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Timothy Byrnes, Roger Chaffee, Summa Health System, Akron, Ohio, USA
**Title: 0002 - CASE**

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

**Abstract.**

Left atrial thrombus can develop in patients with atrial fibrillation and/or a dilated left atrium such as seen in patients with heart failure. In cardiac transplant patients with bi-atrial anastomosis, the suture line can be a potential nidus for thrombus formation. These thrombi can be either organized or unstable with ulcerated surfaces. We present a unique case of a left atrial mass in a cardiac transplant patient with features of atrial myxoma on imaging studies but found to be an organized thrombus on histopathology.

**Key words:** Thrombus. Myxoma. Left atrium

**Case report.**

WM is a 71 year old male with a history of cardiac transplant 14 years ago who presented to the clinic for his annual testing. He was asymptomatic and taking his medications regularly. On physical exam, the only abnormal finding was tachycardia. He had routine blood work, an Allomap score, and 12 hour trough cyclosporine level. Prior to his visit, he underwent myocardial perfusion scanning and a transthoracic echocardiogram (TTE). A perfusion scan was normal. However, the TTE revealed a left atrial mass which was not present on the TTE done the year prior. This prompted us to have him undergo cardiac magnetic resonance imaging (Cardiac MRI). The impression on the cardiac MRI was a 2.2 x 1.6 x 0.8 cm well circumscribed pediculated mass in the left atrium most likely consistent with left atrial myxoma (Fig: 1). CT surgery was consulted. The patient had previously undergone three open heart surgeries. Thus, a right thoracotomy approach was planned for the removal of the mass. Peri-operatively, the patient underwent a transesophageal echocardiography (TEE) that clearly confirmed a pediculated mass attached to the left atrium (Fig: 2). He underwent successful removal of the mass without major complications. The specimen was submitted to surgical pathology, and a diagnosis of organizing thrombus was rendered (Fig: 3). The patient was discharged on oral anticoagulation with a follow up with hematology/Oncology.

**Discussion.**

Heart transplantation has been the treatment of choice for patients with end-stage heart failure. The first case of cardiac transplant was reported in 1967\(^1\). Most orthotopic heart transplants have been performed using bi-atrial anastomotic technique. This was first developed and described by Lower et al\(^2\). Atrial thrombus formation is a rare complication in transplanted recipients. Virchow’s triad could explain the pathophysiology of thrombus formation which includes abnormal blood flow and stasis, abnormalities in the surface endothelium and increased viscosity of the blood. A left atrial thrombus is usually of irregular appearance, has a broad base and layered body. These thrombi are either organized or unstable with ulcerated surface putting the patient at higher risk of having embolic events. A cardio embolic mechanism may exist in as few as 19% to as high as 75% of cases\(^6,7\). These thrombi typically form in a dilated atrium or in patients with atrial fibrillation both of which impair normal blood flow and promote stasis. There are case reports of atrial thrombi seen primarily along the graft to host suture line of the bi-atrial anastomosis.

Left atrial myxomas (LAMs) are among the most common cardiac masses\(^3\). LAMs were first described in 1845 by King as an unusual vascular growth in the left atrium. Cardiac myxomas are benign tumors but are associated with serious complications including embolization and mitral valve obstruction. Even with complete excision there is a recurrence rate of 3%. LAMs in post-transplant patients are very rare and often misdiagnosed as left atrial thrombus formation. In our case, the differential diagnosis included Atrial Myxoma and Atrial Thrombus. Differentiation between thrombus and masses in the atria remains challenging. Typically, an atrial myxoma has broad body and is attached to the atrial surface with a narrow stalk. Most are located in the left atrium and originate from the inter-atrial septum. TTE is the most common method of initial discovery. TEE is both sensitive and specific for the premorbid identification of left atrial masses and is often necessary for accurate delineation of its structure and site of attachment\(^4\). MRI has also been utilized for visualization of LAMs. As demonstrated by our case, thrombi can rarely show radiographic features characteristic of atrial myxoma.

**Conclusion:**

Left atrial thrombus formation in the cardiac transplant patient is rare but serious condition. Most of the time it can be diagnosed with imaging studies like TTE, TEE or cardiac MRI. We present a unique case of left atrial thrombus presenting as classic imaging features of atrial myxoma.
Title: Ventricular Arrhythmias and Mortality after Left Ventricular Assist Devices Implantation: A Meta-analysis

Nader Makki¹, Olurotimi Mesubi², Curtis Steyers², Brian Olshansky², ¹Ohio State University Hospital, Columbus, Ohio, USA, ²University of Iowa Hospitals and Clinics, Iowa City, Iowa, USA

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

Background
Ventricular arrhythmias (VAs) are among the most commonly reported adverse effects associated with left ventricular assist devices (LVADs). However, prevalence of VAs post-LVAD implantation, and their relation to all-cause mortality, remains to be elucidated.

Objective
We conducted a meta-analysis of observational studies with the primary objective of evaluating the risk of VAs after LVAD implantation and the risk of all-cause mortality in patients with LVADs who had VAs.

Methods
We searched Medline, Embase and Cochrane Central from 2001 to 2014. Two reviewers independently searched selected and assessed quality of included studies with differences resolved by consensus. Data were collected and analyzed using random and fixed-effect model, as appropriate, with inverse variance weighting.

Results
Of 2,393 studies identified, 15 observational studies were eligible including 1,517 patients with a mean follow up of 201 days. An LVAD was associated with an increased risk of VA after implantation (OR = 2.21, 95% confidence interval [CI] 1.37-3.59, p<0.001). There was an increased risk of all-cause mortality in LVAD patients who had post-LVAD VA (OR = 1.91, 95% CI 1.18-3.11, p<0.001). Using meta-regression and sensitivity analyses to account for risk factors such as etiology of cardiomyopathy, duration of follow-up, destination LVAD versus bridge therapy and presence of an implantable cardioverter defibrillator at time of LVAD implantation did not change the results of our main analysis.

Conclusions
LVADs are associated with an increased risk of VA and presence of VAs post LVAD implantation is associated with increased risk of all-cause mortality.
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**Abstract:** *(Your abstract must use Normal style and must fit into the box. Do not enter author details)*

**Background:**
The role of the routine use of vascular imaging including intravascular ultrasound (IVUS) and Optical Coherence Tomography (OCT) in guiding percutaneous coronary interventions (PCI) is still controversial especially when using drug-eluting stents. A meta-analysis of trials using bare metal stents was previously published.

**Methods:**
We conducted a meta-analysis of available published trials that compared imaging-guided PCI and angiography-guided PCI. We aimed to study both drug-eluting stents (DES) as well as bare metal stents (BMS). We identified 7 randomized controlled trials on IVUS-guided bare metal stents. We also identified 3 randomized controlled trials on IVUS-guided drug-eluting stents. To improve the power of the drug-eluting stent data, we identified, and included, 9 registries that compared IVUS-guided PCI to angiography guided-PCI in the drug-eluting stent era. Finally, we identified one registry that compared OCT-guided PCI to angiography-guided PCI using either a BMS or a DES. A total of 14,197 patients were studied overall. The meta-analysis was conducted using a random effect model.

**Results:**
Imaging guidance was associated with a significantly larger post-intervention minimal luminal diameter (SMD: 0.289. 95% CI: 0.213-0.365. P <0.01).

Imaging-guided stenting was associated with a significant decrease in the Major Adverse Cardiac Events (MACE) in the DES patients (Odds ratio: 0.810. 95% CI: 0.719-0.912. P< 0.01) and combined DES and BMS patients (Odds ratio: 0.782. 95% CI:0.686-0.890. P<0.01). Imaging guidance was associated with significantly lower events of death of all causes in DES patients (Odds ratio: 0.654. 95% CI: 0.468-0.916. P<0.01) and in the combined DES and BMS patients (Odds ratio: 0.727. 95% CI: 0.540-0.980. P<0.01).

The risk of Myocardial infarction (MI) was significantly lower with Imaging guidance in both, DES patients (Odds ratio: 0.551. 95% CI: 0.363-0.837. P<0.01) and combined DES and BMS patients (Odds ratio: 0.589. 95% CI: 0.425-0.816. P<0.01). This may, in part, be explained by the significantly lower risk of in-stent thrombosis in Imaging-guided DES patients (Odds ratio: 0.651. 95% CI: 0.499-0.850. P< 0.01) and combined DES and BMS patients (Odds ratio: 0.665. 95% CI: 0.513-0.862. P<0.01). Patients who received a DES showed no difference between Imaging guidance and angiography guidance in repeated target lesion revascularization, while the analysis of BMS alone and the DES and BMS combined showed significant superiority of the imaging-guided PCI group.

**Conclusion:**
Imaging-guided PCI significantly lowered the risk of death, MI, in-stent thrombosis and the combined MACE in DES implanted patients and all stented patients (DES or BMS). However, imaging guidance had no significant effect on repeated target vessel or target lesion revascularization in patients who received a DES likely due to the effect of the drug in the stent. The findings of this meta-analysis need to be confirmed in larger randomized controlled trials.
Title: 0007 - Research

Segmental strain and post-systolic shortening in right ventricles of children with hypoplastic left heart syndrome during three stages of repair.
Roshan D'Souza¹, Anirban Banerjee², Saurabh Patel³, ¹Rainbow Babies and Children's Hospital, Cleveland, Ohio, USA, ²Children's Hospital of Philadelphia, Philadelphia, PA, USA, ³St Peter's Hospital, New Jersey, NJ, USA

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

Introduction
The assessment of right ventricular (RV) systolic function by echocardiography, is primarily subjective. In hypoplastic left heart syndrome (HLHS), monitoring RV function becomes more important for evaluation of clinical status and outcome. Myocardial strain is a novel, dimensionless, technique used for assessment of regional ventricular deformation. Segmental strain of RV may provide a more objective assessment of RV function.

