2015; 56: 66-71
[2] Bouchart F, Bessou JP, Tabley A, Redonnet M, Mouton- Schleifer D, Haas-Hubscher C, Soyer R. Urgent surgical re- pair of postinfarction ventricular septal rupture: early and late outcome. *J Card Surg* 1998;13:104-12.
[3] Gaudiani VA, Miller DG, et al. Post-infarction ventricular septal defect: an argument for early operation. *Surgery*. 1981; 89:48-55
[4] ACCF/AHA Guideline:2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013;127:e362-e425
[5] Papalexopoulou N, Young CP, et al. What is the best timing of surgery in patients with post-infarct ventricular septal rupture? *Interact Cardiovasc Thorac Surg* 2013; 16:193-6.
[6] Deville C, Fontan F, Chevalier JM, Madonna F, Ebner A, Besse P. Surgery of post-infarction ventricular septal defect: Risk factors for hospital death and long-term results. *Eur J cardiothorac Surg.* 1991;5: 167-174
[7] Deja MA, Szostek J, Widenka K, et al. Post infarction ventricular septal defect - can we do bet- ter? *Eur J Cardiothorac Surg*. 2000;18:194-201.
[8] Maltais S, Ibrahim R, Basmadjian AJ, Carrier M, Bouchard D, Cartier R, Demers P, Ladouceur M, Pellerin M, Perrault LP. Postinfarction ventric- ular septal defects: towards a new treatment algorithm? Ann Thorac Surg 2009;87:687–692

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Title: (Do not enter author details)

Variant angina with polymorphic ventricular tachycardia: Is an ICD indicated?

Toral Patel[1], Sadeer AlKindi[1], Prashanth Thakker[1], [1]*Case Medical Center, Cleveland, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Objectives:

1. Identify a patient population highly susceptible to ventricular tachycardia/fibrillation.
2. Discuss guidelines for ICD placement in hemodynamically stable VT in variant angina.

Case Presentation:

57 year-old male with history of hypertension and tobacco use, presented to the emergency department after a 10 day history of chest discomfort, lightheadedness, and presyncopal episodes. He was given aspirin and nitroglycerin sublingual, which alleviated the pain. Labs were insignificant with three negative troponin I levels (0.01, then 0.03, then 0.02 ng/ml), electrolytes within normal range, and a lipid panel: cholesterol 152, HDL 32, LDL 104, triglycerides 80. Urine drug screen was negative for cocaine metabolites. Thyroid stimulating hormone was 3.96.

The electrocardiogram showed ST segment elevations in leads II, III, and aVF (figure 1) with short runs of non-sustained polymorphic ventricular tachycardia (VT) on telemetry. With concern for NSTEMI and VT, he underwent coronary angiography that showed normal left main coronary artery, 70% stenosis in left anterior descending artery, and 70 % stenosis in left circumflex artery (figure 2), and a dominant right coronary artery with spasm that improved with 1 mg of nitroglycerin (figure 3). During the procedure, patient was having chest pain and electrocardiogram monitoring showed non-sustained ventricular tachycardia that resolved with 1 mg intracoronary nitroglycerin. During these episodes and the procedure, patient continues to be hemodynamically stable. He was transferred to the cardiac intensive care unit and was started on nifedipine 60mg PO daily and isosorbide mononitrate 60 mg PO daily. Patient was discharged home with an event monitor; however, he was lost to follow-up.

Discussion:

Prinzmetal "variant" angina is a spectrum of symptoms that is caused by coronary artery vasospasm and is precipitated by smoking, cocaine, or medications. [1] Patients with variant angina are highly susceptible to malignant arrhythmias including ventricular tachycardia and ventricular fibrillation and subsequently sudden cardiac death (SCD). Currently, the management of patients with variant angina and ischemic findings on EKG or coronary angiography include medical optimization with calcium channel blockers and sublingual nitrates [2]. However, limited data has been presented to address management of these patients with VT/VF in symptom-free periods.

