Ohio-ACC Poster Competition

CV TEAM

Case Abstracts

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Title: (Do not enter author details)

Cangrelor Use in the Treatment of Left Ventricular Assist Device Thrombosis: A Case Report

Robert Barcelona[1], Justin Delic[1], Chantal ElAmm[1], Daniel Simon[1], [1]*University Hospitals Case Medical Center, Cleveland, Ohio, USA*

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

Introduction/Objective:

Left ventricular assist device (LVAD) thrombosis is associated with significant morbidity and mortality. Reports of thrombosis rates have increased from 2.2% prior to March 2011 to nearly 8.4% since March 2011. Recently, clinicians have investigated a variety of strategies to treat these patients. In our report, we describe the use of cangrelor, an ultra-fast acting intravenous $P2Y_{12}$ inhibitor, for the treatment of a LVAD thrombosis in a patient with concurrent arteriovenous malformation bleeding.

Case Presentation:

The patient presented to his regularly-scheduled heart failure clinic visit for evaluation. Although he had not experienced any shortness of breath, fevers, or dyspnea on exertion, he was found to have an elevated lactate dehydrogenase (LDH) level of 950 units/L (baseline of 521 units/L), weight gain of 20 pounds since the previous month, and multiple pulsatility index events recorded within the previous days. The patient received a HeartMate II LVAD device two months prior as destination therapy and was receiving warfarin but no antiplatelet agents due to a history of gastrointestinal bleeding during a previous admission. Since thrombosis was suspected, the patient was admitted to University Hospitals Case Medical Center for management. Vitals were all within normal limits (mean arterial pressure 93 mmHg, heart rate of 85 beats per minute, respiratory rate of 20 breaths per minute, temperature of 36.7^{o} C, and SpO_2 of 99% on room air). After repeat testing, a peak LDH of 1,448 units/L and hemoglobin of 7.8 g/dL were noted, and the patient was initiated on aspirin and intravenous heparin. Although LDH initially declined over the next two days to 850 units/L, it rose to 1,013 units/L, and power surges were noted. At this time, the patient was initiated on ticagrelor and continued on aspirin and intravenous heparin. However, he experienced severe nausea from ticagrelor and was switched to clopidogrel the following day. With this therapy his LDH continued to decline steadily. However, he developed arteriovenous malformation bleeding with hemoglobin levels dropping to a low of 6 g/dL. He received a total of twelve units of packed red blood cells. As the patient was being prepared for emergent device exchange, it was decided to utilize cangrelor, an ultrafast-acting intravenous $P2Y_{12}$ inhibitor that would provide the benefit of clearing the body quickly if discontinuation was necessary. The patient was converted from clopidogrel to cangrelor at a dose of 0.75 mcg/kg/min and continued on this regimen for six days. Hemoglobin stabilized at 7.8 g/dL and LDH decreased to 403 units/L. The patient was transitioned from cangrelor to dipyridamole and continued on aspirin and intravenous heparin, which was subsequently switched to warfarin.

Discussion/Conclusion:

Medical treatment of device thrombosis typically consists of a combination of both antiplatelet and anticoagulant therapy and is challenging with current agents. This case report describes the first use of cangrelor for the treatment of LVAD thrombosis. The results of our current report suggest that cangrelor, because of its pharmacokinetic profile, may be an option for the treatment of LVAD thrombosis in patients with concomitant bleeding complications. However, the most appropriate dosing and duration, as well as potential safety issues are unknown for this indication.

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Case Report: Proarrhythmia and Pacemaker Pseudo-Malfunction with DDIR Pacing

Melanie Gura[1], Tanya Verga[1], Diego Alcivar[1], Timothy Byrnes[1], Deepak Swaminath[1], Otto Costantini[1], [1]*Summa Health System, Akron, OH, USA*

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

Initially thought of as an upgrade to DVI pacing, the DDI(R) pacing mode promotes AV sequential pacing with dual-chamber sensing. Although DDI pacing allows for atrial sensing as well as ventricular sensing, the mode of response is inhibition only, i.e. no P waves can be tracked as a response to atrial sensing. Consequently, in DDI pacing, the ventricular rate cannot be greater than the programmed lower rate limit (LRL) and in DDIR pacing the ventricular rate cannot be greater than the maximum sensor rate (MSR). The timing cycles include the LRL, AV interval (AVI), post ventricular atrial refractory period (PVARP), the ventricular refractory period (VRP) and MSR. A rare exception may occur when an intrinsic ventricular complex occurs after a paced atrial beat (AR) and inhibits a paced ventricular output before completion of the programmed AVI or due to functional atrial pacing greater the MSR.

Case Report: We report the case of an 86 year-old female with past medical history of hypertension, hypercholesterolemia, and dementia. She presented to the emergency department with extreme lethargy, and a recent change in mental status at home. Chest x-ray revealed cardiomegaly, bilateral effusions, and vascular congestion consistent with congestive heart failure. Computed tomography of the head revealed brain atrophy. An electrocardiogram revealed a junctional rhythm with a rate of 50 bpm. A dual chamber pacing system was implanted and programmed to the DDIR mode with a LRL of 60 ppm, an AV delay of 350 ms, and a MSR of 120 ppm to minimize ventricular pacing and to provide rate modulation. Routine device follow-up and remote monitoring revealed normal DDIR pacemaker function. A routinely scheduled remote evaluation eleven months post implantation revealed electrograms (EGMs) that exhibited atrial pacing markedly exceeding the programmed MSR of 120 ppm. The atrial pacing cycle length was 320 ms (190 bpm) due to ventricular based timing that is seen in non-tracking modes. Careful review of the EGMs revealed a 1:1 sensed ventricular rate comparable to the atrial rate indicative of excellent AV nodal conduction. The rhythm also started with a premature atrial sensed event that occurs within the atrial blanking period, causing a ventricular sensed event, which in turn initiates an atrial paced event occurring at a fast rate due to the ventricular based timing cycles The patient was subsequently reprogrammed to the DDDR mode with a LRL of 60 ppm and MSR of 110 ppm. Remote evaluations done at two weeks, one month and 3 months post reprogramming revealed no high atrial pacing rates and no true ventricular high rate episodes.

Conclusion: While the DDI (R) mode is an effective form of pacing, permitting non-competitive atrioventricular sequential pacing and functional atrial pacing support, limitations not only include effective VVI pacing during intact ventriculoatrial conduction, functional undersensing when long VBP are programmed and inappropriate atrial high rate pacing well above the MSR. DDI (R) pacing is a unique strategy to optimize pacing therapy to minimize ventricular pacing. Reprogramming of the pacemaker should be individualized to the patient to achieve the optimal effect.

Fellow in Training

Case Abstracts

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Title: (Do not enter author details)

Internal Mammary Artery To Pulmonary Vasculature Fistula, Case-Series and Systematic Review.

Ali Abdul Jabbar[2], Amish Patel[1], Nathan Marzlin[1], Omar Mufti[1], Ajay Agarwal[1], [1]*Wright State University, Dayton, Ohio, USA,* [2]*University of Toledo Medical Center, Toledo, Ohio, USA*

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

Background: Internal mammary artery (IMA) to pulmonary vasculature (PV) fistula is a rare anomaly. The etiology can be congenital however; most cases have been associated with coronary artery bypass grafts (CABG), trauma, inflammatory conditions, chronic infections or neoplasia. Most of the information about the formation of these fistulas comes from scattered case reports in current literature.

Objectives: To report the first large case-series study on IMA-PV fistula; establish trends in risk factors, clinical presentation, and discuss the experts' consensus for follow up and treatment strategies of this rare condition.

Methods: A systematic review of the literature was performed. Medline, PubMed, and Google Scholar were searched for case reports of internal mammary artery to pulmonary vasculature fistula. Each case was reviewed and the data was collected. The patients were compared on sex, age of onset, presenting symptoms, time from surgery and treatment when applicable. Other possible contributing factors were also examined. Patient treatment and outcomes were compared when reported. Cases published in a language other than English were excluded.

Results: Sixty-five cases of IMA-PV fistula were published in the literature. Three cases were excluded due to insufficient data or language of publication. Ten were congenital fistulas and the rest were of acquired aetiologies. The average age of presentation for congenital fistulas was 41.3 year old, although few outliers skewed the mean. The classical picture of congenital fistulas is found in a young adult with incidental continuous murmur on physical exam. The mean age of a patient with an acquired fistula was 55.4 years with an average post CABG date of 3.99 years. While the mean was around 5 years post procedure the range of presentation was 2 months to 16 years. By far the most common presenting symptom was angina. Other patients presented with dyspnea with few presented with myocardial infarctions.

Males outnumbered females in the acquired fistula formation. Only 8.2% of the reported acquired fistulas were female. However six out of the ten congenital cases were female. The incidence of fistula detection also seems to be increasing. In the most recent retrospective study of 537 CABG patients, the incidence of IMA-PV fistula was close to 1%. Since the first case-report publication in 1947, 64 other cases were reported. Peak of publications was around 2010. Myocardial perfusion imaging can detect ischemia in the territories supplied by the IMA graft representing a true steal phenomenon. Treatment strategies include, conservative medical management, surgical ligation, and/or percutaneous approach. The latter include coil-embolization, use of vascular plugs or covered stents to occlude the fistulous communication. Success has been reported with either approach.

Discussion: Although uncommon, internal mammary artery to pulmonary vasculature fistula should be considered in the differential diagnoses of worsening symptoms post coronary artery bypass grafting. The aetiology of post-operative IMA-PV fistula is still unclear. It has been suggested that electrocoagulation over surgical clipping may increase the risk of neovascularization. The technique of a pericardial flap in CABG operations has been reported to increase the risk of fistula formation. Two cases were associated with an infectious process and one case was associated with a vasculitis.

Advanced imaging modalities can delineate the course of the fistula and is helpful when the anatomy is complex, but a selective angiogram injection of the IMA graft at the time of cardiac catheterization is the most cost-effective way to establish the diagnosis of IMA-PV connections.

Conclusion: The rate of post-CABG fistula cases is increasing. It is therefore important to understand the presentation of IMA to PV fistulas, be familiar with diagnostic modalities and management options. The treatment of IMA-PV fistulas remains a clinical judgment that is based on the severity of the presentation. While conservative medical management is suitable for mild symptoms, percutaneous intervention with covered stents and/or coil embolization is proven to be a feasible and effective therapeutic option for those who fail medical therapy or with more advanced symptoms. Surgical ligation or clipping may be reserved for low-risk candidates and for those who are not amenable to percutaneous intervention.

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Title: (Do not enter author details)

Double Jeopardy: 2 Distinct cases of Multifocal Bilateral Stroke secondary to Nonbacterial Thromboembolic Endocarditis in the setting of Metastatic Lung Adenocarcinoma

Anthony Ciraldo[1], Diego Alcivar[1], Brian Donelan[1], [1]*Summa Health System, Akron, Ohio, USA*

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

Objectives: 1. Recognize the clues that should lead a physician to consider malignancy as a cause of unexplained Non Bacterial Thrombotic Endocarditis (NBTE). 2. Review the pathophysiology, diagnosis and management of malignancy induced NBTE

Case # 1: 46 yo female with PMH of HLD is admitted for AMS 15 min prior to arrival plus headache, accompanied by spasticity in the left hand as well as left arm paresthesia. CT negative for bleed. Troponin 0.513. ECG unremarkable. MRI head revealed multiple bilateral strokes. TTEcho wnl and TEE showed small mobile mass (0.7 by 0.3cm) on the noncoronary cusp of the aortic valve consistent with a vegetation and likely etiology of emboli source. Blood cultures were negative. Was started on broad-spectrum antibiotics, she had no further fever, chills, or other infectious signs initially. Few days into hospitalization presented left flank pain and was found to have left renal infarct. Also developed worsening pulmonary infiltrates on CXR and was treated for pneumonia. Days later was not improving and began having fevers, diffuse rash as well as worsening of a left lung infiltrate. CT chest was obtained and a large lung mass was found concerning for cancer. CT-guided biopsy revealed adenocarcinoma of the lung. Therapeutic thoracentesis showed malignant cells confirming diagnosis of stage IV adenocarcinoma of the lung. Oncology was consulted for therapeutic options and were decided to be provided as outpatient. Anticoagulation was started and antibiotics were also discontinued due to the suspicion of drug rash, and with discontinuation the rash improved. On discharge day, the patient's VS, PE and labs were improving, with Oncology follow up. Case # 2: 59 yo Male with a recent tooth extraction and a flu-like illness 2 weeks prior presents with aphasia. VS and PE wnl aside from aphasia. CT Head showed scattered hypodensities, troponin 3.36 and EKG NSR. CXR diffuse interstitial pattern and WBCs 14,000. Anticoagulation was held until stroke was ruled out. MRI brain showed multiple areas of acute ischemia cerebral and cerebellar hemispheres bilaterally. Echocardiogram showed normal EF and an oscillating vegetation on the aortic valve, and was placed on IV vancomycin and rocephin with prior blood cultures drawn. Taking the history, vegetation and multiple bilateral strokes, consideration for septic emboli arising from his aortic valve endocarditis. His blood cultures showed no growth. No surgical intervention was planned for the endocarditis and medical management was continued with IV antibiotics. Neurological status continued to improve and repeat echo demonstrated stable aortic valve vegetation, but new posterior mitral valve leaflet thickening, for which continued long term antibiotics. 3 Weeks later returns with AMS and dyspnea; CT head showed a SAH at the left occipital lobe. Following day repeat CT showed improving SAH, but new acute area of cortical infarction in the right MCA distribution. CT Chest showed mediastinal and bilateral supraclavicular and hilar adenopathy with a 1.9 cm irregular soft tissue density in the left upper lobe, with suspicion for malignancy with lymphangitic spread within the left lung and possible superimposed pneumonia. Biopsy of the supraclavicular lymph node displayed metastatic adenocarcinoma of the lung. Given the patient's comorbibities, the complexity of his medical problems, and his rapidly deteriorating state, Palliative Care was consulted with no further aggressive treatments, dying 3 days later.

Discussion: Stroke is a frequent complication in patients with cancer, occurring in nearly 15% of patients with cancer, however cerebrovascular disease and Nonbacterial thrombotic endocarditis (NBTE) may precede a diagnosis of cancer and be the first clinical evidence of an underlying malignancy. Malignancy-related thromboembolism can present as acute cerebral infarction, nonbacterial thrombotic endocarditis and migratory thrombophlebitis. Generally attributed to a cancer-related hypercoagulable period,disseminated intravascular coagulopathy or tumor embolism. In cerebral venous occlusion, the initial symptom is usually a headache. A systemic cancer workup should be considered in patients in whom stroke origin is unclear or who have an early vascular recurrence. In cases of coexisting cancer and embolic stroke, we should consider the possibility of NBTE which commonly occurs in advanced malignancies. Systemic thrombosis, embolism, or hemorrhage can be a clue to the cause, and appropriate neuroimaging and coagulation studies to aid in the diagnosis. NBTE can be diagnosed by transthoracic but mostly transesophageal echocardiography. Cancer treatments may also contribute to this coagulopathy. There is no established treatment for the thrombotic coagulopathy associated with cancer, but anticoagulation should be considered; in the other hand clinical signs of cerebral hemorrhage are fulminant and may be fatal. The bleeding usually occurs in the parenchyma or subdural compartment, and rarely in the subarachnoid space. Outcome is poor and correlates with both severity of neurologic disability and the stage of tumor. Therapy may ameliorate symptoms or prevent further episodes. The identification of one of these unusual stroke syndromes and NBTE that leads to the diagnosis of an occult and treatable cancer can be particularly rewarding, given that treating the underlying disease in early stages may be curative. These cases were found while being in the consult service and emphasize that stroke and NBTE can be the first manifestation of an undiagnosed cancer, also provide their different diagnostic and management options.

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Title: (Do not enter author details)

Takotsubo Cardiomyopathy after Permanent Pace Maker Placement

Sajid Ali[1], Malavika Balachandran[1], Yousuf Kanjwal[1], [1]*Mercy St Vincent Hospital and Medical Center, Toledo, Ohio, USA*

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

Introduction:
Takotsubo cardiomyopathy is characterized by transient apical dysfunction in the absence of obstructive coronary artery disease. Takotsubo most commonly is described occurring after a stressful emotional event. The underlying mechanisms have not been clearly established, but major hypotheses to its etiology include microvascular dysfunction, with catecholamine induced cardiomyopathy. However there are many unique conditions reported including but not limited to snake bites, pancreatitis, post operative, and medication induced which can give the same presentation in the absence of a stressful emotional event. We present the first documented case of takotsubo cardiomyopathy in a male patient after permanent pace maker placement.

Case:
We describe a 65 year old white male with past medical history of diet controlled diabetes mellitus and hypertension who came in with diarrhea for 3 days with generalized weakness. Upon arrival to the emergency department he was found to be in two to one AV block. AV nodal blocking agents were discontinued and a two dimensional echocardiogram and persantine nuclear stress test were ordered. Echocardiogram showed normal left ventricular function with normal wall motion and nuclear stress test showed inferior hibernating myocardium. The patient subsequently received a cardiac catheterization which showed normal coronary arteries with normal wall motion and normal left ventricular ejection fraction on ventriculogram. The patient continued to have intermittent symptomatic 2:1 AV block. The decision was made to place a dual chamber permanent pace maker.
Dual chamber permanent pace maker was successfully placed on day 4 after admission and was shown to be functioning appropriately the day after placement. Shortly after device check, patient had an episode reported by nursing where he felt as though he was going to pass out. His systolic blood pressure was transiently in the 60's with decreased heart rate which normalized after a few minutes. A stat repeat echo, troponin, chest x-ray and cbc were ordered. His haemoglobin was stable, and troponin T was elevated at 0.04. EKG showed atrial sensed ventricular paced rhythm. Chest x-ray at this time was unchanged and patient denied chest discomfort. However the trans-thoracic echocardiogram showed acute findings of septal mid-chamber, lateral mid-chamber and apical hypokinesis with dilatation very suggestive of takotsubo cardiomyopathy. Ejection fraction at this time was rated 35 percent. The vasovagal like symptoms improved immediately and he remained asymptomatic from then on.