Aims
To evaluate changes in systolic function in single RV, between three stages of surgical repair in HLHS.

Hypothesis
1. RV Global strain will be impaired during 3 stages of repair.
2. RV septal segments will show impaired strain compared to lateral segments, due to tethering effect of the hypoplastic LV.

Methods
RV segmental strain was calculated from previously acquired 4 chamber images (DICOM) prior to and within 3 month after each surgical stage. 21 patients with HLHS (ages 0-4.8 years) were retrospectively evaluated, before and after 3 stages of palliation. Age matched controls without congenital heart defects. Tomtec software was used to measure segmental strain of the 6 RV segments along septal and lateral walls.

Results
Global longitudinal strain remained low through all 3 stages of repair. Strain in lateral segments of RV was higher than the septal segments. There was continued improvement in RV septal function between Stage II and stage III. RV fractional area change (FAC) was low through all stages of repair.

Conclusions
Global RV strain remains low during all stages compared to age matched controls. Septal segments showed the phenomenon of PSS. Septal segments had lower strain compared to lateral segments. Despite the presence of the hypoplastic LV, PSS improved in the septal segments of RV after the Stage II operation. FAC is a simple but useful indicator of global RV function.

Implications
Strain is a useful technique for evaluating both segmental and global RV function in an objective manner. PSS may indicate presence of viable myocardium in RV septal segments, which gradually regained contractile properties over time and overcome the tethering effect of hypoplastic LV, attached to those segments.
Title: Aortic Stent Fractures in the Coarctation of the Aorta Stent Trial (COAST).

Abstract: Large “biliary” and other endovascular stainless steel stents have been used off-label as a treatment option for coarctation of the aorta (CoA) in teens and adults for >15 years, yet little information is available on the structural durability of these devices. Even though catheterization physicians are aware that these stents can fracture, the incidence of this when used to treat CoA poorly characterized. As part of the prospective Coarctation of the Aorta Stent Trial (COAST) fluoroscopic examination of the study device (the NuMED Cheatham Platinum (CP) Stent) has been performed on a scheduled basis and provides an opportunity to assess CP stent durability when used to treat CoA.

Methods: In a prospective, multi-center, single-arm clinical study 105 patients received Bare Metal CP Stents for treatment of CoA, using a uniform protocol. Biplane cine-fluoroscopic examination was obtained at 12 and 24 months post implant.

Results: Of 105 patients enrolled 104/105 implants were successful with one stent migration. There were no deaths or serious complications. All patients achieved relief of Systolic Blood Pressure (SBP) gradient from ascending to descending aorta in the cath lab with a residual of 1.9 ± 3.8 mmHg. Ninety-three patients (89%) returned for 1-year follow-up and 90 (85%) returned 2 years. Sustained improvement was observed at 24 months with 96% of patients having < 20 mmHg arm-leg SBP difference.

No stent fractures (SF) occurred on implant. Fluoroscopy at 1 yr demonstrated minor SF in 2/93 (2%) patients. However, fluoroscopic imaging at 2 years revealed 11 additional SF (13/105, 12%). Two fractures involved a single strut, while nine involved multiple struts. The 2 subjects noted to have a single strut fracture at 12 months had additional SF noted at 24 months. Fluoroscopic follow up beyond 24 months is incomplete, but both new fractures and progression of fracturing has been demonstrated. To date we have recorded 20 fractured stents (~20%). No patient with SF has experienced loss of stent integrity, requiring surgical or catheter re-intervention.
Title: Localized ST Segment Elevation at the Onset of Acute Pericarditis
Rey Arcenas¹, Analkumar Parikh¹, Vijai Tivakaran², Vaskar Mukerji², ¹Kettering Cardiovascular Fellowship Program, 3535 Southern Boulevard, Kettering, Ohio 45429, USA, ²Cardiology Department, Dayton Veteran Affairs Medical Center, Dayton, Ohio, Dayton, Ohio, USA

Abstract: The classic electrocardiographic (ECG) finding in acute pericarditis is diffuse ST segment elevation in most leads, except lead aVR. In ST segment elevation myocardial infarction (STEMI), ST segment elevation is characteristically elevated in leads corresponding to a localized vascular area of the infarct only. We are presenting a patient with acute pericarditis who initially presented with localized ST segment elevation.

A 60 year-old hypertensive male smoker with chronic obstructive pulmonary disease was seen in consultation for chest pain. He had pleuritic chest pain located on the right lower chest associated with dyspnea four days after doxycycline pleurodesis for spontaneous pneumothorax. Cardiovascular exam was normal except for tachycardia of 133 beats per minute. He had a normal ejection fraction by 2-d echocardiogram. Initial ECG showed sinus tachycardia. Sixteen hours later, his repeat ECG showed ST segment elevation in the inferior leads. His chest pain was persistent. Acute inferior wall STEMI was suspected. He underwent cardiac catheterization and was found to have normal coronary arteries and preserved left ventricular function. Subsequent ECG performed twenty-one hours after the onset of his chest pain revealed diffuse ST segment elevation in the anterolateral leads and worsening ST segment elevation in the inferior leads. Diffuse PR segment depression was also seen. He was treated with indomethacin and improved clinically with no recurrence of chest pain. ECG changes resolved on the fifth day.

The ST segment elevation in acute pericarditis is characteristically diffuse compared with STEMI where it is limited to anatomical groupings of leads corresponding to a localized vascular territory. There are physical exam features, 2-d echocardiographic and ECG findings that can help distinguish acute pericarditis from STEMI. However, these features can be absent or subtle during the initial presentation. Measurement of serial cardiac enzymes may also help establish the correct diagnosis, but may be delayed in timing. This case demonstrates that a patient with acute pericarditis may initially manifest localized ST segment elevation in the ECG before the development of diffuse ST segment changes. At that stage, acute pericarditis may be confused with STEMI.
Title: Complete Versus Incomplete Angiography Prior to Percutaneous Coronary Intervention in ST-Elevation Myocardial Infarction

Kevin Stiver¹, Satya Shreenivas², Konstantinos Boudoulas¹, Ernest Mazzaferri¹, Scott Lilly¹, ¹Ohio State University Medical Center, Columbus, OH, USA, ²University of Pennsylvania, Philadelphia, PA, USA

Abstract: The necessity of complete diagnostic angiography prior to percutaneous coronary intervention (PCI) in the setting of acute myocardial infarction is unclear. Directed angiography and immediate intervention enables shorter door-to-balloon times but precludes the full atherosclerotic burden which potential ramifications for the urgency, type, and manner of revascularization. We sought to (1) identify the incidence and predictors of incomplete angiography (IA) prior to PCI; (2) determine the frequency with which IA was associated with potentially surgical disease.

Methods: We retrospectively identified cases of coronary angiography in the setting ST-elevation myocardial infarction between March 2013 and April 2014. Angiography was deemed complete (CA) or incomplete (IC) based on the performance of left and right coronary angiography prior to percutaneous intervention. Surgical disease was defined as left main > 50% or three-vessel (> 70%) coronary artery disease, or two-vessel (> 70%) coronary artery disease including proximal LAD. Demographic, clinical and procedural variables were compared with chi-square and analysis of variance as appropriate.

Results: During the study period 131 cases were identified. The mean age was 59 years old and 76% were male. Incomplete angiography occurred in 41 patients (31%), and did not vary with respect to age, infarct vessel, access site, time from onset to presentation or presentation to arrival in the catheterization laboratory (all p > 0.05), though did vary by operator (range 0 – 60%; p < 0.01). Door to balloon times were shorter in the IA group (27 vs 44 min; p < 0.05). The incidence of potentially surgical disease did not vary between CA (n = 24, 27%) and IA (n = 11, 26%), although referral for surgical revascularization occurred only among those with CA (n = 8). Length of stay, death, new onset heart failure, peak troponin values and discharge ejection fraction did not differ between the groups.

Conclusions: Incomplete angiography prior to PCI in the setting of STEMI is common, primarily operator dependent, and associated with shorter door-to-balloon times. However, multi-vessel disease is common in this setting, and IA precludes procedural and peri-procedural considerations that may have merit.
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**Title:** 0012 - Case

**The Direct Effects Of Nifedipine On Cholesterol Crystallization and Vulnerable Plaque's**

Abrar Sayeed¹, Ehtesham Sayeed³, Omair Ali⁴, Abdul Wase⁴, George Abela², ¹Wright State University, Dayton, Ohio, USA, ²Michigan State University, East Lansing, Michigan, USA, ³Deccan College of Medical Sciences, Hyderabad, Telangana, India

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

**Background:** The use of calcium channel blockers (CCBs) in patients with coronary artery disease (CAD) has been controversial. However it has been demonstrated that in patients with CAD, long-acting CCB's, were associated with a reduction in the risk of stroke, angina pectoris, and heart failure. The exact mechanism for these effects was not elucidated.

**Objectives:** The present study evaluated the potential direct effects of Nifedipine in altering cholesterol crystallization as a possible mechanism for plaque stabilization independent of its cardio-protective effect.

**Method:** Cholesterol powder (3 g) was melted in 10-ml graduated cylinders with and without Nifedipine using a heat gun and then allowed to cool at room temperature. Graded doses of Nifedipine were added to the cholesterol to achieve concentrations comparable to serum levels achieved in humans after a single dose of Nifedipine of 60, 90 and 120mg respectively. The average amount of free cholesterol present in a disrupted plaque is 30 mg/g. With a cardiac output of 5 L/min the amount of Nifedipine at above doses that comes in contact with free cholesterol in vivo are 0.38, 0.575 and 0.765mg. Thus, our experimental doses of Nifedipine (36, 55 and 70 mg/3 g cholesterol) were equivalent to in vivo human exposure. Water in 3gm of cholesterol was used as control. Five experiments were conducted at each concentration and the change in volume expansion was measured and averaged for each dose and compared with control. Scanning electron microscopy (SEM) was used to evaluate cholesterol crystal morphology with and without Nifedipine treatment.