Prospective studies have shown that patients with variant angina who have suffered an SCD have benefits from an ICD for secondary prevention, but the role of an ICD for primary prevention is unclear [3]. The ACC/AHA guidelines for ICD placement include those who are survivors of ventricular fibrillation, and those who have hemodynamically significant sustained VT or VF induced at an electrophysiological study [4, 5]. No specific recommendations are made for non-ischemia-induced hemodynamically stable VT, inducible VT in patients without structural heart disease or specifically with vasospastic angina. Also, no reports have been done to estimate the annual risk of SCD. In order to address this gap, Takagi et al, developed a comprehensive risk score for risk stratification of patients presenting with vasospastic angina. This score emphasizes on multivessel involvement, out-of-hospital cardiac arrest, smoking, angina at rest, ST segment elevation and beta blocker use; however, it is still difficult to distinguish patients who may respond to medical treatment from those at high risk of SCD despite medical optimization [6].

Recently, a pt with variant angina patient with recurrent episodes of VT during coronary vasospasm-induced myocardial ischemia underwent an EPS study with reproducible inducible sustained episodes of VT. The patient was asymptomatic and medically optimized during these episodes. ICD for primary SCD prevention was implanted based on the results of EPS [7]. Given this patient's presentation, it is impossible to predict with accuracy if he would continue to have asymptomatic VT in the future while on optimal medication; and if he did, whether it will result in fatal VF. An EPS study had not been conducted and it was decided that the patient would not need an ICD placement. There is a gap in the literature regarding management of patients with Prinzmetal angina and risk stratification for the indication of an ICD; it is important that risk stratification for primary prevention in high risk populations, such as variant angina, be addressed in future studies

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One of These Things Is Not Like the Other: Case Comparison of Thrombus in Transit Across Patent Foramen Ovale and Normal Variant of Inter-Atrial Septum

Jonathan Pollock[1], Thein Tun Aung[1], Patrick Chen[1], Bal Srivastava[1], [1]*Wright State University, Dayton, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction/objective: Patent foramen ovale has become a more recognized cause of cryptogenic stroke and peripheral embolism. Thrombus within the patent foramen ovale is a rare finding. These are commonly discovered from echocardiography when undergoing investigation for the etiology of systemic embolism. Evaluation for cardiac source of embolism is the most common reason of ordering transesophageal echocardiogram (TEE). We would like to present two interesting patients with abnormal TEE.

Case Presentation(s): The first case is an 82 year old man with a prior history of ischemic cardiomyopathy and recent unprovoked deep venous thrombosis who presented with an acute left-sided, occipital cerebral infarct. His transthoracic and TEE were abnormal with a very large, mobile, serpiginous intracardiac thrombus. It was originating in right atrium, crossing the tricuspid valve into the right ventricle; also crossing a patent foramen ovale, into the left atrium and left ventricle. Left heart catheterization showed 50% in-stent restenosis in left anterior descending artery and 99% in-stent restenosis in right coronary artery. Ejection fraction was visually estimated to be 50% with moderate aortic regurgitation. Cardiothoracic surgery was consulted and the patient underwent mechanical thrombectomy through an open thoracotomy, as well as patent foramen ovale closure, coronary artery bypass grafting with saphenous vein graft to right coronary artery, and aortic valve replacement with Edwards 25 mm bioprosthetic valve. Transthoracic echocardiogram was done one month after the surgery, showing low-normal left ventricular systolic function and well-seated bioprosthetic aortic valve.

The second case is an 49 year old African American woman with previously undiagnosed hypertension who presented with subacute right middle cerebral artery infarction. She had no evidence of deep venous thrombosis or pulmonary embolism clinically or on imaging studies. However, TEE showed suspicious mass inside the inter-atrial septum. It was a discrete, non-mobile lesion; similar in echodensity to its' surrounding cardiac tissue. There was no inter-atrial shunt across the septum as displayed by negative saline contrast study. Computerized tomography of the chest with contrast showed a polypoid filling defect at the inter-atrial septum. There was no flow of contrast crossing the inter-atrial septum.