Discussion:
MayoClinic defines takotsubo cardiomyopathy by four defined characteristics: Transient hypo-kinesis, a-kinesis, or dyskinesis of the left ventricular mid segments (more than one coronary territory) with or without apical involvement with a stressful trigger often, but not always present in the absence of obstructive coronary disease. New ECG abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin with absence of pheochromocytoma or myocarditis.
Our patients EKG changes were precluded as he was ventricularly paced and also had underlying LBBB. He did have a mild troponin elevation of 0.04 which is typical for takotsubo. He did not have clinical manifestations of pheochromocytoma and thus did not warrant a work up for this. Echocardiography showed the characteristic mid to apical ballooning with severe hypokinesis of the apex to mid left ventricular cavity which manifested only after the pace maker placement.
No case report in our literature search has shown PPM placement as the sole cause of TCM in a male patient.
Studies have shown that chronic RV pacing causes abnormal electrical and mechanical activation pattern of the ventricles, resulting in mechanical dyssynchrony and deterioration of LV function. A study was conducted in 25 individuals testing the effect of RV pacing on the LV function without any evidence of structural heart disease using 2D speckle tracking stain imaging. The study showed that direct stimulation of RV induces an abnormal activation sequence resulting in asynchronous ventricular contraction. However, this asynchrony would not account for the multi-vessel territory involvement seen in TCM. Another study in animal studies showed that rapid RV pacing required compensatory reductions in myocardial contractility to balance the myocardial metabolic requirements causing acute myocardial hibernation. Again, this is precluded in our case as our patient never had rapid pacing. On follow up echocardiogram two months later, LV function and wall motion returned to normal. Our case demonstrates a new cause of TCM in a male patient from RV pace maker placement. The etiology of this potential outcome needs to be further studied.

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Title: (Do not enter author details)

Turning into a Block,
A Case of Complete Heart Block in Turner Syndrome

Muhanad Al-Zubaidi[1], Omair Ali[1], Thein Aung[1], Yasir Al-Zubaidi[2], Abdul Wase[1], [1]*Wright State University, Dayton/Ohio, USA,* [2]*University of Minnesota, Twin Cities/Minnesota, USA*

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

Background: Various cardiovascular abnormalities have been reported as associated phenomenon in Turner Syndrome (TS) patients. The association between complete heart block and TS is not well established.

Case Presentation: A 56 year-old TS female presented to the emergency room with dyspnea, general weakness and lightheadedness. She had no history of syncope. Heart rate was between 31-42 beats per minute with blood pressure value in the lower normal range.

She received IV atropine with no improvement in her bradycardia. EKG showed complete heart block thus she was transferred to the Intensive care unit for further management. Hypothyroidism and acute coronary syndrome were ruled out. The patient was on verapamil SR 120 mg daily, which was discontinued since presentation. After more than 24 hours off any atrioventricular nodal blocking agents, she remained in complete heart block. Permanent pacemaker was placed and the patient was discharged in stable condition without any dizziness or lightheadedness.

Discussion: TS is the most common chromosomal disorder in females. Recent studies have demonstrated electrocardiographic abnormalities in individuals with TS indicating a more extensive involvement of the cardiovascular system than previously thought. Bondy et al. demonstrate that women with TS were significantly more likely to demonstrate left posterior fascicular block and prolonged corrected QT interval. Gene mutations have been speculated as a possible mechanism of cardiac conduction diseases in TS females. Mutations in the major long QT syndrome genes and in a type of filamin-A gene have been reported as potential etiologies for the cardiovascular anomalies in TS.

To the best of our knowledge, our presented case is the first presentation discussing the possibility of a rare complete heart block phenomenon in TS.

Conclusion: complete heart block can be part of the spectrum of cardiac conduction anomalies in TS. Incidence and mechanism are unknown and further investigated is warranted.

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Title: (Do not enter author details)

A Case of Separate Coronary Ostia and Absent Left Main Coronary Artery in a Patient with Holt-Oram Syndrome and Sinus Node Dysfunction

Thein Tun Aung[1], Samuel Roberto[1], Abdul Wase[1], [1]*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

Holt-Oram syndrome (HOS) is a rare but significant syndrome consisting of structural heart defects, conduction abnormalities and upper extremity anomalies. HOS was first described in 1960 by Mary Holt and Samuel Oram as a report of an atrial septal defect, conduction disturbances, and hand malformations occurring in family members. It is also known as Heart-Hand Syndrome type 1 and the most common of the rare Heart-Hand syndromes or atrio-digital dysplasia. Patients can present with heart blocks or symptoms from underlying congenital heart defects. Upper extremity deformity has to be present to diagnose HOS.

Case presentation

A 41-year-old male with hypertension, hypercholesterolemia, asthma, HOS with repaired heart defect and remote history of syncope presented to the emergency room following a witnessed seizure episode. The patient's wife witnessed him having muscle contractions and called the EMS. He woke up in the ambulance confused for a short time. There was no urinary or bowel incontinence, and no tongue biting. He had never been diagnosed with a seizure disorder.

The patient had been diagnosed with HOS, with an atrial septal defect (ASD) corrected at seven years old. Cardiac catheterization records showed absent left main coronary artery, separate ostia of the left anterior ascending and left circumflex coronary artery. Coronary arteries were patent. Head-CT and MRI revealed no acute intracranial process. Electroencephalogram showed no epileptiform discharges or focal abnormalities. The patient had no recurrence of seizure during his hospitalization.

Physical exam showed bilateral atrophic upper extremities at his baseline, with weakness of the intrinsic hand muscles. Cardiovascular examination revealed an inconsistently irregular rhythm. EKG showed junctional escape rhythm, rate of 57 beats per minute. Interventricular conduction delay was diagnosed due to QRS duration 124 milli-seconds. Corrected QT interval was 432 msec. Telemetry showed frequent sinus arrest with junctional escape rhythm during the night. Cardiology was consulted for heart block. Patient received emergent placement of temporary pacemaker. Echocardiography revealed enlarged left ventricle and generalized hypokinesis with estimated ejection fraction around 45%.

The electrophysiologist evaluated the patient. Considering the singular nature of seizure and evidence of sinus arrest, there was concern for the possibility of bradyarrhythmia including asystole leading to seizure-like activity. The patient received temporary transvenous pacemaker placement for precaution. Electrophysiology study was performed the subsequent day. The corrected sinus node recovery time was 885 msec at cycle length of 600 msec, clearly an abnormal finding. Sick sinus syndrome was diagnosed. AV nodal wenckeback block was noted at 410 msec. Ventricular tachycardia was non-inducible during EPS with different cycle lengths and also procainamide infusion. The patient underwent pacemaker placement since the likelihood of advancement in his heart block was high. He was discharged the next day in stable condition.

Discussion

This is a young male with Holt Oram who presented with a seizure-like episode attributed to hypoxia during asystole from an underlying cardiac conduction defect associated with HOS. Patients can present with heart blocks or symptoms from underlying congenital heart defects, including congestive heart failure. Arrhythmias and heart blocks are common in HOS patients, and conduction defects are highly associated with congenital heart defects. Atrial and ventricular septal defects are the most common defects. Our patient had an ASD repaired at a young age.

Holt-Oram Syndrome rarely presents with coronary artery anomali. There was one case report by Vianna, et al in 2011 regarding an anomalous right coronary artery. There is no reported case of separate coronary ostia and absent left main coronary artery to our knowledge. Our patient has separate coronary ostia for the left anterior descending artery and left circumflex artery, with an absent left main coronary artery. Prompt diagnosis is important since anomalies in coronary and upper extremity vasculature might be challenging for invasive procedures.

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Title: (Do not enter author details)

A Tale of Two Outcomes: Pseudoaneursym of the Mitral-Aortic Intervalvular Fibrosa; A Rare and Dangerous Condition.

R. Jordan Bohinc[1], Karishma Samtani[2], Vijai Tivakaran[3], Jacob Gibson[1], [1]*Kettering Medical Center, Kettering Ohio, USA,* [2]*Wright State Univerisity, Dayton, Ohio, USA,* [3]*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)

Fewer than two hundred cases of pseudoaneurysm of the mitral-aortic intervalvular fibrosa (P-MAIVF) have been reported in English literature since 1960. We report two cases of P-MAIVF discovered in Dayton, OH. The first is a 70 year-old male who underwent aortic valve replacement in 2004 with a porcine valve. He was later found to have P-MAIVF on transthoracic echo which was confirmed by transesophageal echo. He remained asymptomatic. He decided against pursuing surgical treatment of his pseudoaneurysm and later died at age 80 of an unrelated cause. The next was a 43 year old male who had undergone AV replacement on two separate occasions for recurrent endocarditis, initially with a bioprosthetic valve followed by a mechanical valve. He was admitted with another episode of infective endocarditis involving the mechanical aortic valve. TEE revealed P-MAIVF. Patient was transferred to tertiary care hospital, however expired prior to surgery.

The mitral-aortic intervalvular fibrosa (MAIVF) is a relatively avascular structure which lies between the left half of the noncoronary cusp and the adjacent third of the left coronary cusp of the aortic valve, alongside the left ventricular outflow tract. The limited blood supply to this area, makes it prone to injury from conditions such as aortic valve surgery, trauma or infection and may lead to irreversible damage and distortion of the tissue.

P-MAIVF is a rare and potentially fatal abnormality. Review of the literature identified a total of 166 cases in articles published from 1960-March 2014. There is a slight male predominance and any age can be affected. Unless complications develop, P-MAIVF is usually asymptomatic. Common presenting symptoms include signs and symptoms of infection, heart failure, systemic embolization and chest pain. Complications include rupture, fistula formation, coronary artery compression, and damage the mitral apparatus. Frequently there is compression of the left atrium.

While MAIVF can be seen with TTE, TEE has greater sensitivity (90% vs 43%). Identification of an echo-free area near the aortic root with systolic expansion and diastolic collapse confirms the diagnosis of P-MAIVF. An important clinical distinction is with aortic ring abscess in which this phenomenon is absent, Cardiac computed tomography (CT) and magnetic resonance imaging (MRI) can be further helpful in surgical evaluation. The clinical outcome of P-MAIVF is hard to predict. Rupture of the pseudoaneurysm into the pericardium may be fatal and hence when P-MAVIF is diagnosed, surgical treatment should be recommended to all patients even if they are asymptomatic.

In conclusion, P-MAIVF is a rare condition usually associated with infectious endocarditis or surgical trauma. Still, in the appropriate clinical setting physicians should be mindful of P-MAIVF and its complications. Our cases highlight the variable outcomes for P-MAIVF. Optimal management remains unclear and further studies are required in order to earlier identify and effectively treat patients with P-MAIVF.

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Takotsubo Cardiomyopathy Induced Conduction Disorder After Beta Blocker Discontinuation

Timothy Byrnes[1], Ottorino Costantini[1], [1]*Summa Health System/NEOMED Program, Akron, Ohio, USA*

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

Introduction:
Takotsubo cardiomyopathy was first described in 1991 and is an established cause of transient left ventricular systolic dysfunction. Several case reports have described a link between Takotsubo cardiomyopathy and the development of conduction disorders. We present the first definitive case of Takotsubo cardiomyopathy induced left bundle branch block.

Case:
-HPI: 86 year old female presents with the chief complaint of retrosternal chest pain and new left bundle branch block. She describes the chest pain as intermittent for 7 days, onset shortly after an argument with her husband, and occasionally exacerbated by activity and relieved with oral antacid. Denis any other exacerbating or remitting factors. Her primary care physician discontinued her beta blocker 2 months prior due to asymptomatic bradycardia. She admits to frequent arguments with her husband, but none since her medication change until 1 week ago.
-Past Medical History: stroke, aortic stenosis, left ventricular hypertrophy with left ventricular ejection fraction of 65%, peripheral artery disease, hypertension, diabetes mellitus type 2, glaucoma, gastritis
-Past Surgical History: aortic valve bioprosthetic replacement, glaucoma, bilateral carotid endarterectomy, hip replacement
-Home Medications: clopidogrel 75 mg daily, fish oil 1000 mg daily, hydrochlorothiazide 25 mg dialy, metformin 500 mg daily, simvastatin 40 mg daily, and omeprazole 20 mg daily.
-Vital Signs: 98.2* F, 78 bpm, 174/76 mm Hg, 14 rpm, 97% on room air
-Physical Exam: normal, including regular rate and rhythm with normal S1 and S2 and absence of S3 or S4.
-Labs: BMP and CBC within normal limits. Troponin initially 0.310 ng/mL and on 6 hours later 0.431 ng/mL.
-Imaging: ECG: sinus rhythm with new left bundle branch block compared to ECG from two months earlier.
-Hospital Course: Patient was loaded with aspirin and admitted to cardiac intensive care unit for non-ST-elevation myocardial infarction on heparin and nitroglycerin drips, clopidogrel 75 mg daily, metoprolol 12.5 mg twice a day, and lisinopril 5 mg daily. The following day she underwent coronary angiography and transthoracic echocardiogram, which found a left ventricular ejection fraction of 35% with mid anterior, mid anteroseptal, and apical akinesis, hyperkinesis of the basal segments, normal bioprosthetic aortic valve, and no obstructive coronary artery disease. Patient was discharged home the following day.

Discussion:
Takotsubo cardiomyoapthy is classically described as a transient stress induced myocardial dysfunction. In recent years, several reports have described the development of conduction disorders with Takotsubo cardiomyopathy, most commonly advanced AV block. Although, the myocardial dysfunction of Takotsubo cardiomyopathy will typically resolve within a few months, the conduction disorder is usually permanent. Based on our review of the literature we were only able to find two cases of patient's with "new" left bundle branch blocks on presentation of Takotsubo cardiomyopathy, however neither provided or stated if a prior electrocardiogram was available for comparison. Our case represents the first definitively described case of Takotsubo cardiomyopathy induced left bundle branch block. Additionally, our case illustrates that beta blockers may prevent the development of stress induced cardiomyopathy, as our patient was subjected to similar stress in the past without the development of Takotsubo cardiomyopathy and only developed this condition after she discontinued her beta blocker.

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Title: (Do not enter author details)

Percutaneous Removal of Right Atrial Thrombus in a Combined Liver-Kidney Transplant Patient

Darek Sanford[1], Patrick Daly[1], Timothy Smith[1], [1]*University of Cincinnati Medical Center, Cincinnati, Ohio, USA*

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

Introduction/objective
Intracardiac thrombus formation is a known but rare complication (1-1.5%) of orthotopic liver transplantation. Large thrombi do not resolve spontaneously and typically require lysis or removal to prevent pulmonary embolism. The mortality rate in such cases is high (68%) with the majority of patients dying in the operating room. The management of right heart thromboembolic disease still remains unclear as no large-scale, blinded, randomized prospective trial has compared the current available therapies of anticoagulation, surgical embolectomy, systemic and catheter-directed thrombolysis and percutaneous thrombectomy. We present a case of percutaneous removal of a large, 2x2cm right atrial thrombus in a patient undergoing combined liver-kidney transplant immediately after liver transplant.
Case presentation
A 64 year old male with cirrhosis from Hepatitis C and orthotopic liver transplant (OLT) five years prior complicated by de-novo autoimmune hepatitis and chronic rejection of allograft was admitted for acute renal failure. He was re-listed for liver-kidney transplant due to steadily increasing MELD. He was taken to the operating room for repeat liver transplant with kidney transplant to follow. Following the liver transplant, the patient had a transthoracic echocardiogram (TTE) due to hypoxia and hypotension. TTE revealed a large, extremely mobile right atrial thrombus with prolapse into the right ventricle. There was moderate dilation of the right ventricle (RV) and RV systolic dysfunction along with moderate pericardial effusion. Heparin infusion was started and patient was moved to the catheterization lab while under general anesthesia for percutaneous thrombus removal. Venous access was obtained in the left femoral vein and a pigtail catheter was advanced to the IVC. A Swan-Ganz catheter was advanced into the left pulmonary artery under fluoroscopic guidance. Pulmonary angiogram at this time showed no distal embolization after which the pigtail catheter was exchanged for an AngioJet Omni catheter. Two pulse thrombectomies with tissue plasminogen activator (tPA) were performed followed by serial suction thrombectomies, each not exceeding 20 seconds. Echocardiography confirmed effective removal of the thrombus. Repeat pulmonary angiogram revealed no evidence of pulmonary embolism. Expected transient bradycardia was noted during suctioning which was minimized by atropine. The patient was taken directly back to the operating room for kidney transplantation. Repeat TTE following successful kidney transplant confirmed complete removal of right atrial clot burden. The previously seen RV dilation and dysfunction improved but did not return to normal. After a prolonged ICU stay, the patient was discharged to a skilled nursing facility.
Discussion/Conclusion
Conservative management with thrombolysis was found to be superior to anticoagulation and surgical embolectomy by Rose et al. with respective mortality: 29%, 24% and 11%. It has also been proven to have mortality benefit in massive pulmonary embolism, though it did not change outcomes in submassive pulmonary embolism. Though it avoids the trauma of surgical embolectomy it is not without complications. Major bleeding occurs in 20% of patients and a contraindication to therapy is seen in as many as 50%. Percutaneous thrombectomy is an alternative in this population that has been shown to be safe and effective. Large clinical trials are needed to compare outcomes between systemic thrombolysis and percutaneous thrombectomy. In this case percutaneous thrombectomy allowed the patient to proceed with planned combined liver-kidney transplant which would not have been possible with systemic thrombolysis.