**Results:** The effect of Nifedipine on cholesterol crystallization was evident by a significant dose-dependent suppression of volume expansion ($\Delta$VE) during crystallization. The p value was significant at less than 0.005 for all the above drug concentrations. By scanning electron microscopy (SEM), the structure of crystals was altered from pointed tipped to a blunt and dissolving morphology.

**Conclusion:** These findings of attenuation of volume expansion with crystallization and alteration in crystal morphology from pointed tip to blunt with dissolving suggest that CCB's may have additional cardio-protective action by its stabilizing effect on vulnerable plaques.
Title: 0013 - Research

Myocardial Perfusion Imaging in Emergency Department Patients with Negative Cardiac Biomarkers: Yield for Detecting Ischemia, Short-Term Adverse Events, and Impact of Downstream Revascularization on Mortality

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

Background:
In patients with possible acute coronary syndromes, guidelines recommend routine provocative testing after negative cardiac biomarkers. We hypothesized that myocardial perfusion imaging (MPI) would be low yield with limited short-term value and that early revascularization would not affect mortality.

Methods:
We identified consecutive patients referred from our emergency department (ED) between October 2004 and September 2011 who had MPI following negative troponin T tests and non-diagnostic ECGs. We assessed the incidence of abnormal MPI, coronary angiography, revascularization, and mortality.

Results:
In a cohort of 5,354 patients (58.7% female, age 59 + 13, 78.6% TIMI < 2), 9% had >5% and 3.6% had >10% ischemic myocardium. Among patients with TIMI scores <= 2, 6.1% had >5% ischemic myocardium compared to 19.6% of patients with TIMI scores >/= 3 (p<0.001). At 30 days, 7 patients were deceased, 187 had revascularization, and 6 had revascularization for an acute myocardial infarction. Over 3.4 +/- 1.9 years of follow-up, 347 patients died. In propensity matched groups of patients with ischemia, there was no association between early revascularization and mortality (HR 1.00, 95% CI 0.49-2.07).

Conclusions:
Routine provocative testing to detect ischemia prior to ED discharge is low yield in patients with negative troponins and TIMI scores <= 2 and modest yield in patients with TIMI scores >/= 3. In all patients, 30 days adverse events are rare. Finally, in patients with ischemia, we are unable to demonstrate a mortality benefit with early revascularization.
Title: 0014 - CASE
Type I Dual Left Anterior Descending Artery ST Elevation Acute Coronary Syndrome: Acute occlusion of the anomalous long LAD.

Allen McGrew¹, Thomas Ruff⁰, ¹Grandview Medical Center, Dayton, OH, USA, ²Ohio University Heritage College of Osteopathic Medicine, Athens, OH, USA

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

Introduction: Anomalous duplication of the left anterior descending artery (dual LAD) is a rare finding, representing under 1% of patients undergoing coronary angiography. There are a few reports of identifying dual LAD anatomy in patients who present with acute coronary syndrome, but no reports of ST elevation acute coronary syndrome with acute occlusive disease within the anomalous coronary artery. In this report, we present a case of an acute occlusion within the long LAD branch of a Type I dual LAD coronary anomaly and the unique electrocardiographic injury pattern associated with this lesion.

Case Presentation: A 46-year-old white male with a history of smoking, alcohol abuse, and previous use of cocaine, presented to the emergency department with chest pain that started 4 hours prior to presentation. The pain was described as very sharp, substernal, and radiating to his back. Accompanying his pain was dyspnea, diaphoresis, and numbness of his left upper extremity. His symptoms worsened with exertion and became somewhat better with rest but never resolved. He finally presented due to the unrelenting pain. He denied any previous past medical history or surgeries. On physical examination he was hypertensive (175/140 mmHg) and apparently in mild distress with an otherwise unremarkable exam. An EKG was obtained and demonstrated ST segment elevation in leads V4-V6, I, and AVL; with reciprocal ST segment depression in leads AVF and III.

The patient emergently underwent coronary angiography. The initial left coronary angiograms revealed a LAD from which multiple septal perforators arose and what appeared to be a single, proximally occluded diagonal branch. However, the LAD was anatomically unusual only giving rise to one high diagonal branch and continuing a short course before terminating early in the interventricular septum far from the LV apex. The left main and left circumflex arteries were angiographically normal. Right coronary angiography gave the first clue of dual arterial supply of the anterior interventricular septum with retrograde collateral flow identified up the distal anterior interventricular septum. After crossing the culprit lesion with an intracoronary guidewire, the long LAD branch reperfused and was noted to give rise to multiple diagonal branches as it coursed the left ventricular side of the anterior interventricular sulcus before finally returning to the central sulcus to supply the left ventricular apex. The lesion was stented with good angiographic result.

Discussion: Dual arterial supply of the anterior interventricular septum is a rare anomalous finding with now six described anatomical subtypes that have all been presented in case reports. ST elevation acute coronary syndrome in a patient with this anomalous coronary anatomy has not been described, to our knowledge, until now. The unique electrocardiographic presentation of acute extensive anterior and high lateral ST elevation injury pattern that spares leads V1-3 can be suggestive of an acute occlusion of the long LAD branch of a Type I dual LAD anomalous coronary variant. This information can allow preparation and planning by the interventional cardiologist during the revascularization procedure, shortening presentation to revascularization time, and improving morbidity and mortality.

Conclusion: Dual LAD is a rare coronary anomaly and an acute occlusion within one of these anomalous vessels creates a unique electrocardiographic injury pattern. Early identification, aided by recognition of this unique EKG pattern and awareness of the dual LAD variants allows accurate interpretation of the extent of myocardial territory involved and prioritization of diagnostic and therapeutic interventions.
Title: 0015 - Research

Outcomes of Coronary Stenoses Deferred Revascularization for Borderline Versus Non-borderline Fractional Flow Reserve Values

Jayendrakumar Patel¹, Jeremiah Depta², Eric Novak³, Shriti Masran³, David Raymer³, Gabrielle Facey³, Yogesh Patel³, Alan Zajarias³, John Lasala³, Jasvindar Singh³, Richard Bach³, Howard Kurz³, 1Cleveland Clinic Foundation, Cleveland, OH, USA, ²Brigham and Womens Hospital, Boston, MA, USA, ³Washington University in St. Louis, Saint Louis, MO, USA

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

Background: Current evidence supports deferral of percutaneous coronary intervention (PCI) for intermediate severity coronary lesions with fractional flow reserve (FFR) values > 0.80. The real world natural history following deferral of PCI of intermediate lesions with borderline FFR values (0.81 to 0.85) is unknown.

Objective: This study evaluated the outcomes of patients after deferred revascularization of coronary stenoses based on a borderline FFR value.

Methods: We retrospectively studied 720 patients with 881 intermediate-severity coronary stenoses who underwent FFR assessment from October 2002 to July 2010 and were deferred revascularization. Patients were divided into gray zone (0.75 to 0.80), borderline (0.81 to 0.85), and non-borderline (>0.85) FFR groups. Any subsequent percutaneous coronary intervention or coronary artery bypass grafting of a deferred stenosis during follow-up was classified as a deferred lesion intervention (DLI). Patient and/or lesion characteristics and clinical outcomes were compared between the FFR groups using univariate and propensity score adjusted inverse probability of weighting Cox proportional hazards analyses.

Results: During a mean follow-up of 4.5 +/- 2.1 years, 157 deferred lesions (18%) underwent DLI by percutaneous coronary intervention (n = 117) or coronary artery bypass grafting (n = 40). No statistically significant differences were observed in clinical outcomes between the gray zone and borderline FFR groups. Lesions with a borderline FFR were associated with a significantly higher risk of DLI compared with lesions with non-borderline FFR values (hazard ratio 1.63, 95% confidence interval 1.14 to 2.33, p = 0.007). Lesions deferred revascularization because of a borderline FFR (0.81 to 0.85) were associated with a higher risk of DLI compared with lesions with a non-borderline FFR (>0.85).

Conclusion: Coronary lesions deferred revascularization based on FFR assessment with a borderline FFR value (0.81 to 0.85) were associated with a higher risk of DLI compared with lesions with a non-borderline FFR (>0.85). Further study is needed to refine the management of coronary lesions with a borderline FFR value.
Title: 0016 - RESEARCH
Transient Ischemic Dilatation using Regadenoson – What does it tell us about coronary disease?
Mashhood Ajaz Kakroo¹, Mohammed Andaleeb Chowdhury¹, Mujeeb Sheikh¹, Lucy Goodendy¹, Sadiq Khuder¹, Jodi Tinkel¹,
¹University of Toledo, Toledo, Ohio, USA

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

Abstract
Background: Transient ischemic dilatation (TID) of the left ventricle with stress testing is associated with multivessel coronary artery disease (CAD) and is a significant predictor of future cardiovascular events. The objective of this study was to determine the relationship between TID ratio reported during regadenoson gated single-photon emission computed tomography (SPECT) myocardial perfusion imaging (MPI) single isotope studies, and extent of CAD noted on angiography.

Methods:
3591 patients underwent clinically indicated single isotope MPI using regadenoson stress and Tc-99m tetrofosmin between April 2010 and September 2013. Of these 120 patients were found to have TID (by visual interpretation) on MPI. 98 patients with TID underwent coronary angiography. Among these patients, 21 patients had prior coronary artery bypass grafting and were excluded from the study. TID value was calculated with the commercially available software (Corridor 4DM). Mean TID values were compared across the extent of coronary artery disease found on coronary angiography. Patients with >70% angiographic stenosis in epicardial coronary arteries or a major branch or >50 stenosis in a left main artery were defined as having multivessel disease.