Discussion: Due to the rarity of thrombus straddling the patent foramen ovale (TSFO), the first line treatment remains debatable. Treatment is either surgical with mechanical thrombectomy or medical with thrombolysis and/or anticoagulation. Fauveaua et al. reviewed all 88 cases of TSFO between 1985 through 2007. They described risk of paradoxical embolism in patients with TSFO to be 44%; 30% cerebral and 14% to an extremity. Moreover, they found that only a slight difference in the mortality rates after heparin when compared to surgical treatments. Patients treated with thrombolysis had the highest rate of mortality, due to greater hemodynamic compromise in the patient. Nonetheless, investigators believed that mortality was more closely linked to severity of clinic presentation of the patient rather than morbidity attributed to chosen treatment strategies. Risk for systemic embolism decreased with anticoagulation treatment. Additionally, they found that anticoagulation was an acceptable therapeutic alternative to surgery particularly in patients with comorbidities (increased age, stroke, cancer) who were high risk surgical candidates.

These two cases exhibit the difficulty in truly diagnosing this condition. The decision making in the second case presented was more challenging than the former case which was treated with surgery. Considering the patient had recent stroke and had the above-mentioned imaging findings, it was difficult not to attribute the event to a thrombus in transit. However, considering the mass was discrete, having the same echo density as cardiac tissue, and no intracardiac shunt across the inter-atrial septum, we decided to monitor further without surgical intervention. Shortly after discharge, she was diagnosed with atrial fibrillation which could have been a potential etiology for her stroke. She was placed on anticoagulation and monitored with close follow-up.

Conclusion: Normal variants of inter-atrial septum can be confusing and can appear as pathological for inexperienced observers. It is very important to recognize the normal variants because differentiating these findings can dramatically impact clinical decision making.

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Title: (Do not enter author details)

Coronary Artery Fistula Presenting As Myocardial Infarction: Case Study and Review of Operative Management with Percutaneous versus Surgical Intervention

E.S. Roberto[1], Thein Aung[1], Brian Cothern[1], Anwural Kabir[2], *[1]Wright State University Boonshoft School of Medicine, Dayton, OH, USA, [2]Good Samaritan Hospital, Dayton, OH, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
Coronary artery fistula (CAF) is a direct connection between one or more coronary arteries and another vessel or cardiac chamber. Exact incidence of this rare condition is not known but estimated at 0.002%; clinically silent CAFs are incidentally found on 0.3-0.8% of cardiac catheterizations. Although often congenital in etiology, acquired cases have been associated with infection, neoplasm, trauma, or iatrogenic as post-surgical and post-interventional sequelae. Pathophysiologic changes and symptoms are often silent before the age of 20, but later manifest as angina, arrhythmia, endocarditis, myocardial infarction, heart failure, and spontaneous rupture. To date, management of CAFs has been complicated and not well defined. Approaches include percutaneous transcatheter devices or surgical closure with possible bypass and ventriculotomy.

Case presentation:
A 25 year-old male with history of anxiety was admitted due to chest pain. Non-exertional chest pain started one day ago, 10/10 in intensity and radiating to his back. It occurred intermittently with no apparent exacerbating and relieving factor. He had 2D echocardiogram done as outpatient last month due to intermittent dizziness for several months. Echocardiogram showed normal ejection fraction, normal chamber sizes with dilated, non-collapsing inferior vena cava.

Computed tomography of the chest showed no aortic aneurysm or dissection. Electrocardiogram showed normal sinus rhythm, normal axis, narrow QRS with non-specific ST changes suggesting repolarization abnormality. Troponin was elevated at 0.62 and trended up 1.05 before we proceeded with right and left heart catheterization. Right heart catheterization showed normal right atrial, ventricular and pulmonary artery pressures. Pulmonary capillary wedge pressure 10 mmHg; Pulmonary artery pressure 18/7 mmHg, pulmonary vascular resistance of 0.5 woods unit. Oxygen saturations in inferior vena cava, right atrium, ventricle, and pulmonary artery were consistent, 70-75% without significant step up, and hence, no evidence of intracardiac shunting.