Image 1. TTE showing RA thrombus

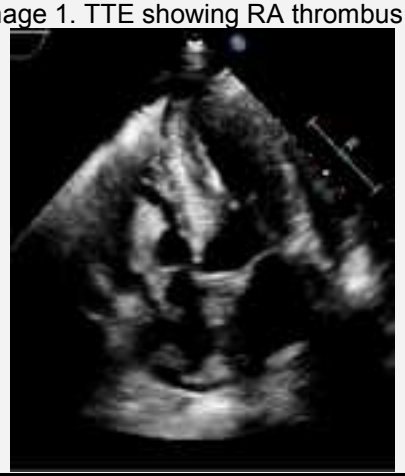


Image 2. Catheterization film of AngioJet

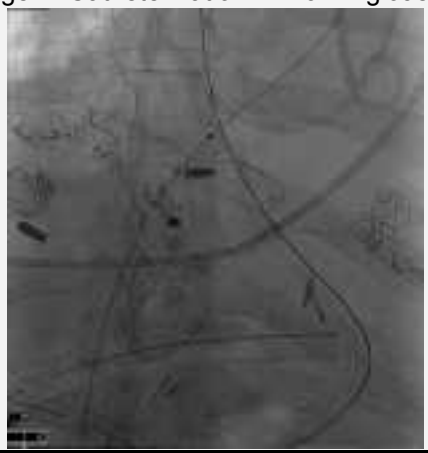
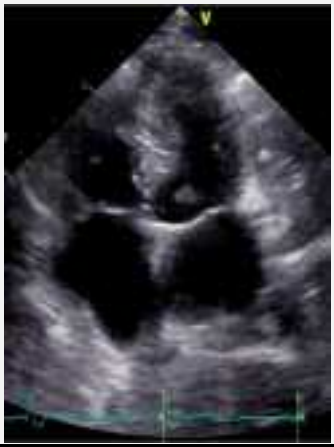


Image 3. TTE post thrombectomy



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Title: (Do not enter author details)

A Rare Case of Restrictive Cardiomyopathy with Nemaline Rods

Mohamed Elamin[1], Amber Patton[2], William Gunning, III[1], Samer Khouri[1], *[1]University of Toledo, Toledo, OH, USA, [2]Pathology Laboratories, Toledo, OH, USA*

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

Introduction:
Nemaline Myopathy is typically a skeletal muscle disorder defined by the presence of cytoplasmic inclusions called nemaline rods in myocytes that results in generalized muscle weakness.

Case Presentation:
This report is an unusual presentation of a 51-year-old female with symptoms of increasing dyspnea and lower extremity edema for a year. Her medical history included concentric left ventricular hypertrophy, severe restrictive cardiomyopathy, and pulmonary hypertension. Microscopic examination of an endomyocardial biopsy revealed individual cardiomyocyte hypertrophy with non-specific degenerative changes. Electron microscopy revealed mild interstitial fibrosis and cardiomyocytes with multiple crystalloid inclusions synonymous with nemaline bodies seen in nemaline myopathy.

Discussion:
Nemaline rods associated with myopathy are typically seen only in skeletal muscle. However, a few cases of nemaline rods associated with cardiomyopathy have been reported, but most have been associated with dilated cardiomyopathy. This is a novel case of restrictive cardiomyopathy with nemaline body inclusions.

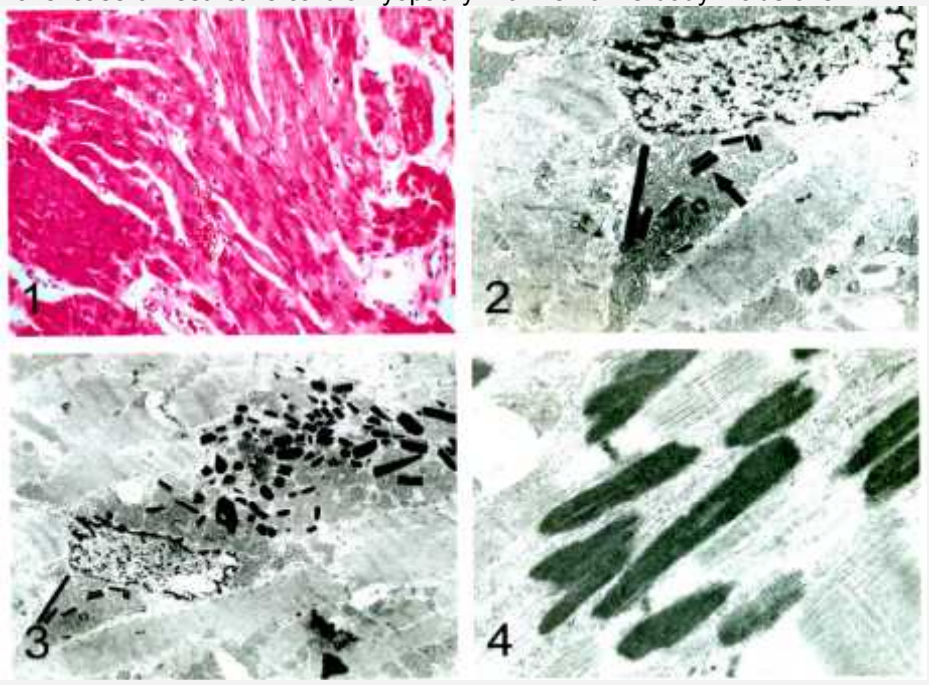

Illustration Legends
Figure 1. Endomyocardial biopsy showing hypertrophy of the individual myocytes and non-specific degenerative changes of the fibers consisting of rarefaction and slight disruption of the fibrillar cytoplasm. (lOX, H&E)
Figure 2. Ultrastructural appearance of rectangular Nemaline rods (arrow) located in the cytoplasm of a hypertrophic cardiac myocyte. (9600X)
Figure 3. Numerous Nemaline rods surrounding the nucleus and mitochondria of a cardiac myocyte. Most Nemaline rods are rectangular, however, in this example, the rods are quite variable in size and shape. (4800X)
Figure 4. Higher magnification of Nemaline rods demonstrating the striated lattice pattern very similar to that of normal Z discs in cardiac and skeletal muscle. (38400X)

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Title: (Do not enter author details)

Right Sided Pleural Effusion Causing Cardiac Tamponade in a 24-year-old Female

Jonathan Forquer[1], David Harris[1], [1]*University of Cincinnati, Cincinnati, Ohio, USA*

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

INTRODUCTION
Cardiac tamponade is characterized by hemodynamic compromise due to impaired venous return and cardiac dysfunction due to compression of the right atrium and right ventricle. The most common cause is pericardial effusion, resulting in increased pericardial pressure that exceeds intra-cardiac pressure and leads to cardiac chamber collapse. However, any external structure, mass or fluid collection that can cause cardiac chamber collapse can lead to cardiac tamponade physiology. Large pleural effusions have been previously documented to cause tamponade. In this case, the treatment of choice was thoracentesis to remove pleural fluid thus relieving extra-cardiac pressure and resolving the tamponade physiology.

CASE PRESENTATION
This is a 24 year-old female who presented with chest pain, dyspnea and abdominal fullness. She was found to have a large pleural effusion with right lower lobe and partial right middle lobe collapse. She subsequently was found to have multiple pulmonary emboli and DVTs. An abdominal CT on further work up found a 17 x 14 x 15 cm mass with septa and mural nodularity, as well as irregularly enhancing components. There was moderate ascites and underwent paracentesis with removal of 3L of fluid.
An echocardiogram was ordered in the evaluation of her dyspnea. It revealed a large effusion that was believed to be a loculated pericardial effusion with right atrial and right ventricular collapse, as well as a right ventricular cavity mass or thrombus.
When a physical exam was performed later that day, the patient was sitting comfortably in bed not requiring oxygen. She did not have a significant pulsus paradoxus and her jugular venous pressure was not elevated. She had decreased breath sounds in the right lower lung fields.
Repeat echocardiogram in the left lateral decubitus position again showed right sided chamber collapse due to an effusion with early tamponade physiology. (picture 1-5)
A repeat chest CT revealed no pericardial effusion, but a right pleural effusion.
Therapeutic thoracentesis and avoiding left lateral decubitus positioning were recommended. Her symptoms improved after the thoracentesis and she was started on heparin therapy and eventually underwent an MRI to further assess the RV thrombus. (picture 6)
Once stabilized, she went to the operating room for excision of her pelvic mass, which was noted to be mucinous adenocarcinoma.

DISCUSSION
Several case reports have shown large pleural effusions with minimal pericardial effusions causing clear echocardiographic signs of cardiac tamponade with right atrial and ventricular diastolic collapse and an impaired filling pattern. Animal models have shown that right ventricular diastolic collapse due to pleural effusion was inducible by infusing saline into the pleural space. Finally, several case reports and one larger case series describe right sided cardiac chamber collapse cause by large pleural effusions which was relieved by thoracentesis.

This case illustrates the importance of physical examination in the evaluation of a patient with echocardiographic evidence of cardiac tamponade physiology. When this patient was examined she had no signs of hemodynamic compromise or other physical signs of tamponade. Left lateral decubitus positioning for the echocardiographic exam caused the fluid in the right thorax to layer with gravity on right atrium and ventricle. Her signs and symptoms improved when she was able to lay flat or on her right side. Making the correct diagnosis allowed the patient receive the correct therapeutic intervention and avoid a potentially dangerous pericardiocentesis.

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Right Heart Failure secondary to Right Ventricular Undifferentiated Pleomorphic Sarcoma

Federico G. Trobo[2], Luciano Pastori[1], Deephak Swaminath[2], Gerald Pekler[1], [1]*New York Medical College, New York, New York, USA,* [2]*Summa Health, Akron, Ohio, USA*

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

Malignant primary tumors of the heart are very rare and in most cases are located in the left side of the heart. We report the case of a 57 year old Hispanic female, who presented in to the ambulatory medicine clinic in our hospital complaining of malaise and progressive bilateral pedal edema over the past seven weeks associated with puffiness and swelling offace and a recent weight gain of approximately 10 lbs. Her Exercise tolerance was 8-9 blocks limited by dyspnea but denied orthopnea and PND. Vital signs at presentation were: blood pressure 159/116 mmHg, heart rate 96 bpm, oxygen saturation 97% and temperature of 98.0 F.

The physical examination showed no jugular venous -distention or hepato- jugular reflux, but was notable for +2 pitting edema in lower extremities. Cardiac auscultation revealed normal S1 and S2,witha grade II/IV early systolic murmur with a decrescendo configuration murmur best heard at the tricuspid area, no S3 or S4, pulses were normal and symmetric bilateral. EKG –had a regular sinus rhythm -at a 91 bpm, QRS and P Axis 72 and 62 grades respectively, T wave inversions in V1-V2 and normal ST segments. Chest radiograph was unremarkable.

The echocardiogram revealed a severely dilated right atrium. A large mass with central clearing occupied most of the right ventricle, and obstructed RV inflow and protruded into the RV outflow tract. The presence of this mass was confirmed by cardiac computed tomography (CT) and magnetic resonance imaging (MRI).

The following day, the patient underwent open heart surgery. The procedure was done with no complications. A friable tan-white to red-brown specimen measuring 2x1.5x0.5x0.4 cm was excised and sent for pathology evaluation. Results of histopathology studies concluded that the mass was a high grade undifferentiated pleomorphic sarcoma.

Undifferentiated sarcomas are the most common subtypes in adults. They account for one-third of all cardiac sarcomas and are included in the pleomorphic sarcoma subgroup; including the historically defined malignant fibrous histiocytoma. Sarcomas have an ominous prognosis and data on patients with primary cardiac sarcomas have shown that median survival is 6 months from the time of diagnosis.

Due to the limited data, it is not clear the role of chemotherapy and radiation therapy in this group of patients. One of the mayor determinants of survival is the extent of resection given the high metastatic and recurrence rate. The estimate survival after surgical treatment is approximately 9 to 10 months.

One of the few clinical parameters available that have a good correlation with long term mortality is the presence of Class III and IV symptoms (NYHA).

To our knowledge, among the fifty cases that have been reported in the literature for primary Undifferentiated Pleomorphic Sarcoma of the heart, this is only the second case of right heart involvement.

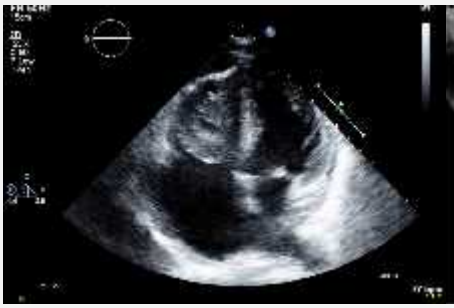


Figure 1. Echocardiogram

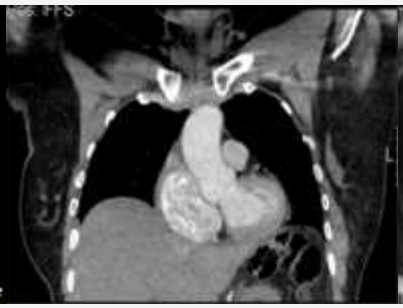


Figure 2 Chest CT

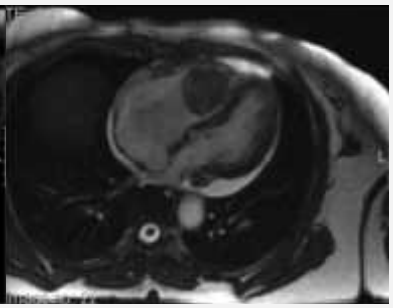
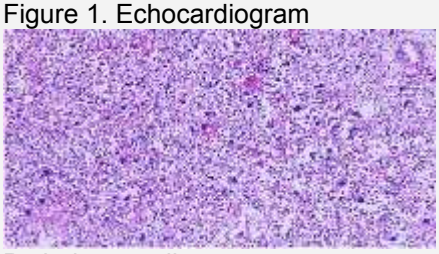


Figure 3 Cardiac MRI



Pathology outline

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Diastolic heart failure due to massive myocardial iron overload in a patient with sickle cell disease

Avirup Guha[1], Emily Ruden[1], Subha Raman[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)

Introduction: Sickle cell disease (SCD) produces considerable morbidity and mortality worldwide. Patients with SCD who receive frequent transfusions are at risk for cellular toxicity and often develop hepatic iron overload; however, myocardial iron overload is a rare phenomenon. Here we present an interesting case of myocardial iron overload in a patient with SCD presenting with clinical heart failure.

Case: Mr. S is a 28 year old male with a history of SCD (HbSS) receiving exchange transfusions every three weeks and with known cardiac iron overload who presented with dyspnea and lower extremity swelling one month after self-discontinuing deferasirox therapy. Physical examination revealed elevated JVP, bilateral pleural effusions, and pedal edema. Laboratory analysis was notable for a BNP of 411 and a ferritin of 4617. Echocardiogram showed low-normal left ventricular (LV) systolic function with a small pericardial effusion and bilateral pleural effusions. Cardiac MRI confirmed an ejection fraction (EF) of 50% with markedly shorted T2-star (T2*) relaxation times (myocardial T2* 4.5 ms [normal 52 +/- 16 ms]; hepatic T2* 3.5 ms [normal 33 +/- 7 ms), consistent with severe cardiac and hepatic iron overload. Calculated cardiac iron content was 9.8mg/g of cardiac tissue. He was diagnosed with acute heart failure with preserved EF (HFpEF) secondary to severe myocardial iron deposition. He was aggressively diuresed and discharged to follow up in our heart failure clinic.

Discussion: Cardiac MRI is uniquely positioned to assess for myocardial iron overload. Iron overload causes signal loss in affected tissues as iron deposits become magnetized, inducing local irregularities in the magnetic field and causing water protons around these deposits to lose phase coherence. This effect can be quantified using T2*-based imaging techniques, where decreasing T2* values are inversely related to iron concentrations. At myocardial T2* times below 20 ms, it has been shown that there is progressive decline in LV EF, increase in LV end-systolic volume index, and increase in LV mass index. Sickle cell cardiomyopathy is most commonly characterized by LV dilatation and diastolic dysfunction (18-57%). Despite frequent transfusions in this patient population, myocardial iron overload is a rare complication (prevalence 0.0-3.8%), whereas hepatic iron overload is frequently seen. In a small study comparing T2* times between 38 patients with SCD and 13 controls, there was no difference in mean myocardial T2* times between groups despite a statistically significant increase in LV systolic dysfunction and chamber dilatation in SCD patients. In fact, only one patient with SCD exhibited shortened T2* time consistent with cardiac iron overload despite a wide range of transfusion burdens. In contrast, 50% of SCD patients had shortened hepatic T2* times, whereas none of the control patients had abnormal hepatic T2* imaging. In a separate study of 26 SCD patients with increased hepatic iron concentrations, only 1 had abnormal T2* imaging. Interestingly, myocardial T2* times have not been shown to correlate with serum ferritin levels. Late-gadolinium enhancement has also been reported, predominantly in patients with HbSS SCD, in various patterns (non-infarction, focal, and localized) and in various distributions (inferior, anterolateral, apical, or lateral walls).

In contrast to patients with SCD, patients with beta-thalassemia major frequently develop transfusion-related myocardial iron overload, and heart failure remains the most common cause of death among this patient population. However, since 1999, there has been a dramatic improvement in survival, driven predominantly by T2*-guided identification of myocardial iron overload and appropriate intensification of iron chelation treatment. The reason for the disparity in myocardial iron deposition in patients with SCD and with beta thalassemia major despite similar transfusion burdens is unclear.

We present a rare case of clinical diastolic heart failure due to transfusion-related myocardial iron overload in a patient with SCD. Increased use of T2* imaging in this patient population may help to earlier identify patients with myocardial iron overload and thus influence the intensity of chelation-treatment, which could impact clinical outcomes as in the thalassemia population. Future studies in this area are indicated.