Results:
In the 79 patients who were analyzed the mean TID ratio for non-obstructive disease (n=49) was found to be 1.20 with a standard deviation (SD) of 0.19. For obstructive coronary disease involving single vessel distribution (n=19) the mean TID was 1.16 with SD of 0.13. For multivessel disease (n=13) the mean TID ratio was 1.18 and SD was 0.09. History of Diabetes, hypertension and perfusion defect on MPI was not found to be a significant confounder of relationship between abnormal TID and multivessel disease.

Conclusion:
The mean TID in patients with multi-vessel disease was 1.18, which was not statistically significant as compared to non-obstructive coronary artery disease. Higher numeric TID value may not necessarily predict the greater extent of the coronary disease on angiography. And TID value cut off may not be an ideal way of risk stratifying patients with coronary disease. However TID reported as an abnormal finding may still have relevance as 39% of these patients had obstructive disease in one or more coronary distribution on angiography.
Title: Utility of Glycated Hemoglobin for assessment of glucose metabolism in patients with ST segment Elevation Myocardial Infarction

Abstract: The performance of glucose tolerance test (OGTT) in patients with STEMI results in the new recognition of diabetes mellitus (DM) in over 25% of subjects. Current American Diabetes Association (ADA) guidelines enable use of glycated hemoglobin (HbA1c) for diagnosis of DM. We sought to evaluate the incidence and trends in newly diagnosed DM based on admission HbA1c (Figure 1). There was no significant increase in mean body mass index (BMI) and incidence of DM from year 2005 to 2012.

Figure 1.
Title: 0019 - RESEARCH

Relevance of Pulmonary Arterial Capacitance in Mechanically Ventilated Critically Ill Trauma and Surgical Patients

Muddassir Mehmood1, Ronald Markert1, Mukul Chandra1, Mary McCarthy3, Kathryn Tchorz1, 1Wright State University, Dayton, USA

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

Background: Pulmonary arterial capacitance (PAC) accounts for pulsatile right ventricular (RV) afterload. Compared to pulmonary vascular resistance (PVR), PAC better predicts mortality in pulmonary arterial hypertension and correlates with RV function in advanced heart failure. The relationship of PAC and PVR in mechanically ventilated patients under the conditions of acute hemodynamic stress is unknown. We determined the correlation of PAC and PVR and the association of these with RV ejection fraction (EF) and mortality in critically ill mechanically ventilated trauma/surgical patients.

Methods: Thirty two consecutive critically ill and/or injured mechanical ventilated adult surgical patients at a Level I Trauma Center were prospectively enrolled within 6 hr of admission. Invasive hemodynamics were transduced from pulmonary artery catheter every 12 hr for 48 hr. PAC was calculated as the ratio of stroke volume and pulmonary pulse pressure. Spearman’s rank correlation assessed relationship, repeated measures analysis of variance compared survivors and non-survivors and receiver operating characteristic curves examined the association with mortality.

Results: The mean age was 49 ± 20 years (69% males, 84% trauma, 7/32 non-survivors). PAC (range, 0.5-17.6 mL/mmHg) showed strong inverse correlation with PVR (r = -0.62 to -0.78, p <0.001). Wedge pressure was normally distributed (10.1 ± 4.3 to 12.0 ± 3.8 mmHg) and was not related to PAC. Non-survivors had lower RVEF at study initiation and at 48 hr (27 ± 8 vs. 39 ± 11 %, p = 0.036 and 29 ± 8 vs. 36 ± 8 %, p = 0.043 respectively). At 48-hrs, non-survivors had lower PAC (3.1±1.5 mL/mmHg vs. 5.4±1.7 mL/mmHg; p = 0.007) and higher PVR (4.1±1.8 WU vs. 1.8±0.9 WU, p = 0.001). The optimal cut off for PAC at 48-hr to predict mortality was 3.6 mL/mmHg, [sensitivity 71%; specificity 83%; area under the curve (AUC) 84%] and for PVR was 3.25 WU (Sensitivity 71%; specificity 95%; AUC 89%).

Conclusion: In mechanically ventilated trauma/surgical patients during the first 48 hr of resuscitation, assuming the left atrial pressure is constant and not elevated, the pulsatile RV load should be a predictable and a constant proportion of the resistive load.
Title: Correlation of Cardiac Sympathetic Nervous System Dysfunction with Diastolic Left Ventricular Dysfunction in Patients with Controlled Hypertension

Elsayed Abo-salem1, Mouhamad Abdallah1, Mohamed Effat1, Said Alsidawi1, Myron Gerson1, Division of cardiovascular health and diseases, University of Cincinnati, Cincinnati, Oh, USA

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

Introduction: Sympathetic nervous system activity is increased in patients with systemic hypertension. Angiotensin converting enzyme (ACE) inhibitors can effectively control hypertension without a reflex sympathetic stimulation. Limited data are available about the role of sympathetic dysfunction in the pathophysiology of diastolic dysfunction among patients with controlled hypertension receiving ACE-inhibitors.

Methods: Twenty four non-diabetic patients with controlled hypertension on ACE-inhibitors, normal left ventricular systolic function, without heart failure and not currently on β-blocker therapy were included in the study. Patients were divided into 2 groups based on diastolic function as defined by echocardiography, group A with diastolic dysfunction (10 subjects) and group B without diastolic dysfunction (14 subjects). Patients underwent ambulatory blood pressure monitoring for assessment of nocturnal blood pressure dip and 123I-MIBG imaging to determine heart to mediastinum (H/M) ratio. Plasma norepinephrine (NE) levels were measured.

Result: Patients with diastolic dysfunction had a higher level of plasma NE (0.46 vs 0.26 ng/ml, p=0.01) as compared to patients with normal diastolic function. There was no statistically significant difference in the H/M ratio or the wash-out rate among the two groups (Table). There was a significant inverse correlation between E/A ratio and the log of plasma NE level (Figure).

Conclusion: Generalized sympathetic overflow, but not localized cardiac autonomic dysfunction, may play a role in the pathogenesis of diastolic dysfunction in patients with systemic hypertension that is controlled on ACE inhibitors.

<table>
<thead>
<tr>
<th></th>
<th>Diastolic dysfunction</th>
<th>Normal diastolic function</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>84.5 ± 6.19</td>
<td>58.81 ± 10.99</td>
<td>0.5</td>
</tr>
<tr>
<td>Gender (F %)</td>
<td>50%</td>
<td>64.23%</td>
<td>0.78</td>
</tr>
<tr>
<td>Echocardiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral E/A ratio (median)</td>
<td>0.83</td>
<td>1.22</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>E/e ratio</td>
<td>7 ± 2.26</td>
<td>6.21 ± 1.58</td>
<td>0.33</td>
</tr>
<tr>
<td>Deceleration time (ms)</td>
<td>221.11 ± 22.05</td>
<td>194.14 ± 26.19</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>123I-MIBG scan</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H/M 240 minutes</td>
<td>1.59 ± 0.20</td>
<td>1.00 ± 0.12</td>
<td>0.92</td>
</tr>
<tr>
<td>Washout ratio %</td>
<td>3.1 ± 8.39</td>
<td>6.36 ± 8.89</td>
<td>0.38</td>
</tr>
<tr>
<td>Laboratory data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma NE (ng/ml), median</td>
<td>0.46</td>
<td>0.26</td>
<td>0.018</td>
</tr>
<tr>
<td>Ambulatory blood pressure monitoring</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>124.1 ± 10.7</td>
<td>125.29 ± 12.65</td>
<td>0.81</td>
</tr>
<tr>
<td>Systolic BP dip (mmHg)</td>
<td>10.52 ± 11.4</td>
<td>11.79 ± 11.66</td>
<td>0.79</td>
</tr>
</tbody>
</table>
Title: 0022 - CASE

Magnetic Resonance Imaging of Vascular Malformations
Emily Ruden, Subha Raman, The Ohio State University, Columbus, OH, USA

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

Introduction

Arteriovenous malformations (AVMs) and arteriovenous fistulas (AVFs) are high flow malformations that bypass the normal capillary bed. Magnetic resonance angiography (MRA) may aid in diagnosis and treatment planning.

Case Description

Case 1:
A 52 year-old female presented with a slowly-growing mass on her left foot. Examination demonstrated a 2x3 cm non-pulsatile mass on the plantar medial aspect of the left foot with an audible Doppler signal. MRA delineated an AVM involving the distal posterior tibial artery and vein amenable to surgical repair.

Case 2:
46 year-old male with remote left ankle trauma presented with recurrent left leg ulcerations. Physical examination identified a thrill over the medial aspect of the left calf. MRA confirmed a left posterior tibial artery AV fistula, which was successfully ligated with resolution of the thrill upon discharge. At one month follow-up, a palpable thrill was again present. Repeat imaging confirmed a second AV fistula, for which he subsequently underwent ligation.

Case 3:
A 24 year-old male presented with dyspnea and right heart failure. The estimated right ventricular systolic pressure by initial echocardiography was 65 mmHg. Cardiac magnetic resonance confirmed severe right ventricular enlargement and dysfunction while also demonstrating a previously-undetected pulmonary AVM (PAVM). Subsequent angiography confirmed a moderate-sized aorto-venous pulmonary malformation. Genetic testing for hereditary hemorrhagic telangiectasia was negative. Despite advanced therapies for pulmonary arterial hypertension, functional capacity continued to decline and he underwent bilateral lung transplantation 4 years later.

Discussion

AVMs are congenital lesions of vascular origin believed to be the result of a focal failure in the normal sequence of fetal vascular development. AVMs consist of feeding arteries, draining veins, and multiple dysplastic vessels that connect these arteries and veins, while AVFs are formed by direct connections between arteries and veins.