Left heart catheterization showed right dominant system with normal, patent coronaries. However, the septal branch of left anterior descending coronary artery has small coronary artery fistula opening directly into the left ventricle. Multiple views were obtained and the fistula was determined to be 2-3 mm in size, believed to be most likely precipitating the patient's troponin leak and chest pain. Treatment plan was yet undecided as this involved the small septal branch, fistula size was 2-3 mm, and the patient was symptomatic with myocardial infarction.

Discussion:
CAF presentation with myocardial infarction, and septal branch location as seen in our patient, are both extremely rare. Approach to managing CAFs remains complicated and complex, and long-term outcome data is limited. Medium and large size fistulas are associated with greater long-term complications compared to smaller ones.

In review of the literature, the most important factors to consider when intervening appear to be fistula size, symptoms, anatomic complexity, and location within the cardiac vascular system. Percutaneous closure is often the preferred route in many patients owing to a less invasive approach with decreased recovery time and complications, especially for higher risk surgical candidates. Current techniques of percutaneous closure include detachable balloons, umbrella device, covered stent, Amplatzer devices for PDA, ASD, VSD or Vascular Plug closure, and embolization with coils. Surgical closure involving cardiopulmonary bypass and possible atriotomy or ventriculotomy is best suited for large diameter fistulas with unstable clinical presentation, those with highly turbulent flow, or significant vessel tortuosity.

2008 ACC guidelines for management of adult congenital heart disease recommend symptomatic patients and asymptomatic patients with hemo-dynamically significant or at high risk of complications should undergo closure of the fistula. However, other studies suggest closing all fistulas, regardless of size or symptoms, due to increased risk of infarction and complications. Despite their rarity, coronary artery fistulas should be considered as a differential diagnosis, especially in patients without risk factors. Correct diagnosis and management of this rare disorder is important to avoid long-term complications and mortality.

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Title: (Do not enter author details)

Pulmonary Embolism: The Great Maquerader.

Fatima Shahid[1], [1]*Cleveland Clinic Foundation, Cleveland, Ohio, USA*

Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Pulmonary embolism (PE) is a life threatening disorder with non-specific presenting signs and symptoms. Approximately 200,000 cases of PE are diagnosed annually in US alone. A missed diagnosis of pulmonary embolism can have catastrophic outcomes, with mortality rates approaching 30 percent in some reports. Herein, we report a case of PE with elusive presentation.

A 71 year old male was admitted to the coronary intensive care unit with acute onset of dyspnea on exertion. His electrocardiogram (EKG) revealed non-specific ST changes in leads V1-V2 and he had slightly elevated cardiac biomarkers. The transthoracic echocardiogram showed moderate left ventricular dysfunction (ejection fraction 38%) and normal right ventricular (RV) systolic function with no significant valvular abnormalities. On admission, left heart catheterization was performed which re-demonstrated known non-obstructive left anterior descending artery disease. However, patient went into intermittent slow wide complex junctional rhythm during the procedure. This was accompanied by hypotension and bradycardia requiring bolus of atropine and norepinephrine before reverting to sinus rhythm. He had similar episodes on telemetry later. He was initially determined to have accelerated idioventricular rhythm secondary to myocardial infarction. Cardiac MRI was performed to evaluate anterior wall injury which incidentally revealed saddle pulmonary embolism. Further testing revealed acute left leg DVT. Patient was discharged on warfarin.

Pulmonary embolism can present with various EKG findings ranging from normal to ST segment elevations. However, normal coronary arteries on an angiogram should alert one of this condition. It is also important to note that massive PE can be present in the absence of findings of RV strain on EKG and echocardiogram. Hemodynamic collapse can occur in the absence of such findings.

Ours is a case of novel presentation of PE masquerading as myocardial infarction with severe hemodynamic collapse at the time of angiography. Therefore, it is important to have a high degree of suspicion as correct diagnosis along with early intervention is the key to prevent adverse outcomes including death.