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Title: (Do not enter author details)

The Unrevealed Colossus: A Serious Case of Primary Aortic Mural Thrombus

Angela Igwe[1], Diego Alcivar[1], Roger Chaffee[1], [1]*Summa Akron City Hospital, Akron, Ohio, USA*

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

Objectives:
1. Describe the presentation, clinical evolution and diagnosis of Primary Aortic Mural Thrombus (PAMT)
2. Outline the importance of management strategies depending on the different types of PAMT

Case Presentation:
72yo Male with PMH significant for MVP, HTN, Hypothyroidism, GERD and previous Cholecystectomy who presents with 5 weeks of progressive epigastric pain, malaise, fatigue, unquantified fever, chills, nightsweats, anorexia, and 5 pound weight loss. He reports no change in his diet in his diet, habits or medications. His epigastric pain is dull, constant, 5/10, irradiated to the hypogastrium, with no known aggravating or relieving factors. VS: T99.5F, HR 100 RR 16 BP 150/80mmHg, O2Sat 94%. PE: Eyes: clear conjunctiva, no jaundiced, no JVD, CTAB, his heart sounds regular, normal, no murmurs or gallop; has mild epigastric tenderness, no rebound or guarding; no peripheral edema; he has no splinter hemorrhages on his fingernails or toenails. There are no embolic skin lesions or nodules. Labs WBC 14400, Hb 12.6, Hto 39.6, Plt 523000, Na 143, K 4.6, CO2 28, Crea0.88, BUN 22 AST 52 ALT 28 AlkPhos 121 Protein 6.2 Albumin 2.1 Bilirubin 0.6 Lactate 1.6 Lipase 263 Troponin 0.015 CRP 20 ESR 14 UA WNL. ECG NSR; CXR WNL; CT Abd/Pel shows splenic infarctions and multiple right lateral wedge shape renal hypodensities suggesting an embolic events. Patient was given IV normal saline, had an infectious disease work up. Transthoracic ECHO: EF >55%, trace valvular insufficiency in all four valves. Empiric Broad spectrum antibiotics was also started. Given unexplained subacute febrile illness characterized by splenic and possible renal infarcts; transesophageal echocardiogram was done to rule out subacute bacterial endocarditis which showed structurally and functionally normal mitral, aortic, tricuspid, and pulmonic valves, the aortic root and ascending aorta appeared normal but before finishing the study the descending aorta is filled with mobile densities, two attached to the wall of the aorta, longest 2cm in length and is 0.6cm in width; having the appearance of multiple thrombi. The aortic diameter is normal with mild linear or layered plaques, but no ulcerations or evidence of a dissection. The mobile densities did not involve the aortic arch. futher Infectious, Neoplastic, Hypercoagulable and Hematologic work up was ordered. Vascular surgery and Interventional Peripheral Vascular Cardiology were consulted. Infectious work up was negative in blood cultures and VDRL. Antibiotics were discontinued and anticoagulation with Enoxaparin and Coumadin was started. Symptoms improved and the patient was discharge 72 hours later with adequate follow up.

Discussion:
Primary aortic mural thrombus is defined as thrombus attached to the aortic wall in the absence of any atherosclerotic or aneurysmal disease in the aorta and a cardiac source of embolus. It could be either sessile or pedunculated. It is a relatively uncommon. It is Idiopathic in many patients. Prothrombotic conditions could cause aortic mural thrombus such as neoplasms, thrombocytosis, polycythemia, hypercoagulable states (60%) and primary tumors of the aorta. It usually affects younger patients with female predominance. Lower limb ischemia is the most common presentation, followed by visceral ischemia. Diagnosis is made by CT Aortography and by TEE. Because management strategies have largely been dependent on anatomic location as well as on morphologic features of the thrombus, PAMT is classified into 4 types. The distal arch and the descending thoracic aorta are the most common sites of thrombus (74%), followed by the abdominal aorta (14%) and the ascending aorta (12%). Most published reports of this entity are limited to case reports or series, there is no on appropriate management. Current management includes anticoagulation, systemic thrombolysis, and surgical removal of thrombus under cardiopulmonary bypass through median sternotomy. Alternatively, hybrid supra-aortic debranching and antegrade stent graft implantation can be done. Recurrent embolism risk is high (>25%) compared with open surgical removal of thrombus (9%), with risk of major amputation and life-threatening visceral ischemia. Minimally invasive options like catheter aspiration and systemic or catheter-directed thrombolysis with varying success rates, carries high risk of distal embolization during the procedure itself and do not promise complete removal or exclusion of thrombus. Surgical thrombectomy yields an aortic wall histopathologic diagnosis, which may be of diagnostic utility in this relatively unknown entity . Despite adequate removal of thrombus, perioperative mortality could be high and should be adequately consented. Also, in the setting of visceral ischemia and sepsis, use of endografts might have increased risk of graft infection. PAMT is a significant problem in young patients and has poor outcomes despite aggressive treatment. Advanced organ ischemia is a poor prognostic sign.

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Title: (Do not enter author details)

A Case for Multiple Coronary-Cameral Fistulas Causing Chest Pain

Omar Kahaly[1], Dilesh Patel[1], Konstantinos Boudoulas[1], [1]*Ohio State Medical 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)

Introduction

Maude Abbott published the first pathological account of a coronary-cameral fistula in 1908. The incidence of coronary fistulas is quite low and in one prospective study of 33,600 patients undergoing a left heart catheterization 34 (0.1%) had a coronary artery fistula. In our case we present a patient who came to the hospital with exertional chest pain and was found to have a coronary-cameral fistula.

Case Presentation

A 47 year old male with no significant past medical history was admitted to the hospital with fatigue and exertional chest pain. He initially underwent an exercise stress test; however, this test was inadequate as the patient was limited by fatigue. The TTE revealed an ejection fraction of 45-50%, biatrial enlargement and mild LV dilatation. To further work-up his chest pain a left heart catheterization was performed and revealed no coronary artery disease, but multiple fistulas were noted arising from his left coronary tree and draining into the left ventricle. During his admission he was also noted to be bradycardic down into the 30-40 bpm range. This was further worked up with an electrophysiology study which revealed sinus bradycardia in the setting of high vagal tone. The patient requested to be discharged and was sent out with an event monitor and close outpatient follow-up with cardiology.

Discussion

Patients with coronary-cameral fistulas may be symptomatic secondary to a coronary steal phenomenon occurring at sites distal to the coronary fistula; moreover, patients with a coronary-cameral fistula to the left ventricle may theoretically suffer from a physiology similar to those with aortic insufficiency. Therefore the conventional wisdom has been to close large coronary- cameral fistulas via a transcatheter or surgical approach; however, there is less guidance on how to proceed with smaller fistulas. We therefore searched the literature for medical treatment options for those who suffer from non-iatrogenic coronary to left ventricle fistulas that were either too small or too numerous to render themselves amenable to transcatheter or surgical closure. We were able to identify a total of 8 cases that met our search criteria. All of the patients presented with exertional chest pain and/or dyspnea. 5 of the 8 cases mentioned discharging the patient on a β-Blocker in order to reduce myocardial oxygen demand and provide symptomatic relief.

Conclusion

It is thought that patients with multiple small coronary-cameral fistulas have a favorable prognosis, but long term data is unknown. Our case represents a unique challenge as he was not a β-Blocker candidate given his pronounced bradycardia. Patients like this should be followed closely in the cardiovascular clinic given their structural heart disease and indeterminate trajectory.

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Title: (Do not enter author details)

ST-segment Elevation Myocardial Infarction in Ventricular Paced Rhythm- A Diagnosis not to be missed

SWARNALATHA KANNEGANTI[1], AJAY AGARWAL[2], VASKAR MUKERJI[2], [1]*Wright State University, Dayton, Ohio, USA,* [2]*Veterans Affairs Medical Center, Dayton, Ohio, USA*

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

INTRODUCTION: Diagnosis of ST-segment elevation myocardial infarction (STEMI) in the presence of a paced rhythm can be challenging as ST segment elevation may have no validity in a paced rhythm. Sgarbossa criteria are well recognized scoring system to increase the reliability of diagnosing STEMI in the presence of left bundle branch block (LBBB). But so far, we have no established criteria to diagnose STEMI in paced rhythms.

CASE REPORT: A 74-year-old man with history of second degree type 2 AV nodal block status post permanent pacemaker implantation in 2006, presented with shortness of breath from two weeks. No chest pain. EKG at presentation (Figure 1) showed 4-5mm acute ST segment elevation in anterior leads with underlying atrial sensed ventricular paced rhythm. EKG after turning off the pacemaker (Figure 2) showed 5-6mm ST segment elevation in anterior leads with underlying complete heart block. Subsequent emergent cardiac catheterization revealed multivessel coronary artery disease with chronic total occlusion of mid left anterior descending artery. Troponin I went up to 35.6. Viability study showed nonviable myocardium near the culprit vessel territory. So, patient was managed conservatively and was discharged with a life vest.

DISCUSSION: Clinical history and EKG findings alone should prompt immediate consideration of reperfusion therapy. However, abnormal baseline rhythms, such as left bundle branch block (LBBB) and ventricular paced rhythm (VPR), often conceal the EKG changes. During right ventricular pacing the ECG shows left bundle branch block and Sgarbossa rules may be used for the diagnosis of myocardial infarction during pacing, however they are less specific. The three Sgarbossa criteria, are:

1—Concordant ST elevation of 1 mm or more in any lead with a positive QRS complex

2—Concordant ST depression of 1 mm or more in V1–V3

3—Excessively discordant ST elevation (5 mm or more) in any lead with a negative QRS complex

The ASSENT 2 and 3 trials demonstrated that the third criterion (ST elevation of at least 5 mm discordant with QRS complex) had minimal diagnostic and prognostic use in patients with LBBB. However, other studies like GUSTO-1 trial have shown that this 3rd criterion has a high specificity and statistical significance when diagnosing STEMI in ventricular paced rhythm. So, patients presenting with chest pain and a ventricular paced rhythm who meet Sgarbossa's 3rd criterion should be considered to have a STEMI, and a cardiac catheterization lab should be activated by medical personnel of first contact.

CONCLUSION: This case demonstrates the utility of Sgarbossa criteria for the diagnosis of acute MI in paced rhythm. Criterion 3 is particularly applicable in ventricular paced rhythm. However, with advanced pacing modalities and multisite pacing, none of these criteria work and make the diagnosis further challenging. High index of suspicion and further studies are required in this regard.

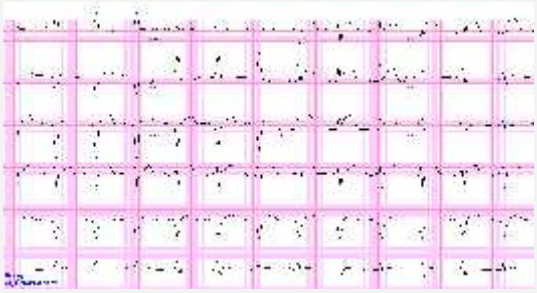

Figure 1: ST segment elevations in ventricular paced rhythm

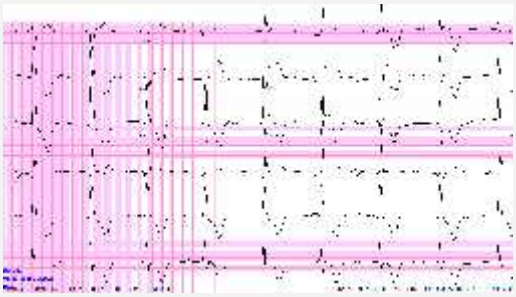

Figure 2: ST segment elevations after turning off pacemaker

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One complication and three anomalies; A case report
Ruptured noncoronary sinus of valsalva aneurysm, patent foramen ovale and atrial septal defect

Himad Khattak[1], Vijay Tivakaran[1], Arpan Patel[1], Muhanad Al-Zubaidi[1], [1]*Kettering Medical Center, Kettering, OH, USA*

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

Background: Sinus of valsalva aneurysm (SVA) is a rare cardiac anomaly with prevalence of 1% in patients undergoing open heart surgery [1]. About 65-85% originate from the right coronary sinus, 10-20% from the noncoronary sinus and less than 5% from the left coronary sinus [2]. SVA is often associated with ventricular septal defect, bicuspid aortic valve, and aortic insufficiency. It is less commonly associated with pulmonic stenosis, coarctation of the aorta, patent ductus arteriosus, atrial septal defect, subaortic stenosis, atrial septal aneurysm (ASA) and teratology of Fallot.
ASA has reported incidence of 2-10% and is often associated with atrial septal defect and patent foramen ovale (PFO) [3].
After review of literature, a combined anomaly of noncoronary sinus of valsalva aneurysm (SVA), atrial septal aneurysm, and PFO have not been documented in English literature.

Case Presentation: A 53 year-old Caucasian male with past medical history of hypertension, hyperlipidemia, and former tobacco abuse was referred to the VA Medical Center, Dayton, Ohio for symptoms of shortness of breath and abnormal stress test. His symptoms of shortness of breath were of 4 months duration. He denied any history of known coronary artery disease, congestive heart failure, or other cardiac conditions. He also denied any recent fevers, infection, or chest trauma. Symptoms had been progressive.
Physical examination was remarkable for grade 3/6 continuous murmur at 5th interspace to the left of the sternum. He had jugular venous distension and 2+ leg edema. Vitals signs were normal. EKG showed nonspecific ST segment. Laboratory blood showed normal hemoglobin level and renal function. Transthoracic echocardiogram revealed high velocity jet in the right atrium above the tricuspid valve concerning for sinus of valsalva aneurysm (SVA). Transesophageal echocardiogram confirmed noncoronary sinus of valsalva aneurysm with concern of rupture. Bubble study was consistent with PFO. Right heart catheterization revealed elevated RA pressures, and positive shunt study with step-up in the right atrium. Aortography demonstrated SVA rupture with a regurgitant jet into the RA. Coronary angiography revealed single vessel CAD involving the RCA. The patient was referred to tertiary care center for surgical evaluation. *Images, and Shunt study value (can be on a cardiac diagram/table)

Discussion: SVA is a dilation of the aortic wall between the aortic valve and the sinotubular junction. It is caused by the lack of continuity between the middle layer of the aortic wall and the aortic valve. This aneurysm is usually congenital, although cases associated with syphilis, infective endocarditis, trauma, atherosclerosis and aortic dissection have been reported. The most common complication of the ASV is rupture into the right ventricle (60%), RA (29%), LA (6%), left ventricle (4%) or pericardium (1%). If left without repair, the average survival rate is one year [4].
Both SVA and ASA are considered to be disease of the connective tissue. A similar inherent deficiency during embryonic development of the connective tissue could be the etiology of ruptured SVA and or ASA.

To the best of our knowledge, our case is the first documented case of ruptured noncoronary SVA, ASA, and PFO. *Images, and Shunt study value (can be on a cardiac diagram/table).

Conclusion: ASV is a rare cardiac anomaly with various associations. Ruptured ASV is a severe complication that requires time sensitive surgical repair.

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Simultaneous Diagnosis of Acute Aortic Dissection and Polycystic Kidney Disease

Jun Li[1], Amber Makani[1], Sahil Parikh[1], [1]*University Hospitals Case Medical Center, Cleveland, OH, USA*

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

Introduction/Objective: Autosomal dominant polycystic kidney disease (PKD) is one of the most commonly inherited genetic disorders, estimated to have an incidence of 1 in 400 to 1000 persons.[1, 2] It is due to germline mutations in two genes, *PKD1* and *PKD2*.[3, 4] Commonly described extrarenal manifestations include cystic disease of other intraabdominal organs and abdominal hernia.[2, 3] Cardiovascular manifestations include hypertension, left ventricular hypertrophy, valvular insufficiency, and intracranial aneurysms.[2] Aortopathies and coronary pathologies have been seldom reported in the literature.[5-9] Here we discuss a patient who presented with acute abdominal aortic dissection with a new diagnosis of PKD.

Case Presentation: A 43 year old African-American man with no previous medical history presented with sudden onset bilateral lower extremity numbness and pain, after feeling a popping sensation in his back while shifting positions in bed. On presentation to his local emergency department, he was noted to have a heart rate of 60 and blood pressure of 170/90. His exam was notable for diminished right lower extremity peripheral pulses. His initial laboratory findings was only significant for a mildly elevated creatinine of 1.76 mg/dL.

Given his clinical presentation, computed tomography angiogram was performed of the chest, abdomen, and pelvis with peripheral runoff. This revealed an acute aortic dissection extending from the origin of the left subclavian artery and terminating in the origin of the right common iliac artery (CIA), with near occlusion of right CIA as a result of the dissection flap. There was false lumen supply to the celiac, left renal, inferior mesenteric, and left common iliac arteries. Incidentally, innumerable cysts were noted within the bilateral kidneys and liver, consistent with PKD.

The patient was transferred to our tertiary care center for further management of his acute dissection. Medical management was pursued, initially with use of intravenous anti-hypertensives. After stabilization of his hemodynamics, he was transitioned to an oral regimen of lisinopril, carvedilol, and amlodipine. His creatinine stabilized at 1.37 mg/dL prior to his discharge. Subsequent follow up in cardiology and nephrology clinics showed well-controlled heart rate and blood pressure.

Discussion/Conclusion: There is a clinical association between PKD and aortopathies in certain patients.[8] However, the pathophysiology is not yet elucidated. *PKD1* and *PKD2* encode for polycystin 1 and 2, respectively. These molecules are both membrane proteins, with polycystin 1 involved in cell-to-cell or cell-matrix interactions, and polycystin 2 functioning as a calcium permeable channel.[10-12] These proteins have been previously isolated in the endothelium and vascular smooth muscle cells, although their exact function in the variable clinical expression of vasculopathies, including cerebral aneurysms, coronary aneurysms and dissections, and aortopathies, is poorly understood.[4, 13-15] Furthermore, the role of hypertension in this patient population should not be understated as a potential contributor to these vascular manifestations.