Magnetic resonance imaging plays an important role in the characterization of vascular malformations. Imaging findings include high-flow, serpentine, and enlarged feeding arteries and draining veins, which appear as large flow voids on fast spin echo images or high signal foci on gradient echo images with absence of a well-defined mass. Areas of high signal intensity on T1-weighted images may represent areas of hemorrhage, intravascular thrombosis, or flow-related enhancement. Three-dimensional gadolinium-enhanced MRA provides detailed evaluation of feeding arteries and draining veins and relationships to adjacent structures for surgical planning.

PAVMs most often involve direct connections between pulmonary arteries and veins, resulting in intrapulmonary right-to-left shunts. As a consequence, patients can have hypoxemia and complications from paradoxical embolization. The incidence of PAVMs is estimated to be two to three per 100,000 with a slightly higher female predominance. Most PAVMs are hereditary, with 80% to 95% occurring in patients with hereditary hemorrhagic telangiectasia (HHT). The genes mutated in HHT encode proteins involved in the transforming growth factor β (TGF-β) superfamily signaling pathway, which are important in regulating the key processes of angiogenesis. PAVMs may also be idiopathic or occur secondary to trauma, infection, hepatopulmonary syndrome, or bidirectional cavopulmonary shunting. Transthoracic contrast echocardiography is recommended for PAVM screening, while computed tomography angiography (CTA) or MRA provide more definitive data for diagnosis.

Conclusion

Vascular malformations are rare but important pathologic conditions that often require aggressive treatment. Imaging plays an important role in the diagnosis and characterization of these lesions.
Title: 0023 - RESEARCH

Comparative Short and Long Term Clinical Effectiveness of Bivalirudin versus Heparin plus Glycoprotein IIb/IIIa Inhibitors in Patients Undergoing Percutaneous Coronary Intervention: A Meta-Analysis of Randomized Controlled Trials
Tilak Pasala¹, Ajay Vallakati¹, Shari Bolen⁲, Pradeep K. Bhat¹, Sanjay Gandhi¹, John McB. Hodgson¹, ¹Case Western Reserve University/MetroHealth, Cleveland, OH/Cuyahoga, USA, ²Center for Health Care Research and Policy, MetroHealth/CWRU school of medicine, Cleveland, OH/Cuyahoga, USA

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

Background: In randomized control trials (RCTs) bivalirudin has been shown to be non-inferior to heparin plus glycoprotein IIb/IIIa inhibitors (HGPI) in patients undergoing percutaneous coronary intervention (PCI). The purpose of this meta-analysis was to assess whether significant differences exist in short and long term outcomes with bivalirudin use compared to HGPI.

Methods: Systematic search identified 9 RCTs (patients = 21,542) comparing bivalirudin (n = 10,665) to HGPI (n = 10,877) for PCI. Long-term (6-12 months) follow up was available for 17,959 patients. Pooled odds ratio (OR) for major bleeding (MB), MACE (mortality, MI, or repeat revascularization) and net adverse clinical events (NACE: MACE or MB) were calculated with random-effects meta-analyses.

Results: Bivalirudin significantly reduced NACE by 16.7% (OR: 0.78, 95% CI: 0.66 to 0.91) (Figure) and MB by 45.6% (OR: 0.52, 95% CI: 0.45 to 0.60) in the short-term (2 to 30 days). The odds of all-cause mortality, MACE, MI, and repeat revascularization were no different with bivalirudin than HGPI during the short and long term follow up. In STEMI patients, bivalirudin use was associated with higher incidence of short term stent thrombosis but paradoxically lower long term all-cause mortality.

Conclusions: Our analysis demonstrates a short term beneficial effect of bivalirudin over HGPI in patients undergoing PCI, primarily due to a significant reduction in MB. At 6-12 months after PCI, patient outcomes using bivalirudin are no different to HGPI. In STEMI patients, bivalirudin use was associated with lower long term all-cause mortality despite a short term increase in stent thrombosis.

<table>
<thead>
<tr>
<th>Study or Subgroup</th>
<th>Bivalirudin</th>
<th>Heparin plus GPI</th>
<th>Odds Ratio</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Events</td>
<td>Total</td>
<td>Weight</td>
<td>M-H, Random, 95% CI</td>
</tr>
<tr>
<td>1.1.1 UNSTEMI or elective</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CATCHET</td>
<td>5</td>
<td>144</td>
<td>2.1%</td>
<td>0.37 [0.13, 1.04]</td>
</tr>
<tr>
<td>REPLACE-2</td>
<td>275</td>
<td>2375</td>
<td>16.8%</td>
<td>0.82 [0.77, 1.09]</td>
</tr>
<tr>
<td>PROTECT-TIMI 30</td>
<td>24</td>
<td>202</td>
<td>6.4%</td>
<td>1.26 [0.74, 2.14]</td>
</tr>
<tr>
<td>ACUITY</td>
<td>303</td>
<td>2519</td>
<td>17.2%</td>
<td>0.65 [0.72, 1.00]</td>
</tr>
<tr>
<td>NAPLES</td>
<td>18</td>
<td>187</td>
<td>4.8%</td>
<td>0.69 [0.63, 1.32]</td>
</tr>
<tr>
<td>ISAR-REACT 4</td>
<td>130</td>
<td>860</td>
<td>13.5%</td>
<td>0.94 [0.72, 1.22]</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>7047</td>
<td>7242</td>
<td>60.9%</td>
<td>0.89 [0.79, 1.00]</td>
</tr>
<tr>
<td>Total events</td>
<td>756</td>
<td>851</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity: Tau² = 0.00; Chi² = 5.57, df = 5 (P = 0.35); I² = 10%</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Test for overall effect: Z = 1.93 (P = 0.05)</td>
<td></td>
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</tr>
</tbody>
</table>

| 1.1.2 STEMI | HORIZONS-AMI | 166 1800 218 1902 16.3% | 0.74 [0.60, 0.81] | 2008 |
| EUROMAX       | 85 1009 110 1139 12.4% | 0.71 [0.53, 0.93] | 2012 |
| BRIGHT       | 64 729 124 724 11.4% | 0.67 [0.49, 0.90] | 2014 |
| Subtotal (95% CI) | 3618        | 3635            | 39.1%      | 0.64 [0.49, 0.83] |      |
| Total events  | 315         | 450             |            |                  |
| Heterogeneity: Tau² = 0.04; Chi² = 8.84, df = 2 (P = 0.05); I² = 65% |
| Test for overall effect: Z = 3.29 (P = 0.001) |

| Total (95% CI) | 10565       | 10877           | 100.0%     | 0.70 [0.56, 0.81] |      |
| Total events   | 1071        | 1311            |            |                  |
| Heterogeneity: Tau² = 0.05; Chi² = 21.63, df = 8 (P = 0.009); I² = 63% |
| Test for overall effect: Z = 3.05 (P = 0.002) |
| Test for subgroup differences: Chi² = 5.03, df = 1 (P = 0.02); I² = 80.1% |
Title: 0024 - CASE

Retrieval of a Fractured and Retained Intracoronary Balloon: a Case Report
Jonathan Forquer1, Mohamed Effat6, 7 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
The devices and techniques used during percutaneous coronary interventions continue to expand in variety and complexity. Despite the growth of the field, the incidence of retained PCI equipment remains low. Although rare, embolization of all or part of these retain devices poses a significant risk of myocardial infarction, arrhythmia and death1-2. Percutaneous retrieval can be difficult and dangerous, especially in more distal coronary arteries 2-4. In this report, we describe the use of the simple, balloon assisted method of retrieving a broken balloon-shaft system from the left anterior descending artery of a patient with ischemic coronary heart disease.

CASE REPORT
A 63 year old male was referred to the cardiac catheterization laboratory for a recent history of recurrent chest pain and a stress test showing ischemia in the apical-apical, apical-septal and apical inferior walls of the left ventricle. The diagnostic angiogram showed a 99% stenosis in the mid-LAD with TIMI 2 distal flow. Additionally, the previously placed stent in the proximal LAD was widely patent. The remainder of the coronary arteries showed only mild coronary artery disease. An XB 3.5 guide catheter was used to engage the left main coronary ostium and a Terumo Runthru 0.014” standard length wire was used to cross the mid-LAD lesion. Next, an Apex 2.5 mm x 15 mm semi-compliant balloon was inflated to 14 atm of pressure for 20 seconds for pre-dilation. This was followed by a Promus Premier 3 mm x 18 mm drug-eluting stent. After the stent was deployed, a NC Quantum Apex 3.5 x 12 mm balloon was introduced into the guide, with minimal bending during insertion. Upon entrance into the stent, the balloon was unable to be pulled back. At this point in time it was realized that the shaft of the balloon had broken, and the balloon with approximately one-half of the shaft was retained within the left anterior descending artery. At this point in time we provided supplemental heparin, and introduced a Cougar 0.014” standard length wire. This was navigated under fluoroscopy past the balloon and distally in the LAD. We next introduced a 2 mm x 15 mm semi-compliant balloon in to the coronary ostium and a NC Quantum Apex balloon and both balloons and wires were withdrawn into the guide catheter. The 2 mm x 15 mm Apex balloon was inflated to 30 atm of pressure, so as to trap the shaft of the NC Quantum Apex 3.5 x 12 mm balloon in the guide catheter. While visualizing under fluoroscopy, all devices and wires were withdrawn from the patient. Final angiographic images were obtained showing no coronary complication. The patient had no complaints throughout the procedure.

DISCUSSION
While uncommon relative to the number of percutaneous coronary interventions every year, the incidence of retained PCI equipment is reported in the literature. Older data report an incidence between 0.1% and 0.8%3, however many of these devices have been redesigned and updated. The devices that have been described include guidewires, guiding catheters, IVUS catheters, rotational atherectomy burrs, expanded and unexpanded stents, and balloon delivery systems 4-6. Brilakis et al. reported an incidence specifically for stent loss at 0.32%6. Prompt identification and retrieval of these potential emboli is necessary to prevent serious complications, including myocardial infarction, unstable arrhythmias, perforation and death. Typically reported methods for retrieving retained equipment have been emergent CABG, snare devices, entangled two-wire technique (or modified double helix) and a simple balloon technique, used here3-6.