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Title: (Do not enter author details)

Leriche's syndrome: A rare complication following anterior approach lumbar spinal surgery

Ramyashree Tummala[1], Raktim Ghosh[1], Sravani Kamatam[1], Keyvan Ravakhah[1], Anjan Gupta[1], [1]*St Vincent Charity Medical Center, Cleveland, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

BACKGROUND: Leriche's syndrome is an aortic occlusive disease, which is due to obliteration of distal aorta above the site of bifurcation of common iliac arteries. The classic triad of symptoms include claudication, impotence, and absent or decreased femoral pulses. It may be acute or chronic in onset. Injuries of the thoracic and abdominal aorta after spine surgery are rare but may result in severe life threatening complications. Acute and chronic vascular injuries such as perforations leading to major bleeding or hematoma formation, erosions or pseudoaneurysm formation are some of the vascular complications of lower spinal surgeries. Post spinal surgery Leriche's syndrome often misdiagnosed because of overlapping symptoms of pseudo-claudication from spinal canal stenosis. We highlight a case of acute Leriche's syndrome after Anterior Lumbar Interbody Fusion (ALIF) surgery, and its presentation.

CASE PRESENTATION: A 58-year-old male patient presented to the hospital 3 weeks after ALIF surgery at L2-S1, performed due to lumbar spinal stenosis. He reported sudden numbness, tingling and weakness of both lower extremities from the waist down. He had none of these lower extremity symptoms before surgery. His vitals were unremarkable, whereas laboratory tests were inconspicuous. His past medical history was significant for osteoarthritis, chronic back pain, and hypertension. He was a chronic smoker with 30 pack years history. Our differentials were spinal cord compression, spinal cord abscess, retroperitoneal hematoma and myelopathy.
In neurological examination, there was loss of sensation to fine and crude touch in both lower extremities up to the mid thighs (L2-S1) and 4/5 power with +2 reflexes (patellar and ankle). Review of operative note revealed during disk removal and positioning the cages, the major vessels were mobilized to right side and protected by the retractor with no direct injury to any vessels. CT of the lumbar spine showed anterior interbody fusion changes at L2-S1 with intact hardware. CT angio showed extensive aortoiliac atherosclerotic disease with long segment occlusive thrombosis of infrarenal abdominal aorta by a crescentic mural thrombus. Diagnosis of acute Leriche's syndrome was established which was attributed to acute vascular injury following ALIF. The patient underwent emergent aortoiliac endarterectomy and aortobifemoral bypass.

DISCUSSION: Post-surgical Leriche's syndrome is rare and needs a strong index of suspicion to diagnose. A systematic review conducted by Wood et al showed that vascular injury in ALIF is less than 5% and complications being thrombosis and PE. A case report of an acute Leriche syndrome after PLIF highlighting iatrogenic trauma to aorta during spinal surgeries was described by in a 47-year-old female who developed an acute occlusion of the infrarenal aorta after surgery. In the literature review, ALIF surgery at L2-L5 levels is most likely prone to cause vascular injury due to retraction of vessels promoting thrombus formation by mechanical injury to the intima of artery. The retraction of iliac vessels in our case during ALIF could have promoted endothelial injury leading to thrombus formation.

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Rapid Desensitization to overcome severe contrast Allergy

Saurav Uppal[1], Anthony DeCicco[1], Yoon Kim[1], [1]*University Hospital Case Medical Center, Cleveland, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introductions: This is a case of recurrent, severe allergic reaction to contrast media complicating percutaneous coronary intervention (PCI) for a post-operative NSTEMI.

Case Description: Our patient is a 67 year-old man with a history of non-obstructive coronary artery disease (CAD) who was initially admitted to our affiliated Veterans Hospital for a planned cystoscopy/retrograde pyelogram. Following recovery he informed his nurse of intra-operative chest pain that had since resolved. An electrocardiogram (ECG) was unchanged from baseline. His first troponin was 0.18. A coronary angiogram from 2013 was known to have demonstrated a 50% stenosis in the mid left anterior descending artery (LAD), and otherwise mild disease. He was started on antiplatelet therapy, anticoagulation and continued on his beta-blocker and statin. His troponin peaked at 0.20 the following day. Urgent revascularization for NSTEMI was scheduled for the following morning.