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Radiation Induced Valvular Heart Disease

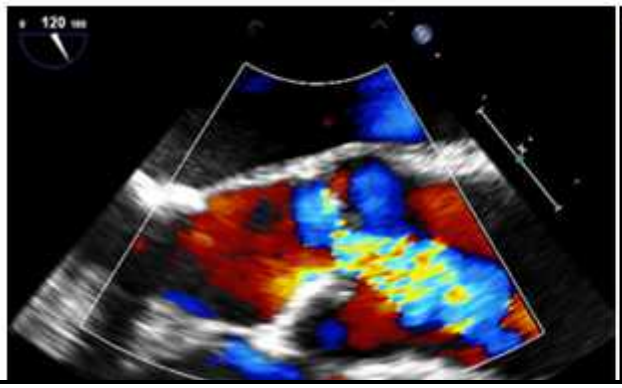
Ashish Mahajan[2], Ajay Agarwal[0], [1]*Dayton VA Medical Center, Dayton , OH, USA,* [2]*Kettering Medical Center, Dayton, OH, USA*

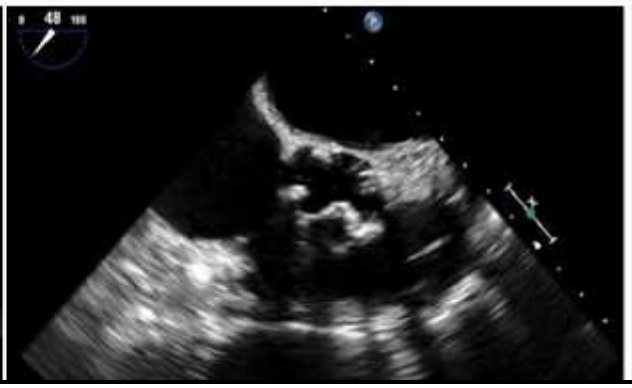
Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Introduction:
The history of radiation induced cardiovascular diseases dates back to 1897 when Seguy et al published the first clinical report on the effects of radiation on the heart . Radiation therapy has been a longstanding part of the therapeutic regimen for Hodgkin's lymphoma (HL). Despite recent advances in radiation therapy, patients treated decades ago have exposure to high doses of mediastinal radiation. Cardiac complications of mediastinal radiation including coronary artery disease, congestive heart failure, conduction system disease, pericarditis have been well documented, however radiation induced valvular heart disease have received relatively less attention. We present a patient who developed significant aortic and mitral valve disease secondary to radiation therapy for Hodgkin lymphoma.

Case:
Our patient is a 53 year old white female with history of Hodgkin's lymphoma, who was treated with radiation therapy to neck and mediastinum in 1980. Patient was first seen in our outpatient cardiology clinic with complaints of dyspnea with NYHA Class II symptoms and reduced exercise capacity. On physical exam, vital signs were within normal limits. Cardiovascular exam revealed 2/6 pan systolic murmur at the apex of the heart, without palpable thrill. The rest of physical exam did not reveal any significant abnormalities. EKG showed normal sinus rhythm without any significant abnormalities. Other diagnostic tests including complete blood count, basal metabolic panel, TSH were all within normal limits. A possibility of radiation induced coronary artery disease was considered. Patient underwent a Lexiscan stress test with myocardial perfusion imaging which did not show any evidence of fixed perfusion defect or reversible ischemia. To further evaluate patient's symptoms, a transthoracic echocardiogram was performed which showed normal right and left ventricular systolic function with ejection fraction of 60%, trileaflet aortic valve, calcified aortic valve leaflets with mild to moderate aortic stenosis and mild aortic regurgitation. Heavily calcified mitral valve leaflets with mild regurgitation was also noted. No mitral stenosis was seen. Patient was treated with ACE inhibitor due to history of concomitant hypertension. At follow up after 6 months, patient reported mild exertional dyspnea with no significant worsening of her symptoms. A follow up trans-esophageal echocardiogram 6 months after the transthoracic echocardiogram showed normal ejection fraction, calcified aortic valve leaflets with mild valvular aortic stenosis, along with moderate aortic and mitral regurgitation. Currently, patient is being treated with medical therapy with close follow up.

Discussion:
Valvular heart disease has been reported as a complication of radiation treatment for Hodgkin's lymphoma. Recent studies in HL survivors have reported that 32% of patients who received mediastinal irradiation develop asymptomatic valvular defects in 6 years, while at 20 years, 42% have imaging evidence of valvular dysfunction. The valve leaflets are fibrotic with focal calcification and marked thickening. The more common involvement of aortic and mitral valves is thought to be due to higher pressure blood flow occurring across these valves making them more susceptible to physical damage after initial radiation-induced injury. A significant proportion of patients with valvular heart disease do not exhibit symptoms of valvular dysfunction There should be high index of suspicion in patients who had received radiation therapy to the chest, especially those who received therapy before the advent of newer lower dose radiation therapy. A high index of suspicion along with imaging in patients presenting with unexplained symptoms may potentially aid in early diagnosis and treatment of radiation induced valvular heart disease.





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Emergent Left Main Coronary Intervention without Onsite Surgical Backup: Case report and review

Christopher Manhart[1], J. Bradley Gibson[0], *[1]Grandview Medical Center, Dayton, OH, USA, [2]Southview Medical Center, Dayton, OH, USA*

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

Introduction

Significant involvement of the left main coronary artery represents approximately 5% of patients with acute myocardial Infarction (AMI). It is associated with a high morbidity and mortality often complicated by cardiogenic shock and cardiac arrest. Many cases result in sudden cardiac death and never reach medical attention or the cardiac catheterization lab. PCI to the left main over the last several years has been increasing and a number of studies have shown the safety and efficacy of the procedure. In addition PCI at non-surgical back up sites has been increasing and a number of studies have demonstrated the feasibly and safety of PCI at those qualifying centers. The expansion of PCI capabilities to centers without surgical back up offers the ability to reduce morbidity and mortality to patients that may not otherwise have access to lifesaving procedures.

Case

A 64 year old female presents to the emergency department of a PCI center without surgical backup with the complaints of chest discomfort and back pain. Her EKG obtained in the ER revealed sinus tachycardia with a RBBB and LAFB. The ST segments in the anterior leads were concerning for ST elevation but interpretation was limited due to the RBBB. Unfortunately shortly after evaluation she quickly decompensated into cardiogenic shock and polymorphic VT arrest. ACLS protocol regained spontaneous circulation. A cardiac alert was initiated and she was taken to the cardiac cath lab for definitive diagnosis and treatment. Left coronary angiography was performed revealing a totally occluded left main. She underwent PTCA with a bare metal stent to the left main with great success. Final angiography was performed revealing TIMI 3 flow in the LAD and circumflex. An intra-aortic balloon pump was placed and the patient was transferred urgently to the large affiliated hospital for further cardiac critical care. The patient was evaluated by cardio-thoracic surgery but the percutaneous result was felt to be successful, eliminating the need for surgical revascularization. Unfortunately throughout the night the patient continued to deteriorate and eventually succumb to multi-organ failure and cardiogenic shock.

Discussion/Conclusion

Left main coronary artery involvement represents a small subset of acute myocardial infarctions and frequently present in cardiogenic shock or cardiac arrest. This subset of patients is at high risk for major cardiac adverse events. There is limited data on patients with AMI and UPLM as the culprit lesion. Those with cardiogenic shock and cardiac arrest represent and even smaller sample of patients only described in small cohorts. The above described case represents all the above high risk features including AMI with UPLM as the culprit lesion, cardiogenic shock and cardiac arrest. In addition the patient presented to a PCI capable hospital without surgical backup. This case scenario therefore represents and even smaller subset of patients adding to the importance and rarity of the case presentation.

Primary PCI has become the preferred reperfusion strategy for AMI. Primary and nonprimary PCI has been limited in a number of states to centers with surgical backup. As older and sicker patients present to our ER the complexity of CAD will mirror the patient population. The ability to offer the preferred reperfusion strategy will improve in and out of hospital outcomes. Strategic cooperation with local EMS services and implementation of the STEMI guidelines will continue to improve outcomes of patients presenting to non-PCI capable hospitals and sites without surgical backup.

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Four's a Crowd: A Rare Presentation of Peripartum Cardiomyopathy

Brad Martin[1], Prashanth Thakker[1], Jun Li[1], Anthony Deciccio[1], Chantel ElAmm[1], Brian Hoit[1], [1]*University Hospitals Case Medical Center, Cleveland, OH, USA*

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

Our patient is a 39 year old morbidly obese female, previously in good health, presented at 36 week gestation with acute hypoxic respiratory failure and pre-eclampsia. Her course was complicated by prolonged ventilator support in the setting of pulmonary edema. Her diagnostic workup included a transthoracic echocardiogram that showed moderate to severe aortic insufficiency but, due to technical limitations of the TTE, a transesophageal echocardiogram was obtained and was consistent with a quadricuspid aortic valve, severe aortic insufficiency, moderately dilated left ventricle, and an ejection fraction of 35%. Patient was medically optimized, extubated, and discharged. She is to follow up for a repeat TTE and consideration for aortic valve replacement versus repair should the severity of her aortic insufficiency and left ventricular dysfunction persists.

This case represents several unique challenges in presentation, diagnosis, and in follow up. Most cases of QAV are diagnosed ante mortem by echocardiography. Although alternative modalities such as cardiac magnetic resonance or gated cardiac computed tomography angiography have been utilized, valve structure and function is arguably best assessed by 2 and 3-dimensional TEE. The latest guidelines recommend that the degree of regurgitation and stenosis in QAV be assessed using the same parameters as in tricuspid aortic valves. Given the rarity of this congenital malformation, contemporary knowledge of QAV is obtained almost exclusively from autopsy or case reports.

The estimated prevalence of quadricuspid aortic valve (QAV) in the general population ranges from 0.008-0.033% based on historical autopsy data, and between 0.013-0.043% based on echocardiographic data from the late 1980s.[1] Although QAV has been described in association with other congenital abnormalities, including coronary anomalies, patent ductus arteriosus, ventricular septal defect, and other valvulopathies, it can occur in isolation. Valve dysfunction usually presents in older adults with aortic insufficiency. To date, there are no reports of QAV presenting in the peripartum period with hemodynamic compromise; as such, our patient presents a unique diagnostic challenge given her age and clinical scenario.

In addition to the rarity of her presentation and the initial diagnostic challenge, this patient also presents us with a challenge in regards to her management. Even though the patient was noted to have aortic insufficiency in the setting of a QAV, it should be highlighted that the patient was in the peripartum period which is associated with anemia, high intravascular volume, and elevated cardiac output which could exaggerate the findings of aortic insufficiency. In addition to these features, her hypoxic respiratory failure was in the setting of pre-eclampsia and resultant hypertensive crisis further clouding the picture as to whether this was an acute process related to her pregnancy that was complicated by a congenital abnormality or whether she had been suffering long standing moderate to severe aortic insufficiency that was exacerbated by the hemodynamic changes of her pregnancy.

The patient improved with medical management. Given the concern for potentially reversible causes of the cardiomyopathy and severity of the aortic insufficiency, the consensus was to continue medical management and reassess the patient in the outpatient setting to watch possible improvement in LV size, function, and degree of aortic insufficiency. The patient was consulted on the increased risk of future pregnancies and should there prove to be persistent cardiomyopathy with severe AI, surgical placement will need to be considered.

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Experiences using the Impella CP® percutaneous ventricular assist device for bridge to transplant in patients with refractory cardiogenic shock from chronic disease progression

James Monaco[1], Robert Cole[2], Divya Gupta[2], Anurag Sahu[2], Neal Dickert[2], Daniel Sims[2], Reza Fazel[2], *[1]Ohio State, Columbus, OH, USA, [2]Emory University, Atlanta, GA, USA*

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

Patients awaiting heart transplantation who develop refractory cardiogenic shock despite maximal medical support and the use of an intra-aortic balloon pump (IABP) present an extreme challenge if a donor organ is not available. The use of fully implantable, durable mechanical circulatory support (MCS) with continuous flow left ventricular assist devices (LVADs) has clearly been shown to improve survival as a bridge to transplant (BTT) in these patients(1, 2). However, there are patients who are ineligible for durable LVADs either for medical reasons or due to patient preferences. The options for management of these patients are exceedingly limited and their mortality is high. Herein we report our experiences attempting to bridge four patients with IABP/inotrope-refractory cardiogenic shock to transplant with the Impella® CP (3.5) percutaneous LVAD.

The Impella® line of devices are catheter-directed, microaxial pumps that can be inserted percutaneously or via surgical cut-down to an amenable artery. Prior members of the Impella product line include the Impella® 2.5 and Impella® 5.0, capable of providing up to 2.5 and 5.0 liters per minute of flow with a maximum device diameter of 12 French and 21 French, respectively. The Impella® CP (3.5) delivers up to 3.5 liters per minute of flow on a 14 French platform. As such, it may theoretically provide more adequate cardiac output than the 2.5 system, while avoiding complications of limb ischemia related to the size of the 5.0 pump. These devices are currently employed in patients with acute cardiogenic shock and those undergoing high-risk coronary interventions; however, they have not been extensively studied in patients with refractory cardiogenic shock from progression of chronic heart failure or in those awaiting transplant requiring prolonged hemodynamic support. We were unable to find any reports on the use of the Impella® CP (3.5) for this purpose.

All four of our patients were male with chronic, non-ischemic cardiomyopathy listed UNOS status 1A for heart transplant. All patients were receiving maximum tolerated doses of milrinone and dobutamine (and in some cases dopamine) as well as IABP support and experienced progressive decline after prolonged hospitalization. All demonstrated evidence of cardiogenic shock despite support, as evidenced by a Fick Cardiac Index (CI) less than 2.0 L/min/m_2 and systolic blood pressure (SBP) less than 90 mmHg. Three patients had worsening acute kidney injury (AKI) despite volume optimization. All underwent placement of the Impella® CP (3.5) LVAD.

All patients had demonstrably improved hemodynamics for several days after Impella® placement confirmed by invasive monitoring, and survived on therapy for 5 to 10 days. However, severe complications occurred in all patients. All patients experienced worsening kidney injury despite improved hemodynamics. Hemolysis occurred and was severe enough that all patients required blood transfusions, putting them at risk for pre-transplant sensitization. None of the patients had appreciable reductions in inotropic support. All four patients ultimately developed a progressive vasoplegic state in addition to their underlying cardiogenic shock and required initiation of vasopressors. While we had hoped that the device's smaller size relative to the Impella® 5.0 would simplify the process of device placement, only one patient was amenable to fully percutaneous delivery, despite all being free of peripheral vascular disease. Ultimately, only one patient was successfully bridged to transplant.

While it remains an interesting addition to the small stable of heroic measures available for bridging patients with refractory cardiogenic shock who are not candidates for durable MCS to transplant, severe complications are associated with prolonged support using the Impella® CP. More investigation and, ideally, direct trials against other available percutaneous LVADs are needed to better elucidate the ideal role of the Impella® CP in this population.

Title: **(Do not enter author details)**

Coronary artery to pulmonary artery fistula with concomitant atrial fibrillation

Mohammed Chowdhury[1], Sana Chikodi[1], Ankush Moza[1], Jacob Bieszczad[1], Ehab Eltahawy[1], [1]*University of Toledo Medical Center, Toledo, Ohio, USA*

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

Introduction: A coronary artery fistula is an abnormal communication between coronary vessels and cardiac chambers or a great vessel. This was first reported in 1865 by Krauss et al[1]. It is a rare cardiac anomaly, estimated to be present in 0.002 % of the general population[2] and constitutes < 15% of all congenital coronary artery anomalies. The majority of patients with such coronary abnormalities are asymptomatic and the anomalies are usually found incidentally during coronary angiography. They have an estimated incidence of < 1% in angiographic series.

Case presentation: A 53 year old otherwise healthy man with no previous medical history presented to the outpatient clinic for evaluation of new onset exertional dyspnea over several months. Enlargement. He was found to have atrial fibrillation and an echocardiogram revealed severely reduced ejection fraction. Coronary angiography showed no obstructive coronary artery disease but a fistula between the LAD and the pulmonary artery was detected. Right heart chatheterization for shunt fraction revealed a Qp/Qs ratio of 1. Therefore no closure of the fistula was done. Rate control of atrial fibrillation was pursued due to failre of rhythm control strategies. The left ventricular ejection fraction improvd with rate control. Serial monitoring of the fistula with repeat CT imaging is planned in one to two years.

Conclusion: The case described demonstrates that not all coronary artery fistulae require intervention or closure; the patient's functional status, symptoms and results of non-invasive and/or invasive testing should guide management.

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Title: (Do not enter author details)

A rare case of purulent pericarditis in a young immunocompetent male

Zubair Khan[1], Bhavana Siddegowda-Bangalore[1], Ghattas Alkhoury[1], Laura Murphy[1], Claudiu Georgescu[1], [1]*University of Toledo, Toledo, Ohio, USA*

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

Introduction: Bacterial Pericarditis is rare in the modern era of antibiotics. Mechanism of spread can be through direct inoculation during trauma, thoracic surgery and catheter drainage or through contiguous spread from intrathoracic, myocardial, sub diaphragmatic foci or hematogenous spread. Bacterial pericarditis is associated with a high mortality of 40% despite advancements in diagnosis and management. Nonspecific presentation often delays diagnosis contributing to increased mortality and morbidity. Most common cause of death in purulent pericarditis is cardiac tamponade. The most common pathogens isolated are staphylococcus and streptococcus species. We report a very rare case of primary purulent pericarditis due to streptococcus intermedius in an immunocompetent individual without predisposing factors

Case Report:

A 33 years old Caucasian male presented to the hospital with sudden onset pleuritic chest pain of one day duration, aggravated after lifting a heavy trash bag. It was associated with back pain, shortness of breath and body aches but no fever. An initial CT scan from outside hospital revealed small pericardial effusion and possible small left lower lobe infiltrate. MRI/MRA of chest was performed which ruled out an aortic dissection and any focus of infection. Echocardiogram showed an ejection fraction of 55% with small pericardial effusion. EKG showed sinus tachycardia and diffuse ST segment elevation typical of pericarditis. Patient was discharged home with a diagnosis of acute pericarditis and was prescribed indomethacin presuming a viral etiology. He was seen in the office 2 weeks later with worsening dyspnea and he was found to be in cardiac tamponade. An emergent pericardiocentesis drained 550 ml of purulent fluid. Pericardial fluid culture grew Streptococcus intermedius. No other source of the infection could be identified and all other cultures remained negative. He was started on IV ceftriaxone. His hospital course was complicated with development of pulmonary embolism and heparin induced thrombocytopenia, which were managed appropriately. During the subsequent hospital course, patient developed echocardiographic and hemodynamic features consistent with constrictive pericarditis for which a high risk median sternotomy and pericardiectomy was performed. Patient's condition stabilized after this and he was subsequently discharged home in a stable condition. Antibiotic therapy with IV ceftriaxone was continued as an outpatient, for total duration of 7 weeks with complete clinical resolution of his symptoms.