Snares and two-wire technique pose potential problems if dealing with under-deployed stents. Manipulation within the coronary artery could also entangle a free stent strut and possibly dislodge the previously placed stent4-6. The simple balloon technique is a safe and effective method of securing the retained equipment without excessive manipulation within the coronary artery or stents. Kharage, et al. reported the use of a semi-compliant balloon to entrap the broken shaft within the guide catheter, as was performed in this case1. Previous case reports describe using a non-compliant balloon alternatively, with similar success3.

Understanding the devices and techniques available for retrieving retained percutaneous coronary intervention equipment is key to avoiding further damage and complications, unnecessary emergent CABG, and death. An important point to emphasize with this case was the need to optimize the alignment of the guiding catheter with the ostium of the left main in a coaxial orientation so as to avoid injuring the left main with the broken balloon shaft as the broken device and rescue balloon were withdrawn with the catheter as one assembly.
Title: Utility of late gadolinium enhancement to predict response to cardiac resynchronization therapy: a meta-analysis

Marissa Edmiston¹, Ajay Vallakati¹, Ashish Aneja¹, Metrohealth Medical Center, Cleveland, USA

Abstract: Late gadolinium enhancement on cardiac magnetic resonance imaging (LGE-CMR) has been shown to predict adverse cardiac outcomes including morbidity and mortality and ICD response. Few studies, limited by small sample size, have examined the relationship between myocardial scar and cardiac resynchronization (CRT) response. We performed a meta-analysis to determine whether scar identified on LGE predicts response to CRT in non-ischemic and ischemic cardiomyopathy.

Background: Late gadolinium enhancement on cardiac magnetic resonance imaging (LGE-CMR) has been shown to predict adverse cardiac outcomes including morbidity and mortality and ICD response. Few studies, limited by small sample size, have examined the relationship between myocardial scar and cardiac resynchronization (CRT) response. We performed a meta-analysis to determine whether scar identified on LGE predicts response to CRT in non-ischemic and ischemic cardiomyopathy.

Methods: Electronic database PubMed was systematically searched for clinical trials reporting CRT response based on scar determined by LGE-CMR. Primary outcome was defined as improvement in NYHA class or improvement in echocardiographic parameters (including dP/dT, radial strain and reduction of LV end systolic volume). Random effects model was used to pool the data across the studies.

Results: After screening 1876 articles, we identified 14 clinical trials. A total of 984 patients were included in this study. There was no significant heterogeneity across the studies (I²=30%, p=0.13). Presence of scar on LGE-CMR decreased CRT response by 39% (RR: 0.61 (95% CI 0.53 – 0.71; p<0.001). Exclusion sensitivity analysis did not change the effect size. Pooled analysis of studies reporting only ischemic cardiomyopathy showed presence of scar decreased CRT response by 38% (RR: 0.62 (0.44 – 0.86; p=0.004).

Conclusions: The absence of myocardial scar detected by LGE predicts an optimal response to CRT in both ischemic and non-ischemic cardiomyopathy. This shows that identification of scar using LGE-CMR can be used as an important risk stratification tool for CRT response.

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Total (95% CI) 505 (479, 100.0%) 0.61 (0.53, 0.71)

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Total (95% CI) 226 (96, 100.0%) 0.62 (0.44, 0.86)

Favors SCAR + Favors SCAR -
Title: Point-of-Care Platelet Function Testing Predicts Bleeding in Patients Exposed to Clopidogrel Undergoing Coronary Artery Bypass Grafting: Verify Pre-Op TIMI 45 – A Proof-of-Principle Study

Grant Reed¹, Amit Kumar², Jianping Guo³, Sary Aranki³, Prem Shekar⁴, Arvind Agnihotri⁵, Andrew Maree⁶, Kenneth Rosenfield¹, Christopher Cannon³, ¹Cleveland Clinic, Cleveland, OH, USA, ²Lahey Clinic, Burlington, MA, USA, ³Brigham and Women’s Hospital, Boston, MA, USA, ⁴St. Elizabeth’s Medical Center, Brighton, MA, USA, ⁵St. James’ Hospital, Dublin, Ireland, ⁶Massachusetts General Hospital, Boston, MA, USA

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

Background
Guidelines recommend delaying coronary artery bypass grafting (CABG) for 5-days after discontinuing clopidogrel. However, platelet function may recover quicker in certain individuals.

Objective
This pilot study evaluated whether preoperative measurement of platelet function with a point-of-care assay could predict bleeding during CABG.

Methods
Verify Pre-Op – TIMI 45 was a prospective study of 39 patients on clopidogrel who subsequently underwent CABG. Preoperative on-treatment platelet reactivity was assessed with VerifyNow® P2Y12 Reaction Units (PRU), with higher PRU indicating more reactive platelets. Outcomes were stratified by PRU quartiles, as well as cut-points for the lowest quartile (PRU ≤ 173), a cut-point for major bleeding optimized by receiver operator curve (ROC) analysis (PRU ≤ 207), and clopidogrel resistance (PRU ≤ 230).

Results
Patients in higher PRU quartiles experienced smaller decreases in hemoglobin and hematocrit (p<0.05 for all comparisons), less major bleeding (p=0.021), and less major or minor bleeding (p=0.003). Patients above the PRU 207 and 230 cut-points had less chest tube output (p=0.041 and p=0.012, respectively), less major bleeding (p=0.005 and p=0.036, respectively), and less major or minor bleeding (p=0.013 and p<0.001, respectively). By ROC analysis, pre-operative PRU discriminated between patients with and without major bleeding during surgery (area under the curve: 0.76; 95% confidence interval: 0.59-0.94; p=0.018). By regression, each increase of 1 PRU decreased the odds of major bleeding by 1.5% (Odds Ratio: 0.985; 95% CI: 0.973-0.977; p=0.018).

Conclusions
In this proof-of-principle study, we found that point-of-care platelet function assessment could predict bleeding in patients recently exposed to clopidogrel undergoing CABG.
**Abstract:** (Your abstract must use Normal style and must fit into the box. Do not enter author details)

**Introduction**

Apico-aortic conduit (AAC) is a method of bypassing native aortic valve, and involves creation of a conduit from apex of left ventricle to the aorta. In the past, AAC was primarily considered a treatment option for patients who had severe aortic stenosis (AS) and were not considered surgical candidates. Indications for AAC may include heavily calcified “porcelain” ascending aorta, history of coronary artery bypass surgery with patent grafts, large or small aortic annular diameters, increased risk of stroke etc.

**Case Description**

We present a case of a 65 years old man with history of ascending aortic aneurysm repair who presented with recurrent episodes of congestive heart failure (CHF). He had history of AAC more than 15 years ago, indication of which was not clear. On examination, he did not have murmur in the aortic valve area, however, he had click on posterior thorax examination. He underwent cardiac catheterization, transthoracic echocardiogram (TTE) with doppler imaging and computed tomography (CT) chest for evaluation of AAC. The anatomy of AAC was clearly seen on CT chest with 3D reconstruction. The mechanical valve within the AAC was found to be patent on TTE with doppler imaging. There was no significant gradient across the prosthetic valve within the AAC to suggest significant stenosis.

**Discussion**

AAC is a method of bypassing native aortic valve by creation of a conduit from apex of left ventricle to the aorta. The evolution of newer methods for treatment of aortic valve stenosis such as transcatheter aortic valve replacement (TAVR) may have limited the role of AAC, however, it remains an alternative method of treatment of AS in situations where surgical valve replacement or TAVR may not be a viable option. This was demonstrated by Henry A. Tran et al. in a recent case of a 74 years old man with history of chest radiation for Hodgkin’s disease, coronary artery disease, status post-coronary artery bypass grafting, and mitral valve repair in 2007 who was referred for evaluation of aortic stenosis after presenting with acutely decompensated congestive heart failure, left ventricular ejection fraction 35%. His aortic valve area was 0.6 cm²; the peak and mean gradients were 60 and 34 mmHg, respectively. Surgical aortic valve replacement (AVR) was not considered due to severe diffuse calcifications of the thoracic aorta. His aortic annulus was too large for TAVR. He underwent successful on-pump implantation of an LV apex to the descending thoracic aorta conduit with a porcine bioprosthetic valve (Freestyle®, Medtronic, Inc.). Pre-existing prosthetic valve, native aortic annulus < 16 mm or > 24 mm, vascular access problems are among some of the exclusion criteria for TAVR in the PARTNER trial. CT, MRI scan along with TTE or TEE can be used to evaluate potential complication such as apical pseudoaneurysms, kinking of the conduit and stenosis of the prosthetic valve within the AAC. Satoru Domoto et al. demonstrated use of PC-MRI to evaluate hemodynamic after AAC. PC-MRI revealed bidirectional flow without stagnation in the descending aorta and the to-and fro blood flow in a small part of diastole.

**Conclusion**

In the era of emerging new technologies such as TAVR for treatment of aortic stenosis, AAC may still have a significant role in the management of aortic stenosis in selected patient population. It is vital to have knowledge of this life saving intervention especially in patients who are not candidates for traditional surgical and newer catheter based aortic valve replacement. There are many different imaging modalities, such as TTE, CT and/or MRI that can be used to evaluate patency of the conduit and prosthetic valve within it.
Title: A Unique Cause of Pulmonary Artery Obstruction and Syncope during Pregnancy

Saad Ahmad1, Francisco Lopez-Mendez1, Said Alsidawi1, Angel Lopez-Candales1, Robert O’Donnell1, Erin Conway-Habes1,
1University of Cincinnati, Cincinnati, OH, USA, 2Cincinnati Childrens Hospital Medical Center, Cincinnati, OH, USA

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

Introduction: A pregnant woman presenting with shortness of breath, chest pain and syncope suggests pulmonary embolism. Here we discuss a cystic mass causing near complete obstruction of the pulmonary artery mimicking pulmonary embolism.