Pre-catherization workup revealed a significant history of allergic reaction to contrast media resulting in nausea and an erythematous, pruritic rash following a contrast-enhanced CT. The patient underwent a coronary angiography in 2013 with prednisone 50 mg (13 hours, 7 hours and 1 hour pre-procedure), as well as diphenhydramine pretreatment 1-hour pre-procedure. Despite pretreatment he developed severe nausea and pruritis, with scattered erythema and bullae noted on exam. He was treated with intravenous methylprednisolone, famotidine and diphenhydramine, and remained in the ICU for 2 days. He was hospitalized again in 2014 after developing similar symptoms following another CT scan of the abdomen and pelvis.

Given the extensive history of allergic reaction our Allergy and Immunology team was consulted. Their recommendations were to use non-ionic and iso-osmolar contrast media and to pretreat with prednisone, diphenhydramine and ranitidine. A desensitization protocol prior to catheterization was also recommended. Desensitization was performed in our cardiac intensive care unit (CICU). Specifically, our patient received escalating doses of Visipaque administered intravenously every 10 minutes to build tolerance to contrast media. Notably, our patient was found to have a severe stenosis in the mid-LAD. He underwent successful PCI with a drug eluting stent (DES) immediately following his diagnostic angiography. He tolerated both procedures without any complications. During follow-up he remained asymptomatic, and was discharged home without incident the following day.

Discussion: Angiography is generally very well tolerated, adverse events can occur, commonly including reactions to contrast media. The appropriate management of adverse reactions to contrast media begins with prevention. Patients who are known to have had prior events with contrast should be pre-medicated with one of two now standardized regimens, both involving corticosteroids and antihistamines. Nonetheless, the efficacy of these interventions is not 100% and some patients continue to have breakthrough reactions like our patient. Desensitization is one of the strategies to overcome true refractory contrast reactions as demonstrated in this case.

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A Rare Case of Fungal Endocarditis

Aaron Whipp[0], [1]*Riverside Methodist Hospital, Columbus, OH, USA*

Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction

Infectious endocarditis (IE) is a heterogeneous syndrome most commonly caused by gram positive bacteria such as *Staphylococcus*, *Streptococcus*, and *Enterococcus* species. Although less common, fungal infections have also been shown to cause IE primarily in patients with prosthetic heart valves. Both *Candida* species and *Aspergillus* species have been reported, both with poor prognoses. We describe a case of native valve Aspergillus endocarditis in a patient with granulomatosis with polyangiitis on chronic immunosuppression.

Case Presentation

A 79 y/o white male with a past medical history of Granuomatosis with Polyangiitis (Wegner's vasculitis) with complications of diffuse alveolar hemorrhage and chronic kidney disease stage 4 on chronic cytotoxic medications and corticosteroids presented with chest pain and shortness of breath 1 week after undergoing radiofrequency ablation and permanent pacemaker (PPM) placement for recurrent atrial fibrillation. Trans-thoracic echocardiogram (TTE) revealed a moderately sized pericardial effusion without tamponade physiology. Hospital course was complicated by drug induced febrile neutropenia attributed to recent initiation of Propafenone and supported by bone biopsy. Follow up TTE 1 week after discharge showed resolution of effusion but with a new left atrial mass. The patient was re-admitted for evaluation with trans-esophageal echocardiogram (TEE) which confirmed an atrial mass measuring 2.5 cm x 0.7 cm located on the base of the anterior mitral annulus concerning for thrombus, myxoma, or vegetation. Cardiac MR was recommended but unable to be completed due to PPM. Infectious disease consultants withheld antibiotics in the absence of clinically infectious stigmata and negative blood cultures x2. Cardio-thoracic surgery opted not to perform surgical biopsy since the patient was a poor surgical candidate. After discharge, the patient continued to deteriorate with severe fatigue and weakness now with fever 101 F. Repeat blood cultures were drawn along with fungal cultures and empiric Vancomycin and Ceftriaxone were initiated. The patient underwent CABG x2 with surgical resection of the left atrial mass. Tissue biopsy with fungus cultures grew *Aspergillus fumigatus.* The patient was started on Amphotericin B and was transitioned to Voriconazole on discharge. Subsequent echocardiograms have shown a mildly decreased ejection fraction however no recurrence of vegetation. The patient is currently enrolled in cardiac rehab.