Discussion: Purulent pericarditis in a patient with no predisposing risk factors is extremely rare. Streptococcus intermedius is a member of streptococcus anginosus (milleri) group, which is normally found in the oral cavity and gastro-intestinal tract flora, however it can cause abscesses and systemic infections. There are very few cases of purulent pericarditis due to streptococcus intermedius reported in literature. To the best of our knowledge, this is the sixth case of streptococcus intermedius induced purulent pericarditis reported in English literature. Prompt recognition and treatment is of utmost importance given the rapidity of development of pericardial effusion and cardiac tamponade. In our case the pericardial effusion increased from minimal to large over a period of two weeks. With all the advancements in management and treatment, mortality rate continues to be very high, and the surviving patients usually have a prolonged hospital stay with increased morbidity. Despite pericardiocentesis and appropriate antibiotic therapy, constrictive pericarditis can develop. Some authors suggest prophylactic pericardiectomy in all patients with purulent pericarditis but this remains controversial.

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Title: (Do not enter author details)

Traumatic rupture of a bicuspid aortic valve following blunt chest trauma: A rare case report

Hemindermeet Singh[1], Sajid Ali[1], Justin Ugwu[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 Non-penetrating trauma to the chest wall can lead to injury of various cardiac structures. Rupture of the aortic valve without involvement of aortic root or descending thoracic aorta is a rare complication of blunt chest trauma and can be caused by tear or avulsion of the valve. Traumatic rupture of a bicuspid aortic valve is even rarer. We describe a case of traumatic rupture of a bicuspid aortic valve that presented with severe aortic insufficiency and required surgical replacement.

Case Report A 35-year-old male with medical history of bipolar disorder and schizophrenia who was admitted after jumping from a window height of 25 feet in the air sustaining multiple internal and external organ traumas. Transthoracic echocardiogram was significant for severe aortic insufficiency and a bicuspid aortic valve however it did not show pericardial effusion or myocardial contusion. CT angiogram of chest could not rule out aortic dissection so patient underwent an urgent aortic angiogram and study was negative. Cardiothoracic surgery evaluated the patient and planned for elective surgical valve replacement for aortic insufficiency however the patient remained unable to wean from ventilator. A repeat trans thoracic echocardiogram showed a 2 cm freely mobile suspected vegetation on the anterior aortic valve leaflet, which was confirmed on trans-esophageal echocardiogram. Although blood cultures were negative, the patient was referred for emergent aortic valve surgery because of suspected endocarditis as well as worsening heart failure in setting of severe aortic insufficiency. Intraoperative findings were significant for congenital bicuspid valve, however, a portion of the right coronary cusp was found to be sheared off from the annulus and was freely mobile. This visually gave the appearance of vegetation on echocardiogram however there was no evidence of endocarditis intraoperative. The valve was excised and replaced with a 29 mm bio prosthetic valve. Histology findings of aortic valve confirmed neither inflammation nor infection.

Conclusion/Discussion From cardiac standpoint, the incidence of blunt cardiac injury (BCI) is unknown. Of the BCIs', the most common finding is myocardial contusion. Other rare blunt cardiac injuries include but not limited to commotio cordis, myocardial rupture, septal and valvular injury and aortic dissection. Aortic insufficiency following trauma is a known valvular injury while traumatic rupture of bicuspid artic valve without involvement of thoracic aorta is very rare complication with only 3 such cases ever described. The treatment of choice is surgical valve replacement. Patients with bicuspid aortic valve could be structurally more susceptible to disruption from trauma as compared to normal tri-leaflet valve. There is no objective evidence regarding this association in literature so far. We recommend that this association should be further evaluated in future studies if additional cases become available.

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Title: (Do not enter author details)

Coronary steal syndrome from a giant RCA with coronary sinus fistula- a rare presentation

Jordan Thomas[1], Deephak Swaminath[1], Diego Alcivar Franco[1], Roshni Narayanan[1], William Bauman[1], Mark Iler[1], Marc Penn[1], [1]*Summa Health System, Akron, Ohio, USA*

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

Introduction:
We present the rare case of a patient with symptomatic coronary steal due to a giant right coronary artery with fistulous connection to the coronary sinus.

Case Presentation:
A 69-year-old female was referred to the cardiology clinic after a murmur was detected during a life insurance physical. She reported a three month history of mild substernal chest pressure with exertion, relieved by rest. She endorsed longstanding mild, chronic shortness of breath attributed to childhood asthma, but no orthopnea or other symptoms of heart failure. Past medical history was significant for lupus, psoriasis, and systemic hypertension but no cardiac illness. Electrocardiogram showed normal sinus rhythm and voltage criteria for LVH with no ST or T wave changes.

Echocardiogram showed normal left and right ventricular function. Several abnormal vascular structures were noted in the left inferior AV groove, adjacent to and communicating with the coronary sinus. Agitated saline contrast study from the left arm demonstrated no evidence of persistent left superior vena cava or right to left shunt. She had a dilated RV and severe pulmonary hypertension. Left heart catheterization showed no coronary stenosis and a markedly abnormal giant RCA with an apparent fistula to the coronary sinus.

Coronary CTA showed a giant, tortuous, dominant RCA with a caliber of approximately 10 mm throughout most of its course. Aneurysmal segments were present in the proximal RCA, superior right AV groove (2.4 cm) and distal RCA, inferior left AV groove (1.8 cm). The RCA then folded several times before draining into the coronary sinus. The coronary sinus was also dilated and aneurysmal, draining appropriately into the right atrium. The patient was treated with antihypertensive medication to decrease the afterload and she experienced a reduction in angina and is being followed expectantly.

Discussion:
Coronary artery anomalies are found in invasive coronary angiograms with a prevalence of 1.3%. Among all anomalies, coronary artery fistulae are particularly rare with a prevalence of approximately 0.2%. The right coronary artery or its branches is the site of the fistula in about 55% of cases. Most are incidental findings during angiography.

In case reports, giant coronary aneurysms with coronary fistulae in adults vary size from 20 to 150 mm. Most of the patients with giant aneurysm are female. Fistulae drain mostly into the LV or RV and rarely into the coronary sinus. For most patients, the aneurysm and fistula do not appear to cause symptoms. However, there are patients whose symptoms may be attributed to these coronary anomalies. Currently there are no guidelines for managing these patients. Surgical management is an option but is usually limited to patients with severe and debilitating symptoms. Often, when symptoms are mild or absent, no intervention is necessary and patients are followed clinically. If necessary, surgical treatment of coronary aneurysm may involve resection of the aneurysm, closure of the proximal and distal aneurysm with sutures, and closure with a woven Dacron patch. Another option is to stabilize the aneurysm within the thoracic cavity and leave it in place if there is evidence of sufficient collateral vessels. In another case report the aneurysmal inflow and outflow are mobilized and an end-to-end anastomosis performed instead of aneurysm ligation and coronary artery bypass. Interpositioned vein grafts can also been used to repair coronary artery aneurysms.

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Title: (Do not enter author details)

Subclavian stenosis contributing to myocardial ischemia via LIMA graft

Akira Wada[1], Ernest Mazzaferri[0], [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)

We present a case of a 67-year-old female who was referred to our medical center for left internal mammillary artery (LIMA) graft stenosis. Our patient had a past medical history of coronary artery disease (CAD) status post 2 vessel coronary artery bypass graft (CABG) performed in 2010, hypertension, hyperlipidemia, peripheral arterial disease, carotid stenosis, and tobacco abuse. The patient had been initially evaluated by her local cardiologist for chronic stable angina. A Lexiscan SPECT was obtained which showed no evidence of prior infarct or ischemia. She continued to have class II-III angina and a left heart catheterization was performed. The angiogram showed severe native 3 vessel CAD and 2 out of 2 patent grafts. The LIMA to mid left anterior descending (LAD) artery had a high grade proximal 75% stenosis. She was subsequently referred to cardiac surgery at the Ohio State University Wexner Medical Center for possible surgical revascularization. The patient was felt to be high risk for a redo bypass and a CT angiogram was obtained to evaluate her subclavian artery and LIMA graft. The CTA showed a high-grade (>70%) stenosis in the proximal left subclavian artery as well as ostial stenosis of the LIMA. The patient was then referred to interventional cardiology for possible percutaneous intervention. An adenosine stress cardiac MRI was first obtained to look for evidence of ischemia. There was no evidence of myocardial infarct scar or infiltrate on late gadolinium enhancement imaging, which was consistent with viable myocardium. With adenosine, perfusion imaging demonstrated a marked perfusion abnormality involving the entire septum, anterior wall and apex wrapping around to include the inferoapex. Given the finding of significant ischemia on the stress CMR, the patient proceeded with percutaneous intervention of the LIMA graft and the proximal subclavian stenosis. Percutaneous arterial access was obtained via the right femoral artery. A drug eluting stent was deployed across the ostial LIMA stenosis with good result. Next, the proximal subclavian stenosis was found to have a 25 mmHg gradient across the stenosis. An expandable covered stent was placed in the proximal subclavian with no gradient across the lesion, post stent.

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Title: (Do not enter author details)

Impella 2.5: time for a moratorium?

Rey Arcenas[1], Analkumar Parikh[1], Himad Khattak[1], Pargol Samani[1], Brian Schwartz[1], Harvey Hahn[1], [1]*Kettering Medical Center, Kettering, Ohio, USA*

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

Cardiogenic shock (CS) in a patient with acute myocardial infarction (AMI) portends a poor prognosis with mortality rate of 51-76% despite the use of Impella 2.5 for hemodynamic support. The Impella 2.5 improves cardiac output by 2.5 liters per minute above the baseline. In the current study, we describe our experience with the Impella device at Kettering Medical Center (KMC), a large community teaching hospital. Data from 25 consecutive patients who received the Impella at KMC from May 2011 to January 2014 was reviewed. Fourteen of these patients had CS due to AMI, of whom 10 received the Impella 2.5 and 4 received the Impella CP (cardiac power), a newer version that can augment the cardiac output by 4 liters per minute. The primary end-point was 30-day mortality. Independent predictors of 30-day mortality were evaluated including: the effect of the type of AMI (ST elevation myocardial infarction vs. non-ST elevation myocardial infarction), left ventricular ejection fraction (<30% vs. ≥30%), need for cardiopulmonary resuscitation, and timing of initiation of hemodynamic support (before or after cardiac catheterization). The overall mortality was 71% with all deaths occurring within five days of the Impella placement. Multivariate analysis did not show any significant difference in the 30-day mortality based on the independent predictors. In univariate analysis, mortality was three times higher in patients who received the Impella prior to cardiac catheterization compared with those who received it after. The real world 30-day mortality rate in patients with CS in the setting of AMI remains high despite use of Impella 2.5. This taken together with previously published data calls into question the utility of Impella 2.5 device in the setting of CS due to acute MI.

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Title: (Do not enter author details)

Use of a Pressure Wire System in the Evaluation of Left Ventricular Outflow Obstructions

Omair Ali[1], Abdul Wase[1], Ronald Markert[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)

Background
The left ventricular outflow tract (LVOT) lies between the anterior cusp of the mitral valve and the ventricular septum. Outflow obstructions cause arterial impedance to ventricular performance. As obstruction continues to progress, the left ventricle will become less compliant, and left ventricular end-diastolic pressure can become elevated even though the ventricular size remains normal. Abnormal diastolic function contributes to symptom onset. Complications of cardiac dysrhythmias, endocarditis, calcium emboli and sudden cardiac death may occur. These obstructions may be at the aortic valve (valvular), proximal to the aortic valve (subvalvular), or distal to the aortic valve (supravalvular). The need to accurately define the obstruction and assess peak velocities and pressure gradients is paramount.

Non-invasive Echocardiography (E) employing the Bernoulli equation is widely used in aortic stenosis (AS) assessment.
At times, there can be overestimation of area such as mistaken identification of a jet, failure to consider increased subvalvular velocities, non-representative jet selection, or misestimating during downstream pressure recovery. Such interobserver bias has led to the addition of Doppler during estimation to calculate valve area by the continuity principle. The valve area can be overestimated when there is elevation in transvalvular flow or from physiologic changes in transaortic flow rate. Planimetry of valve area has additional limitations. For example, measurement accuracy is limited when valve calcification is present and corresponds to the anatomic, not physiologic, valve area.

In cardiac catheterization, the cardiac output and pressure gradient between the left ventricle and aorta are simultaneously measured. The dual catheter method can cause more patient discomfort and entail more risk. Moreover, with peripheral vascular disease it is difficult to determine in whom a second access is and is not desirable. Technically, the catheter can induce changes in arterial pressure, decrease area of critical AS by its diameter, and possibly cause aortic regurgitation. The introduction of the single puncture technique has resulted in improvements. However, disadvantages include artifacts caused by catheter oscillation effects and the impact of patient HR on readings. Importantly, catheter entrapment can occur with either of the aforementioned techniques.

Objective
The justification for aortic valve/subaortic surgery is currently based on Doppler/catheterization data, which, as mentioned above, are subject to bias and errors. Our objective was to compare the agreement, procedure time, safety, and reclassification of a pressure wire (PW) system to the standard non-invasive method (E) and invasive methods (PB).

Methods
Eight patients (mean age 73.1 years, including 5 females and 3 males) with LVOT obstruction on Echocardiogram (E) underwent hemodynamic catheterization. Access included 3 radial and 5 femoral techniques. The catheters used for pullback method (PB) included pigtails, JR4, multipurpose, and Langston catheters. For aortic stenosis (4 cases), the transvalvular pressure gradient was obtained from simultaneous pressure recordings (using a pressure wire to measure left ventricular pressure and a diagnostic catheter to measure ascending aortic pressure). For subaortic cases (3 hypertrophic cardiomyopathy and 1 subaortic membrane) the transobstruction pressure gradient was obtained from simultaneous pressure recordings (using a pressure wire to measure LV pressure proximal to obstruction and a diagnostic catheter to measure LV pressure proximal to obstruction).

Results
Agreement
For aortic stenosis cases (n =4), PW aortic valve area (AVA) and E AVA showed limited agreement (intraclass correlation [ICC] = 0.67; 95%CI = -4.06 to 0.98), but PW AVA and PB AVA agreed strongly (ICC=0.99; 95%CI = 0.87 to 1.00). For all patients (n=8), PW mean gradient and E mean gradient showed limited agreement (ICC = 0.45; 95%CI = -1.75 to 0.89), but PW mean gradient and PB mean gradient agreed strongly (ICC=0.93; 95%CI = 0.66 to 0.99). For all patients (n=8), PW peak gradient and E peak gradient showed limited agreement (ICC = 0.27; 95%CI = -2.65 to 0.85), but PW peak gradient and PB peak gradient agreed strongly (ICC=0.98; 95%CI = 0.90 to 1.00).

Procedure time
The mean procedure time for the eight cases was 134 seconds for PW and 145 seconds for PB (p=0.011).

Safety
There were no complications in utilizing E, PB, or PW assessment of LVOT obstruction.

Reclassification
Utilizing PW after Echocardiogram led to reclassification of obstruction in 5/8 patients (62.5%) and utilizing PW after PB led to reclassification in 0/8 patients (0%).

Conclusion
In eight patients with LVOT obstruction a pressure wire system yielded assessments of aortic valve areas and mean and peak gradients that agreed closely with the pullback method. The procedure time was modestly shorter for PW vs. PB, and there were no complications with either method. No patients were reclassified when PW followed PB. Our findings are preliminary in that we continue to enroll eligible subjects with a goal of 25 patients with LVOT obstruction.

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Title: (Do not enter author details)

Coronary bypass graft perforation during percutaneous intervention

Mohammed Andaleeb Chowdhury[1], Mujeeb Abdul Sheikh[0], [1]*University of Toledo, Toledo, Ohio, USA*

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

Introduction

Coronary artery bypass graft surgery (CABG) is the most common cardiac surgical procedure. Saphenous venous grafts (SVG) are prone to atherosclerosis and eventually result in graft stenosis with time. As a result, increasing numbers of graft stenosis are being encountered in clinical practices which are preferably treated with percutaneous intervention (PCI) to avoid risks of redo surgery. However, due to the friable nature of the ageing graft there is always a risk for graft perforation which is a rare but life threatening complication of PCI. Literature regarding predictive factors of perforation and the success rate of recommended strategies are limited. In our review we attempt to identify factors contributing to graft perforations and also compare the efficacy and outcomes of commonly reported management strategies.

Method

We have reviewed all cases of graft perforation reported in Pubmed/Medline from 1987 to 2013 and collected information on patient demographics, presenting symptoms, graft age, perforation classification, nature of the intervention resulting in perforation, stents or balloon used for treatment and the short term outcome.

Results

32 cases of graft perforation were reported from 1987 to 2013. Patients with graft stenosis who underwent PCI commonly presented with unstable angina (31%, N=8 out of 26). Average age of presentation was 71yrs ± 9.7yrs with a male: female ratio of 3:1.