Case: A 26 year old female who was 14 weeks pregnant (Gravida3, Para2-0-0-2) presented with acute onset shortness of breath, chest discomfort and syncope. Non-gated computed tomography (CT) of the chest with intravenous contrast was performed. The study was interpreted as a massive pulmonary arterial filling defect in the proximal pulmonary arterial trunk consistent with pulmonary embolism. The patient was then transferred to a tertiary care center where duplex dopplers and D-Dimer were normal and the diagnosis of a pulmonary embolism was questioned. A transthoracic echocardiography (TTE) was performed demonstrating a dilated right ventricle and a complex cystic resulting in pulmonic valve obstruction. Given the evidence of a mass in the pulmonary artery and the conflicting interpretations between CT and TTE, non-contrasted cardiac magnetic resonance (CMR) imaging was performed for further characterization. A 23 x 25 x 27 mm well circumscribed, heterogeneous cystic mass was identified causing critical obstruction of the proximal pulmonary artery. Specific sequences were used that ruled out a fatty tumor or a simple cyst. Perfusion and late gadolinium enhancement was not performed due to the pregnancy. The velocity across the mass was greater than 5.5 m/s. The right ventricle was both dilated and hypertrophied suggesting gradual onset of elevated right heart pressures. Due to the critical nature of the obstruction, she underwent urgent surgical excision of the mass with an uneventful recovery. Pathology reported a proteinacious cyst with histology suggesting the walls were of valvular origin. Elastin staining ruled out papillary fibroelastoma.

Discussion: Intra-cardiac blood cysts are an extremely rare finding most often associated with the left side of the heart. There are only 12 cases reported with pulmonic valve involvement. To the authors’ knowledge, this is the first case of a proteinacious cyst involving the pulmonic valve. The morphology of the right ventricle on CMR suggested chronic right ventricular pressure overload consistent with slow growth of the proteinacious cyst. Although the patient had chest pain and dyspnea in the months prior to her admission, she presented acutely with syncope. Her presentation was secondary to an increased gradient across the valve/mass either due to hemodynamic changes associated with pregnancy or possibly acute change in the cyst size (an area of hemorrhage was identified in the cyst).

Conclusion: Diagnosis of cardiac masses is often challenging since symptoms may mimic other disease processes. Incorrect diagnosis may lead to inappropriate therapy or delay appropriate management. Cardiac gating of images is essential to allow accurate assessment of the heart and any associated structures. In this case, an extremely rare proteinaceous cyst presented as chest pain, dyspnea and syncope suggesting pulmonary embolism. CMR is the gold standard for assessment of cardiac masses and was essential in characterizing the cyst prior to surgery.
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Do **NOT** write outside the boxes. Any text or images outside the boxes **will** be deleted.  
Do **NOT** alter this form by deleting parts of it or adding new boxes. Simply enter your information into the boxes. The form will be automatically processed – if you alter it your submission will not be processed correctly.

**Title:** 0029 - CASE  
**Massive Hemopericardium as a Subacute Complication of Pectus Excavatum Repair**  
Naveen Saha¹, Akira Wada¹, Kevin Stiver¹, Alex Auseon¹, ¹The Ohio State University Wexner Medical Center, Ross Heart Hospital, Columbus, OH, USA

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

**Introduction**

Pectus excavatum is a congenital anterior chest wall deformity resulting in sternal depression. Symptoms can include dyspnea, chest pain, and poor functional capacity. Both surgical and nonsurgical treatment modalities exist. Catastrophic cardiac complications can occur from surgical management.

**Case presentation**

A 22 year old male with a long standing history of pectus excavatum presented to thoracic surgery for elective repair after noting symptoms of sharp substernal pain and exertional dyspnea. Evaluation prior to consideration for a modified Ravitch procedure included an echocardiogram that demonstrated possible myxomatous mitral valve disease and associated leaflet prolapse, raising suspicion of Marfan’s syndrome. Referral was made to the adult congenital heart disease clinic where he was found to have a lack of associated clinical features and an eventual unremarkable cardiac magnetic resonance imaging study. With the determination of acceptable perioperative risk, the patient proceeded with his planned operation. His post-operative course was complicated by chest pain, ST changes, and a small pericardial effusion attributed to reactive pericarditis. He was discharged home with a four week course of 400 mg ibuprofen four times a day.

Three weeks later, the patient presented to an outside hospital with acute onset of tachycardia, scant hemoptysis, dyspnea, hypoxia and borderline hypotension. Supplemental oxygen was provided and a spiral computed tomography scan showed bilateral pneumonia, pleural effusions, a possible left lower lobe segmental pulmonary embolism and a moderate to large sized circumferential pericardial effusion. Treatment with a continuous heparin infusion and broad spectrum antibiotics were initiated and the patient was transferred to the thoracic surgery service at Ohio State.

The next morning, a surface echocardiogram demonstrated a massive circumferential pericardial effusion with a “swinging heart”, right atrial and right ventricular diastolic collapse, and exaggerated respiratory variation of mitral and tricuspid inflows. The patient received fresh frozen plasma and intravenous phytonadione prior to transfer to the cardiac catheterization suite for anticipated drainage of his effusion (his final read of his computed tomography scan was negative for pulmonary embolism). Emergent pericardiocentesis via the subxiphoid approach with ultrasound as well as fluoroscopic guidance was performed and 1000 mL of frank bloody pericardial fluid was aspirated. A pericardial drain was inserted and removed after 48 hours. The patient clinically improved over the remaining week and was then discharged home in stable condition.

**Discussion**

The decision to undergo pectus excavatum repair is a difficult one and involves a complicated mix of anatomic, physiologic, and psychosocial factors. Complications from surgical management include pneumonia, wound site infection, failed repair, and uncommonly pericarditis. Hemopericardium has also been described as a rare, late complication of repair due to screw migration or bar fracture years after the procedure. Our case describes one of the first instances in the subacute window of massive hemopericardium without evidence of trauma from a mechanical remnant. It remains unclear if this was a slow, indolent bleed over time or hemorrhagic conversion in the setting of reactive pericarditis treated with nonsteroidal anti-inflammatory drugs.

**Conclusion**

This case serves as a stark reminder that despite the high success and low complication rates of pectus excavatum repair, there still exists a risk of severe cardiac complications given the procedure’s invasion of the intrathoracic cavity. Awareness of the cardiac complications of such an elective repair is critical when advising patients who are considering definitive therapy of their pectus excavatum.
Title: Right ventricular outflow to aortic valve velocity ratio: A relative dimensionless index for severe aortic stenosis.

Ali Abdul Jabbar1, Omair Ali1, Ronald Markert1, George Broderick1, Bryan White2, 1Wright State University, Dayton, Ohio, USA, 2Wright Patterson Air-Force Base Medical Center, Dayton, Ohio, USA

Abstract: Echocardiographic indices for evaluation of valvular heart disease have been developed over the past 40 years. While many of them are obsolete, the rest have become standard methods of assessing the severity of aortic valve disease.

Defined by the ratio of left ventricular outflow tract (LVOT) velocity to the aortic valve velocity, the dimensionless index (DVI) has an important role to evaluate the severity of aortic valve stenosis. The value of the latter is evident when there is a discrepancy between other measures of severity, including aortic valve maximal velocity, mean pressure gradient across the aortic valve, inaccurate measurement of LVOT diameter, and when the left ventricular cavity is too small or the ejection fraction is severely impaired. This concept is well validated in prior reports.

In our study, we measured the right ventricular outflow (RVOT) to aortic valve velocity ratio in patients with aortic valve disease who had undergone surgical replacement of their aortic valve in order to derive a relative index that can be used as a surrogate for severe aortic stenosis.

Methods: Patients' charts were reviewed for those who had surgical treatment for valvular heart disease between January 2010 and June of 2014. Patients who had aortic valve replacement due to underlying severe aortic stenosis were included in our study. Those with incomplete echocardiographic assessment, contaminant surgery of other valves, valvular regurgitation (significant aortic and/or pulmonary regurgitation), and those who were treated medically were excluded form the analysis. Out of 231 patients' charts reviewed, only 49 patients matched our inclusion criteria.

Echocardiographic assessment was reviewed and proofread again following data collection for accuracy of measurements. The RVOT TVI was measured from the base-short axis view with pulse-wave Doppler positioned just around 0.5 cm below the level of the pulmonic valve. The RVOT outflow view was used as an alternative to the base-short axis view when the quality of the latter was technically limited.

Discussion: The accuracy of measurement of the mean trans-aortic pressure gradient by Doppler ultrasonography has been validated in previous reports. However, the distinction between critical and noncritical aortic stenosis often requires an accurate calculation of the aortic valve area, which is a flow-dependent measurement. DVI is one of the parameters to assess the severity of aortic valve disease whenever a non-invasive calculation of aortic valve area is technically challenging.

Physiologically, the right ventricular stroke volume should be the same as the left ventricle, even when there is a high-output state, an aortic insufficiency, or a left ventricular pump failure. Using the RVOT velocity to derive a relative dimensionless index (R-DVI) to assess the severity of aortic valve disease is a novel concept.

Results: The patients had a total of 54 echocardiographic assessments during different stages of their aortic valve disease progression. Only 60% of the patients had confirmation with left heart catheterization. All had an aortic valve replacement.

For those patients who had a surgical replacement of aortic valve disease for severe aortic stenosis, the DVI ratio of <0.25 was 100% specific for diagnosing severe aortic stenosis (defined by aortic valve area <1 cm², and/or proved with hemodynamic assessment). The sensitivity of a DVI ratio <0.25 was only 78.6% while that of the R-DVI was 100% with a positive predictive value of 91.3% for diagnosing severe aortic valve stenosis.