Discussion:

The incidence of infective endocarditis (IE) is estimated to be around 10 cases per 100,000 person years with fungal endocarditis accounting for only 2% of reported cases. *Aspergillus* is second only to *Candida* as the major contributing species of fungi. Traditionally, cases of Aspergillus IE have been associated with prosthetic heart valves occurring at the time of surgery or in the post-operative period. However, there is an increasing prevalence of cases among patients with severe immunodeficiency secondary to chemotherapy, steroids, or immunomodulatory pharmacologic therapy. Diagnosis can be challenging since patients will present with non-specific constitutional symptoms of fever, malaise, and weakness. Embolic complications of mycotic aneurysms are a commonly encountered minor Duke criteria that can heighten suspicion for fungal IE. Other diagnostic challenges include fungal cultures that take up to a week to grow and are rarely positive. Key to the diagnosis is trans-esophageal echocardiography (TEE) and is the imaging modality of choice to assess size of vegetation and valve function. Cardiac MR can also be used but has not yet been established. Overall prognosis remains poor with a mortality rate approaching 100%, despite advancements in antifungal therapy. The cornerstone of therapy is combined medical therapy with Voriconazole in conjunction with surgery.

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Title: (Do not enter author details)

Austrian syndrome with both mitral and tricuspid valve involvement: A rare presentation of a rare clinical entity.

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Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction
Austrian syndrome is a rare clinical condition characterized by classical triad of pneumonia, meningitis and endocarditis caused by Streptococcus pneumoniae infection. It was first described by Robert Austrian in 1957. Various cases of Austrian syndrome which have been reported in literature reveals predilection of streptococcus pneumoniae for aortic valve followed by mitral valve. Right heart endocarditis in Austrian syndrome has not been previously reported. We describe a case of 55-year-old male who presented with triad of pneumonia, meningitis and infective endocarditis involving both mitral and tricuspid valve due to pneumococcal infection. He was treated with IV antibiotics and discharged home on 2 weeks course after complete resolution of symptoms.

Case Description
A 55-year-old male with history of chronic alcohol use and schizoaffective disorder presented with progressively worsening headache, neck pain, productive cough, hemoptysis and malaise for two weeks. On initial presentation he was noted to be febrile with altered mentation. Physical examination was pertinent for nuchal rigidity and ejection systolic murmur over precordium. Laboratory work up was positive for leukocytosis and elevated lactic acid. Urine analysis was negative for infection and urine drug screen was also negative. Chest X-ray showed bilateral infiltrates, CT head was unremarkable and CSF analysis suggested bacterial meningitis. He was started on empiric intravenous antibiotics along with dexamethasone. Blood cultures and CSF cultures came back positive for streptococcus pneumoniae. Transthoracic echocardiogram showed moderate to severe mitral regurgitation with suspicion of a mitral valve vegetation. Trans-esophageal echocardiogram was then performed which confirmed vegetation on mitral valve as well as tricuspid valve. Cardiothoracic surgeon recommended antibiotic therapy. Antibiotic regimen was narrowed to IV ceftriaxone based on sensitivities with plan of antibiotic therapy for two weeks and follow up. Patient had resolution of his symptoms and he was discharged in stable condition to extended care facility.

Discussion
With prevalent antibiotic use, incidence of pneumococcal endocarditis has decreased and is now estimated to be 1-3%. However, given high mortality and potential complications like acute heart failure, valvular destruction, peri-valvular abscess and septic shock, early recognition and treatment is important. Alcohol abuse is major predisposing factor for Austrian syndrome. Smoking, chronic bronchopulmonary disease, malignancy, transplant, splenectomy and hepatic disease are other predisposing factors. For unclear reason, pneumococcal endocarditis involves aortic valve followed by mitral valve. Right heart endocarditis is rare specially in patients without intravenous drug use. Treatment of Austrian syndrome is predominantly antibiotic therapy with surgical intervention for valvular complications.