The average time from CABG to the time patient develops symptomatic graft stenosis was 14yrs ± 7.8yrs for SVG and around 4 days for LIMA grafts. Commonly reported perforated vessels were grafts to right coronary artery (35%, N=7 out of 20) and left anterior descending artery (25%, N=5 out of 20) with more than 80% stenosis (83%, N=15 out of 18) located in the mid-portion of the graft (61%, N=11 out of 18).

Average stent diameter of 3 ± 0.8mm, average balloon pressure of 14 ± 3 atm and 3 or more balloon inflations commonly resulted in graft perforation. Perforations were most commonly seen after stent placement (28%) or after repeated balloon pre-dilation followed by stent placement (25%).

74% of cases reported Class III perforation. Stent implantation (95% of cases) was significantly better in controlling acute bleeds after graft perforation than prolonged balloon inflation (3% of cases) (p=0.0001). Majority of cases reported using covered stents (76%, N=16), mainly Jostent.(Jomed GmbH, Rangendingen, Germany), 14% (N=3) used regular stents and 10% (N=2) reported mounted stents. Average stent diameter of 3.9±0.7mm, average stent length of 18.5 ± 6mm and the average deployment pressure of 14±2 atm were reported to be effective in controlling the bleed.

16% (N=5) of the cases were managed conservatively and only 6% (N=2) of cases were taken for surgery. 38% of cases reported hemodynamic compromise (N=12) including 5 cases of cardiac tamponade and two deaths. Average discharge time after perforation was 8 ± 4 days.

Conclusion

Graft perforation is a life threatening complication of PCI. According to our review; a high risk graft lesion could be defined as an old graft (14yrs ± 7.8yrs) with more than 80% stenosis in its mid-portion. Great caution must be exercised during PCI of such lesions especially during stent placement or balloon pre-dilation. Moreover, when dealing with high risk graft lesions the operator should try to avoid stents larger than 3 ± 0.8mm, balloon pressure more than 14 ± 3 atm and 3 or more balloon inflations. In case of perforation, implantation of a covered stent with an average size of 3.9±0.7mm x 18.5 ± 6mm at a pressure of 14±2 atm can be attempted only if the patient remains hemodynamically stable. In conclusion, graft perforation can be treated via percutaneous intervention with good immediate results, short term outcome and minimal peri-procedural risks.

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Prognostic Implications of Exercise-Induced Premature Ventricular Complexes in Asymptomatic Subjects: A Meta-analysis

Joonseok Kim[1], David Harris[1], Myron Gerson[1], Seung-Won Oh[2], Jinsoo Chang[0], *[1]University of Cincinnati Medical Center, Cincinnati, OH, USA, [2]Seoul National University Hospital, Seoul, Republic of Korea, [3]Michigan State university, East Lansing, MI, USA*

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

Introduction: Premature ventricular complexes (PVCs) during stress testing in patients without known heart disease is commonly seen in clinical practice, but its prognostic value is not well understood.
Methods: A comprehensive literature search of Medline, Embase, and the Cochrane Library between January 1970 and May 2015 was conducted. Observational cohort studies on adults without clinically diagnosed heart disease evaluating an association of exercise-induced PVCs and all-cause mortality and cardiovascular mortality were included in the analysis. Two independent authors performed data extraction and quality assessment, and a third author resolved discrepancies.
Results: Nine studies comprising 62,488 participants comparing clinical outcomes of patients with and without exercise-induced PVCs were included. The overall combined relative risks for exercise-induced PVCs were 1.43 (95% confidence interval [CI]: 1.28 to 1.6) for all-cause mortality and 1.89 (95% CI: 1.46 to 2.45) for cardiovascular mortality. In subgroup analysis, both frequent PVCs (RR, 1.47; 95% CI, 1.29–1.67) and infrequent PVCs (RR, 1.57; 95% CI, 1.13–2.18) were associated with adverse outcome. PVCs during recovery was associated with increased risk of death (RR, 1.55; 95% CI, 1.22–1.96). PVCs during exercise did not achieve statistical significance (RR, 1.51; 95% CI, 0.93–2.46), but only a few studies were included in the analysis. There was no evidence of publication bias.
Conclusion: This meta-analysis of retrospective cohort studies suggests that exercise-induced PVCs significantly increase the risk of total mortality and cardiovascular mortality. Although most studies tried to include only patients with no apparent heart disease, only a handful assessed the presence of underlying heart disease by echocardiogram or coronary angiogram. Our study calls for further studies to assess the prognostic significance of exercise-induced PVCs and utility of efforts to decrease PVC burden in order to improve the clinical outcome.

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A Prospective Comparison of Arterial Stiffness and Inflammation in Adults with HIV/Hepatitis C Co-Infection, HIV Infection and Healthy Controls.

Sonika Malik[1], Melissa Osborn[1], Brian Ferrari[2], Amy Graham[2], Sanjay Gandhi[1], Grace McComsey[3], Corrilynn Hileman[1], *[1]Metrohealth Medical Center, Cleveland, Ohio, USA, [2]Case Western Reserve University, Cleveland, Ohio, USA, [3]University Hospitals Case Medical Center, Cleveland, Ohio, USA*

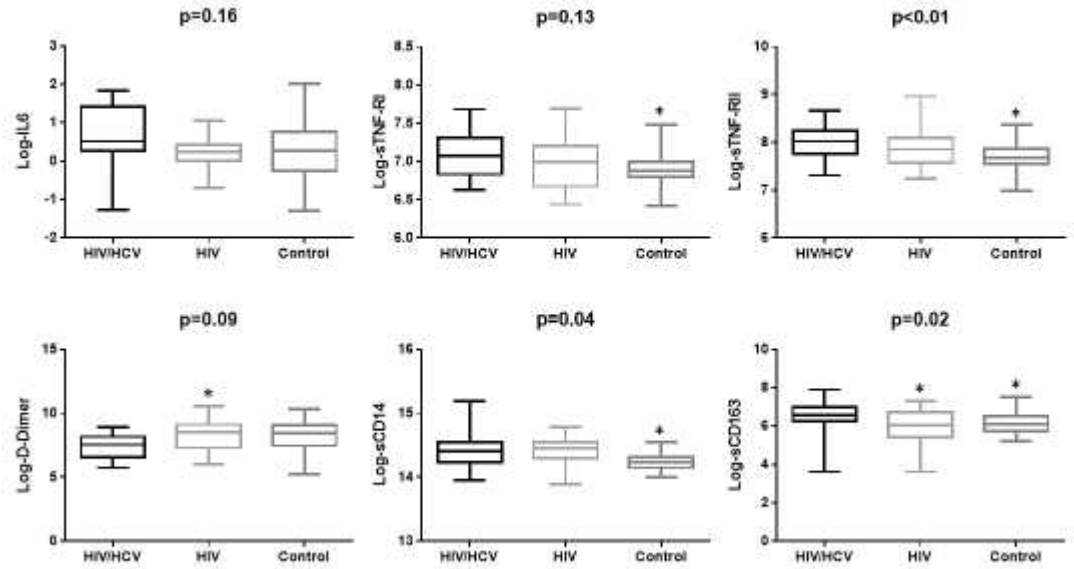
Abstract: **(Your abstract must use Normal style and must fit into the box. Do not enter author details)**

Background: HIV leads to accelerated atherosclerosis and HCV co-infection further increases the risk of cardiovascular disease (CVD) events. Although HCV improves lipoprotein levels, the effect of increased systemic/vascular inflammation and higher prevalence of insulin resistance may heighten CVD risk in this population. This is presently unknown.

Methods: This is a prospective, matched cross-sectional study to compare arterial stiffness measured by aortic pulse wave velocity (PWV), soluble markers of inflammation and monocyte activation, fasting glucose and lipoprotein levels between adults with HIV/HCV co-infection, HIV only and healthy controls. Adults with HIV-1 RNA <400 copies/ml on stable antiretroviral therapy (ART) (HIV groups) without CVD or diabetes (all groups) were included. Matching criteria were age, sex and CD4+ count (HIV only). ANOVA was used to compare mean PWV, biomarkers, glucose and lipoprotein levels.

Results: 25 HIV/HCV, 23 HIV and 23 controls were included. Demographics were similar except there were more African Americans (48% in HIV/HCV, 57% in HIV, 17% in controls; p<0.01) and smokers (64% in HIV/HCV, 45% in HIV, 22% in controls; p=0.02) in the HIV groups; history of alcohol abuse was more common (40% vs 13% in HIV and 4% in controls; p<0.01); and hypertension (HTN) was less common (4% vs 36% in HIV and 26% in controls; p=0.02) in HIV/HCV. Overall, 70% were men. Median age was 53 years and BMI 26 kg/m2. In the HIV groups, median HIV duration was 13 years; current and nadir CD4+ counts were 441 and 192 cells/mm3; 21% were on protease inhibitors and 92% had HIV-1 RNA <48cps/ml. PWVs were similar between groups [8.18 m/s for HIV/HCV, 7.81 m/s for HIV; p=0.4 for HIV/HCV vs HIV, and 7.95 m/s for controls; p=0.53 for HIV/HCV vs controls]. Results were similar adjusting for age, race, BMI, smoking, alcohol use and HTN. The figure shows the biomarker analyses. LDL was lower in HIV/HCV compared to the other groups, but oxidized LDL was similar. There was no difference in glucose between groups.

Conclusion: While arterial stiffness was similar between groups, markers of inflammation and monocyte activation were significantly higher among HIV/HCV participants suggesting a possible mechanism for the increased risk of CVD reported in this patient group.



P-values shown are from ANOVA and are testing the significance of the overall group effect after adjustment for smoking.
*denotes means that are statistically different (p<0.05) from the HIV/HCV group.

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Title: (Do not enter author details)

Coronary Microvascular Dysfunction: A Systematic Review of Treatments

Mark Marinescu[2], Adrián Löffler[1], Michelle Ouellette[1], Lavone Smith[1], Jamieson Bourque[1], *[1]University of Virginia Health System, Charlottesville, VA, USA, [2]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)

Background:
Angina without coronary artery disease (CAD) contributes to morbidity and is present in 10-30% of patients undergoing angiography. Fifty to 65% of these patients have coronary microvascular dysfunction (CMD). The optimal treatment of this cohort is undefined. We performed a systematic review to evaluate the effect of treatment for CMD in the absence of CAD.
Methods:
We performed a Medline search querying common terms used in describing angina without CAD. Articles were limited to English language reporting drugs or therapeutics in humans. Identified articles underwent single investigator review of title and abstract. Accepted articles were combined with articles from a bibliography search. These underwent review by two independent investigators based on inclusion (humans; coronary flow reserve (CFR) <2.5 by PET, CMR, dilution methods, or intracoronary Doppler; presence of angina) and exclusion (coronary artery stenosis ≥50%, structural heart disease) criteria.
Results:
A Medline search, last accessed in November 2014, identified 8,635 articles. Of these, 211 articles were identified for two-investigator review. Only 8 articles met strict inclusion criteria. These studies evaluated pravastatin, sildenafil, quinapril, intravenous L-arginine, intravenous diltiazem, doxazosin, estrogen, and transcutaneous electrical nerve stimulation application in the treatment of CMD. The articles were heterogenous using different primary end-points and different definitions for CMD.
Conclusion:
Our systematic review highlights that the data used to support therapies for CMD are inadequate. The heterogeneity in modes and protocols for defining coronary CMD make it challenging to compare treatment. CMD can now be identified noninvasively with high accuracy; however there is need for further refinement in techniques and definitions. We must take this opportunity to standardize protocols for the diagnosis of coronary CMD, allowing for future accurate population comparisons and advancements in therapy.

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Title: **(Do not enter author details)**

Increased Mortality with Elevated Plasma Endothelin-1 in Acute Heart Failure: An ASCEND-HF Biomarker Sub-study

Antonio Perez[1], Justin Grodin[1], Yuping Wu[2], Adrian Hernandez[3], Javed Butler[4], Marco Metra[5], G. Michael Felker[3], Adriaan Voors[6], John McMurray[7], Paul Armstrong[8], Randall Starling[1], Christopher O'Connor[3], W.H. Wilson Tang[1], *[1]Cleveland Clinic, Cleveland, OH, USA, [2]Cleveland State University, Cleveland, OH, USA, [3]Duke University Medical Center, Durham, NC, USA, [4]Stony Brook University, Stony Brook, NY, USA, [5]University of Brescia, Brescia, Italy, [6]University Medical Center Groningen, Groningen, The Netherlands, [7]University of Glasgow, Glasgow, UK, [8]University of Alberta, Edmonton, Canada*

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

Background: Endothelin-1 (ET-1) is an endogenous vasoconstrictor implicated in pulmonary and systemic hypertension, as well as ventricular dysfunction, through effects on vascular smooth muscle, the kidneys, and cardiomyocytes.

Objective: We aimed to determine the association between serial ET-1 levels and acute heart failure patient outcomes.

Methods: We measured plasma ET-1 at baseline, 48–72 hours, and 30 days in a cohort of 872 patients hospitalized with acute heart failure from the ASCEND-HF trial (randomized to nesiritide vs. placebo), and its association with 30-day mortality, 180-day mortality, in-hospital death or worsening heart failure; and 30-day mortality or rehospitalization.

Results: Median ET-1 was 7.6 (IQR 5.9–10) pg/mL at baseline, 6.3 (IQR 4.9–8.1) pg/mL at 48–72h, and 5.9 (IQR 4.7–7.9) pg/mL at 30 days ($p<0.001$). Baseline and 48–72 hour ET-1 were found to be independently associated with 180-day mortality in a multivariable analysis (HR=1.6, 95% CI 1.3–2.0, $p<0.001$ and HR=1.5, 95% CI 1.2–1.9, $p=0.001$, respectively, log-transformed). ET-1 that was measured at 48–72 hours was also independently associated with death or worsening heart failure prior to discharge (OR=1.6, 95% CI 1.03–2.4, $p=0.03$). These independent associations remained significant after including aminoterminal pro-B-type natriuretic peptide (NT-proBNP) and Troponin I in the multivariable analysis.

Conclusion: ET-1 is an independent predictor of mortality in hospitalized patients with acute heart failure, correlates with short-term in-hospital clinical outcomes, and yields prognostic information supplemental to that provided by NT-proBNP and Troponin I.

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Title: (Do not enter author details)

Heart and Heart-Liver Transplant in Congenital Heart Disease: Analysis of the UNOS Dataset

Krong-on Pinyoluksana[2], Yongjie Miao[2], Curt Daniels[1], Elisa Bradley[1], [1]*The Ohio State University, Columbus, OH, USA,* [2]*Nationwide Children's Hospital, Columbus, OH, USA*

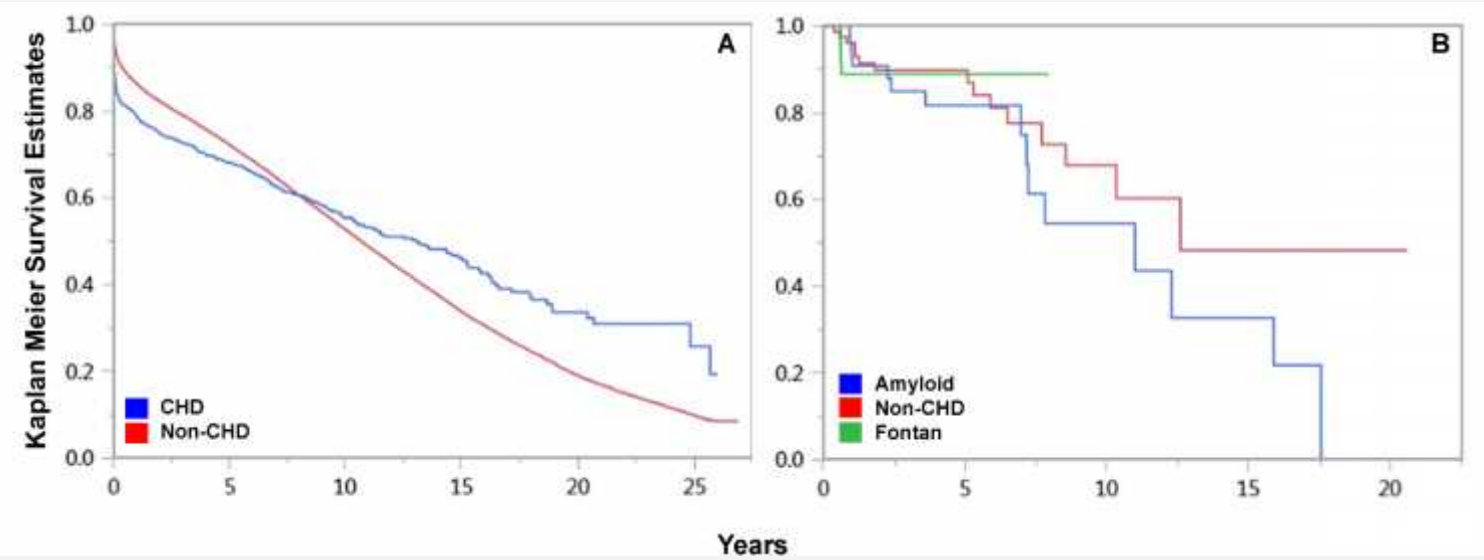
Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Background:
In congenital heart disease (CHD) adults now outnumber children. Moderate and severe CHD groups are increasing the most, including those with single ventricle anatomy that resulted in a Fontan operation. The aging patient with severe CHD faces many challenges including heart failure, arrhythmia, and in the Fontan patient, liver disease. The goal of this study was to define isolated orthotopic heart transplant (OHT) and combined heart liver transplant (CHLT) outcomes in U.S. CHD patients, and in particular, to determine CHLT transplant outcomes in the Fontan group.

Methods:
The U.S. United Network for Organ Sharing (UNOS) thoracic and liver databases were queried for the most common cardiac diagnoses (i.e. dilated cardiomyopathy, restrictive cardiomyopathy, coronary disease) and congenital heart disease diagnoses from inception through December 2014. Fontan patients were identified as those on the CHLT waitlist with a diagnostic code of "congenital heart disease with prior surgery". Descriptive data and comparisons between non-CHD and CHD groups were evaluated in adults.

Results:
For isolated OHT, CHD patients comprised 3% of those listed (Non-CHD n=53,755 vs. CHD n=1599) and transplanted (Non-CHD n=34567 vs. CHD n=1020). In CHLT, CHD made up 28% of waitlist patients (Non-CHD n=210 vs. CHD n=58), and 26% of transplanted patients (Non-CHD n=108 vs. CHD n=27). Fontan patients represented the majority of CHD that received CHLT (n=23(85%)). In CHLT, Fontan patients had higher baseline INR (1.8±0.8 vs. 1.6±0.5, $p<0.01$) and lower MELD score (15±6 vs. 16±6, $p<0.05$) than Non-CHD patients. Long-term survival in CHD OHT was not different than Non-CHD, albeit higher initial mortality ($p=0.44$, Figure 1A). With data available 7 years post transplant in the CHLT Fontan population, there was no 10-year mortality difference in Non-CHD, Fontan, and amyloid patient survival ($p=0.33$, Figure 1B).

Conclusion:
Despite similar overall long-term survival, U.S. CHD patients comprise a minority of those on the waitlist and receiving OHT. Combined heart liver waitlist and transplant groups have a substantially higher proportion of CHD patients, and in particular, those with a Fontan. Fontan and other CHD patients undergoing CHLT have similar survival to the Non-CHD cohort. These findings should encourage consideration of transplant to treat end-stage heart and combined heart liver disease in CHD patients.



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Title: (Do not enter author details)

Tilt Table Testing to Diagnose Pseudosyncope in the Pediatric Population

JEFFREY ROBINSON[1], JILL SHIVAPOUR[1], CHRISTOPHER SNYDER[1], *[1]RAINBOW BABIES AND CHILDREN'S HOSPITAL, UNIVERSITY HOSPITALS, 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)**

Purpose: Pseudosyncope (PS) can be difficult to distinguish from true syncope. The purpose is to describe the diagnostic utility of HUTT to elicit the diagnosis of PS in the pediatric population.

Methods: A retrospective chart review from 11/12 to 7/15 of patients ≤ 23 yrs of age referred for 30-minute, 80-degree tilt with continuous monitoring of ECG and pulse ox. Blood pressure and heart rate were obtained supine, at 80-degree tilt, and q 1 minute. Symptoms were recorded and vital signs taken concurrently.

Results: There were 76 patients referred for HUTT [median age 16 yrs (5-23); 22 (29%) male]. Of the 30 patients with symptoms during HUTT, 21 (28%) exhibited vital sign changes consistent with vasovagal syncope, while 9 (12%) were diagnosed with PS [median age 16 yrs (15-21); 4 (44%) male]. The majority (46, 61%) had a negative HUTT, with 45 true negatives and 1 false negative (diagnosed with pseudosyncope by further history on follow-up). Pretest probability for PS was high if the patient had 1) failed appropriate management, 2) atypical episodes, 3) occurrence during exercise, or 4) prolonged episode duration. Due to suspicion, prior to HUTT the likelihood of an episode during the procedure was discussed with patient. PS episodes were classified as immediate (within 2 minutes of HUTT) in 3 pts and late onset in 6 pts (all of which required induction techniques prior to symptom onset). PS was verified by normal vital signs and disruptive maneuvers: patient's response to questions, hand clap, or sternal rub.

Conclusion: PS should not be treated as a diagnosis of exclusion; it should be considered in patients that have failed appropriate management or who exhibit atypical episodes of syncope. PS can be identified with a HUTT if specific prompting of patients is utilized. Disruptive maneuvers, hand clap, and sternal rub are excellent adjuncts to confirm diagnosis.

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Title: (Do not enter author details)

Multiple Multifocal Cardiac Papillary Fibroelastoma Involving Aortic And Mitral Valve.

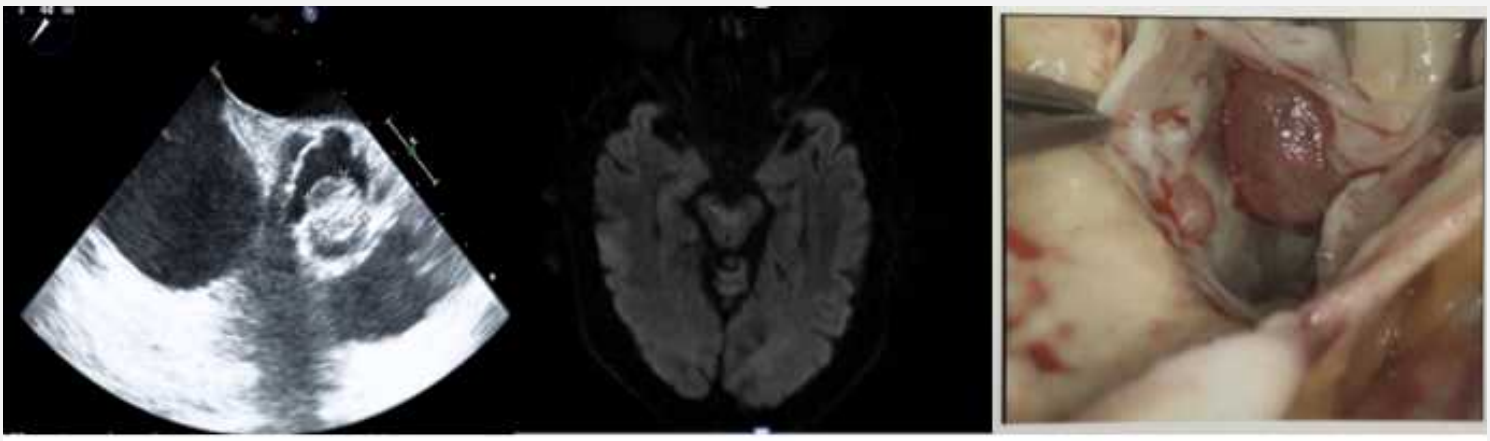
Pargol Samani[1], Himad Khattak[1], Peter M Pavlina[1], Reginald Sequiera[1], [1]*Kettering Medical Center, kettering, Ohio, USA*

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

Introduction:
Cardiac papillary fibroelastomas (CPF), the second most common primary cardiac tumors, are benign endocardial papilloma predominantly affecting the cardiac valves, and account for three fourths of all cardiac valve tumors.(1)These tumors have been reported to arise from aortic, mitral, tricuspid, and pulmonic valves in 35%, 25%, 17%, and 13% of cases, respectively. (2) The majority of patients with CPF are asymptomatic but the most common clinical presentations are embolism to the cerebral, systemic or coronary arterial circulations, followed by heart failure and sudden death. (1-3). These tumors are small, avascular masses, which, in 91% of cases, are single lesions. (4). Here we are presenting a rare case of multiple multifocal papillary fibroelasotma presenting with subacute stroke.

Case:
A 60 year-old man with past medical history of atrial fibrillation and coronary artery disease was referred to our institution after Transthoracic Echocardiography showed possible mass on the right coronary cusp of the aortic valve. Echocardiography was initially was ordered because the patient reported that he had left wrist drop about three weeks prior to the admission which was completely resolved over the next few days to week after the onset. The physical examination at the time of presentation did not reveal any noticeable abnormalities. Transesophageal echocardiogram (TEE) confirmed a 1x1 cm mass involving the ventricular side of the right coronary cusp of the aortic valve. (Fig.A). MRI of the brain showed subacute small infarct within the central midbrain region. (Fig.B). Because of the potential systemic embolic risk, the patient was referred for complete surgical removal. On operative inspection, there was a 1.5x1.2x 0.6 cm mass on the ventricular side of the right coronary cusp of the aortic valve and a 4 x 5 x4 mm mass on the ventricular side of the left coronary cusp, which was separated from the aortic valve with a fairly narrow stalk. (Fig.C). Also there were three distinct very small tumors attached to the posterior leaflet of the mitral valve. The pathology confirmed the diagnosis of papillary fibroelastoma for all the lesions. The patient's postoperative course was uneventful.

Discussion:
The patient had a completely normal 2D echocardiography one year prior to this presentation. To our knowledge this is a rare and unusual case of multiple fibroelastoma with rapid growth and involvement of multiple valves. The risk of embolic events associated with CPF indicates that two dimensional echocardiography should be performed in patients with transient ischemic attacks or strokes, even when they show no clinical or electrocardiographic evidence of heart disease (1-3). It is difficult to find multiple lesions involving aortic valve and mitral valve simultaneously in part because additional lesions may be missed on TEE, which occurred in this case. In many cases, more lesions are detected only upon precise surgical inspection. Therefore, the entire heart should be assessed as thoroughly as possible during surgery. (5-6). Smaller lesions may be attached beneath the leaflet surface, which are most likely represent different stages of the same tumor. (7).



A B C

A.2D echocardiography showed fibroelastoma on the right ventricular side of the aortic valve. B.MRI of the brain : signal hyperintensity within the central midbrain . C: Fibroelastoma of the right and left cups of the aortic valve during a surgical inspection.

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Title: (Do not enter author details)

No Difference in Efficacy of Clopidogrel with heparin versus Prasugrel with Bivalidrudin on Mortality, Bleeding, Target Lesion Revascularization or ST elevation Myocardial Infarction

Abrar Sayeed[1], Ehtesham Sayeed[1], Thomas Vacek[1], [1]*Wright State University, Dayton, USA*

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

In a study to determine the efficacy of clopidogrel with heparin versus Prasugrel with Bivalirudin, we determine that there was no difference in 30-day mortality or 1-year mortality when comparing the results of these two sets against each other (p=0.72 and p=0.24). Moreover, the two groups do not differ on TIMI (Thrombolysis in Myocardial Infarction) bleed at 30 days as well as TIMI bleed in 1 year (p=0.89 and p=0.46). Moreover, the two groups do not differ on Target Lesion Revascularization (TLR) at 30 days or at 1 year (p=0.24 and p=0.26 respectively). Using Fisher's exact test, it was also discovered the two groups do not differ on ST elevation myocardial infarction (p =0.490). The study included a total of 408 members with 200 in the heparin and clopidogrel group as well as 208 in the prasugrel and aspirin group. The groups were analyzed based on age, gender, comorbidities. The prasugrel and aspirin group did have more patients with Drug eluting stents than the heparin and clopidogrel group. The Heparin with clopidogrel group had more STEMI (ST Elevation Myocardial Infarction) patients. However, the two groups did not differ on gender, Diabetes Mellitus incidence, Hypertension, Hyperlipidemia, prior coronary artery disease, prior coronary artery bypass graft surgeries or chronic kidney disease

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Title: (Do not enter author details)

INCIDENCE OF SUPRAVENTRICULAR TACHYCARDIA IN PATIENTS WITH HEMATOLOGICAL MALIGNANCIES – RETROSPECTIVE OBSERVATION STUDY.

Deephak Swaminath[1], Roshni Narayanan[1], Jessica Kline[1], Federico Garcia Trobo[1], Ottorino Costantini[1],
[1]*SummaCardiovascular Institute, Akron, Ohio, USA*

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

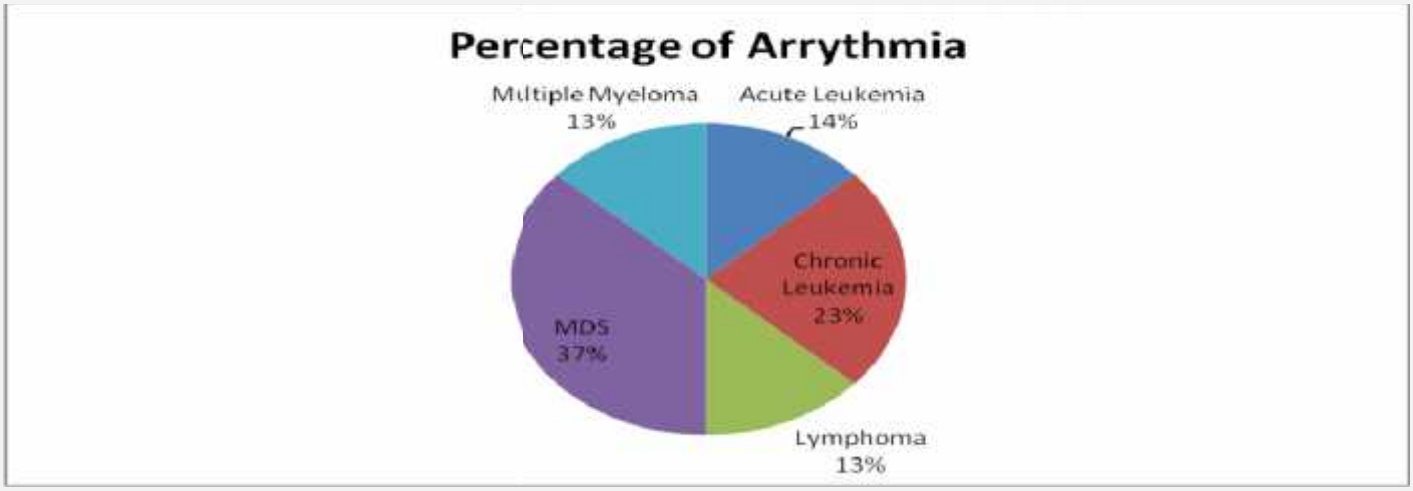
Purpose of the Study: Atrial Fibrillation (AF) is the most common cardiac arrhythmia encountered in clinical practice. Overall prevalence in the general population is estimated to be 0.4%. Patient with hematological malignancies are at increased risk of developing anemia as a result of the underlying disease as well as treatment. Our aim was to estimate the incidence of atrial tachyarrhythmias (including Atrial Fibrillation and flutter) in patients with hematological malignancies.

Methods:

We present a retrospective observational study on the incidence of new onset atrial tachyarrhythmias (AT) in patients with hematological malignancies including leukemia, lymphoma, multiple myeloma and myelodysplastic syndrome (MDS) at University Medical Center, Lubbock, Texas from Jan, 2008 to Dec, 2012. We collected baseline characteristics from the electronic medical record including age, sex, race and type of malignancy.

Results:

A total of 203 patients with the above mentioned hematological malignancies were included in the analysis, of which 41% were female. The average age of the patient was 65 and 81% of the patients were over the age of 60. The overall incidence of ATs in these patients was 13%. However, the highest incidence was 33% in MDS patients and we observed a higher incidence in Caucasians compared to Hispanics with 12% and 10% respectively.



Conclusions:

Our study revealed that the incidence of atrial tachyarrhythmias was higher in patients with hematological malignancy than in the general population and the vast majority was in the form of atrial fibrillation.The incidence is highest in patients with MDS, Caucasians and patients older than 60 years of age. These findings may be due to the higher prevalence of comorbidities in patients over the age of 60 and high sympathetic tone because of malignancy. Hence the patient with hematological malignancy, especially MDS, should be considered as high risk for supraventricular arrhythmias.

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Title: (Do not enter author details)

Left atrial function and its prognostic values in patients who have normal stresss echocardiography

Rayji Tsutsui[1], Kenya Kusunose[1], James Thomas[1], Zoran Popovic[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
The association between left atrial (LA) function and adverse outcomes in patients following an acute myocardial infarction is recognized.

Objective
The purpose of this study was to assess the prognostic value of LA function in patients who have exercise stress echocardiography negative for ischemia.

Methods
Our echocardiogram database was searched for subjects that underwent exercise stress echocardiogram for exclusion of coronary artery disease (CAD) between January to April 2010. Subjects were excluded if they were in atrial fibrillation/flutter at the time of exercise, left ventricular ejection fraction (LVEF) < 45%, moderate or severe valvular disease, echocardiographic evidence of exercise-induced myocardial ischemia, or had known coronary artery disease. Maximum metabolic equivalents (METs), LA total strain (analyzed offline with Siemens Syngo VVI), LV E/e', LA volume index (LAVI) were measured for all subjects. Diagnosis of major adverse cardiac events (MACE; myocardial infarction, coronary revascularization, cardiovascular mortality) and coronary artery disease (CAD; angina, unstable angina) were recorded. Data are reported as mean ± standard deviation (SD).

Results
672 subjects were identified and 517 patients remained after exclusion. 31 patients (6%) were excluded due to poor image quality, therefore, 486 subjects were ultimately included. The mean follow up time was 4.3 ± 0.1 years and the mean LVEF was 56 ± 5%. Following the index stress test, 25(5%) subjects had MACE and 35 (7%) subjects had CAD. Table 1 shows the mean and the HR for MACE. None of the echocardiographic parameters reached statistical significance. However, maximum METs was a strong prognostic factor, especially METs < 9 (median), which was strongly associated with MACE. For CAD, none of the variables tested reached statistical significance including METs.

Conclusions
In patients who have negative exercise stress test for ischemia, reduced METs predicted adverse outcomes. In this low risk population, none of the tested LA echocardiographic parameters appeared to predict adverse outcomes.

Major Adverse Cardiac Events

	Mean ± SD	Hazard ratio	P value
Age	56 ± 13	1.01 (0.98 – 1.04)	0.39
Sex (Male)	n = 306 (63%)	0.49 (0.21 – 1.11)	0.09
BMI	28.4 ± 52	0.95 (0.87 – 1.03)	0.20
Current smoker	n = 222 (46%)	1.51 (0.66 – 3.45)	0.33
LAVI	39.8 ± 15.0	0.98 (0.95 – 1.01)	0.31
LA total strain	30.6 ± 8.8	0.97 (0.92 – 1.02)	0.20
LV E/e' stress	10.7 ± 4.4	0.98 (0.91 – 1.06)	0.80
Max Mets	9.2 ± 2.5	0.74 (0.61 – 0.89)	0.002