Conclusion: An R-DVI ratio may be used to assess the severity of aortic stenosis when the measurement of other traditional indices is technically difficult.
Title: 0031 - CASE
An Elusive Drop in Hemoglobin Post-Catheterization.
Montoya Taylor¹, Revathi Ravi¹, Nikita Desai¹, Dilesh Patel¹, Nishaki Oza¹, ¹The Ohio State University, Columbus, USA

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

Introduction:
The major etiologies for anemia post cardiac catheterization includes major bleeding from RP hematoma, GI bleed or vascular access complications.

Case Presentation:
A 59 year old African American man with history of coronary artery disease status post drug eluting stent to his left anterior descending artery 3 months prior to presentation presented with sub-sternal chest pain radiating to his neck and shortness of breath. On admission physical exam was unremarkable with stable vital signs. His troponins peaked at 0.10 ng/ml. Cardiac catheterization revealed in-stent restenosis and he underwent drug eluting stent in stent placement. He was discharged on dual antiplatelet therapy. He represented within 24 hours with chest pressure, exertional dyspnea and diaphoresis. His exam was remarkable for jaundiced appearance. His hemoglobin dropped from 11.4 g/dl (at discharge) to 7.5 g/dl. Additional his total bilirubin was elevated at 3.4 g/dl with predominant indirect hyperbilirubinemia (indirect bilirubin 3 g/dl). His reticulocyte count was elevated at 13.6 % with a peripheral smear showing schistocytes and blister cells. A detailed history and medication reconciliation revealed trimethoprim-sulfamethoxazole use initiated ten days prior for UTI. Testing revealed he was G-6PD deficient. His antibiotics were discontinued and he received three units of blood. He was discharged with folate supplementation.

Discussion:
Anemia has been established as an independent predictor of mortality after percutaneous intervention. Blood dyscrasias lead to elevation of inflammatory markers, which can exacerbate in-stent restenosis by increased neointimal formation. Our hypothesis is that the anemia with concomitant inflammatory state from smoldering hemolyis likely precipitated his in-stent restenosis. Medication reconciliation and lab review is critical to address issues which could worsen cardiovascular outcomes.
Multi-vessel Thromboembolisms Resulting in Acute Myocardial Infarction in a Twenty-four Year Old Patient
Montoya Taylor¹, Dilesh Patel¹, Nishaki Mehta¹, Konstantinous Dean Boudoulas¹, ¹The Ohio State University Wexner Medical Center - Division of Cardiology, Columbus, USA

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

Introduction/objective
Thromboembolism of coronary arteries can result in acute myocardial infarction; although not often encountered in clinical practice, one must be aware of this phenomenon in the differential diagnosis for acute myocardial infarction.

Case presentation
We report a case of a 24-year-old previously healthy male who developed shortness of breath, chest tightness, and the sensation of his throat swelling. He attributed his symptoms to be from an allergic reaction to cats. He presented to our medical center for further evaluation, and he was found to have acute ST elevations on electrocardiogram in the inferolateral leads. He was taken emergently for coronary angiography which revealed extensive thromboembolic disease throughout his coronary vasculature. Thrombus was present in the left main coronary artery, distal left anterior descending (LAD) artery, first diagonal branch of the LAD, proximal left circumflex (LCx) artery, and first obtuse marginal branch. Manual aspiration thrombectomy was performed with some improvement. Due to persistent thrombosis, mechanical thrombectomy (Angiojet®) was also used in the left main and proximal LCx arteries. However, partial thrombus remained in the distal portion and branch vessels at the end of his left heart catheterization. As a result, the patient remained on a heparin infusion during his hospitalization and was transitioned to Rivaroxaban, an oral anti-coagulant. Extensive search for the source of his emboli was performed during his hospital stay. There was no patent foramen ovale seen by transthoracic echocardiogram, and no deep venous thrombosis identified by lower extremity Duplex ultrasound. Hematologic evaluation did not identify any hypercoagulable disorder, and toxicology screening demonstrated only cannabis. Due to the life-threatening nature of his presentation, we decided, in discussion with our hematology colleagues, that he should continue on a lifelong regimen of aspirin and systemic anti-coagulation. Rivaroxaban was chosen due to its ease of use. The patient was discharged from the hospital without any complications and on follow-up he was asymptomatic and doing well.

Discussion
This case highlights a rarely reported event that cardiologists may encounter in which thromboembolism results in an acute myocardial infarction. In this case, the exactly etiology of the thrombus formation is not clear, as our patient did not have any identifiable precipitating causes. Other case reports of thromboembolism and myocardial infarction have been associated with disorders such as essential thrombocytosis and heparin induced thrombocytopenia; other potential risk factors may also include diabetes, hypertension, smoking and cocaine use.

Conclusion
Thromboembolism to the coronary arteries resulting in acute myocardial infarction is a rare phenomenon. It should be considered in the differential diagnosis of acute myocardial infarction in young patients and warrants prompt therapy.
Title: **0034 - RESEARCH**

**Transesophageal Electrophysiology Studies with Intravenous Adenosine: A Minimally Invasive and Cost-Efficient Strategy to Assess Risk of Sudden Cardiac Death in Patients with Asymptomatic Wolff-Parkinson-White Syndrome**

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**Abstract:**

Patients with Wolf-Parkinson-White Syndrome (WPW), even when asymptomatic (aWPW), are at risk of sudden death due to rapid anterograde conduction during atrial fibrillation (Afib). Standard risk assessment requires induction of Afib during a transvenous electrophysiology study (TVEPS) and measurement of the shortest pre-excited R-R interval (SPERRI). However, Afib can also be induced during a transesophageal electrophysiology study (TEEPS). The purpose of this study is to: 1) evaluate if the administration of adenosine after failed TEEPS can successfully induce Afib and 2) assess if this a cost-effective strategy for determining risk in aWPW patients.

**Methods**

A retrospective review was conducted from 2 institutions (1/10-3/14) on aWPW patients. Inclusion criteria: aWPW, age <18 years and failure to induce Afib during standard TEEPS protocol. Adenosine bolus was administered at 0.2mg/kg via rapid intravenous push during atrial burst pacing. Persistence of Afib for >20 seconds was considered successful. Risk of sudden death if SPERRI of <250ms. Medicare reimbursement rates from 2014 were used to create a cost comparison between TEEPS with adenosine versus TVEPS.

**Results**

Inclusion criteria were met by 8 patients. Adenosine administration induced Afib in 4 patients (50%) and 1 (12.5%) had risk. No complications occurred. The total per-patient cost of TEEPS with adenosine after failed TEEPS was $3,979 per patient versus TVEPS $5,020, resulting in cost savings of $1,041. Even with TEEPS with adenosine failure and conversion to TVEPS, TEEPS with adenosine attempt resulted in an average cost increase of only $1,151 per patient (p = 0.44).

**Conclusion**

Assessment of risk for sudden death in patients with aWPW can be performed in a more cost-efficient manner with addition of adenosine during burst pacing when TEEPS alone fails to induce Afib, as compared to TVEPS alone, resulting in cost savings of $1,041.

Figure 1. Decision Model for Risk Assessment in Patients with aWPW
Title: 0036 - CASE
Massive Pulmonary Emboli Following Discontinuation of Rivaroxiban
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Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)
Introduction: The use of novel oral anticoagulants (NOACs) for stroke and systemic embolism prevention in non-valvular atrial fibrillation and as treatment of pulmonary and systemic venous thromboembolism (VTE) has become increasingly common¹. To date, data regarding patient outcomes following discontinuation has not been well studied. We present a patient who developed massive bilateral pulmonary emboli following discontinuation of rivaroxiban.
Case Study: A 69 year old male initially presented to our facility with a right thalamic intracranial hemorrhage. The patient was chronically on rivaroxiban for paroxysmal atrial fibrillation, which was discontinued on admission. Two days later he developed shortness of breath and substernal chest pain. A Computed tomography (CT) angiogram revealed multiple bilateral pulmonary emboli (PE) (Image1). Ultrasound of the lower extremities did not reveal deep vein thrombosis. An inferior vena cava filter was placed. The patient was discharged to inpatient rehab. On hospital day 32, the patient developed cardiogenic shock and acute respiratory failure. Echocardiography revealed a severely dilated right ventricle (RV), severely reduced RV systolic function and underfilling of the left ventricle (LV) (image2). The patient went for emergent pulmonary embolectomy at which time massive bilateral thrombosis was noted. Ultrasound of the lower extremities on hospital revealed bilateral acute and subacute deep venous thrombosis (DVT).
Discussion: Our patient presented with massive bilateral pulmonary emboli within 48 hours after discontinuation of rivaroxiban following an acute intracranial bleed. To our knowledge, this is the first case of post marketing massive pulmonary emboli following discontinuation of rivaroxiban. Workup revealed no other obvious cause for hypercoagulable state. The vast majority of data regarding VTE risk following discontinuation of anticoagulation have been done with vitamin K antagonists.¹-³ Data on the newer factor Xa inhibitors is not well known. This case brings to light the potential complications with discontinuation of NOAC therapy, and confirms the decision to discontinue systemic anticoagulation must be individualized. Whether or not NOACs promote a hypercoagulable state remains unclear and deserves further investigation.

Image1.2. Computed tomography (left) revealing bilateral centrally located pulmonary emboli (red arrows). Image2. Apical four-chamber view (right) showing severely dilated right ventricle
Ruptured Non-coronary Sinus of Valsalva Aneurysm Causing Right Heart Failure
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Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction/Objective:
Sinus of Valsalva aneurysms (SVA) are rare cardiac defects that can be either congenital or acquired. The greatest concern is the risk of rupture. Even if unruptured, they can obstruct adjacent structures. SVA are also associated with malignant arrhythmias and endocarditis. Rupture most commonly occurs in patients between 20-30 years of age. Abnormalities of sinus anatomy, such as the presence of aneurysm, may profoundly alter the distribution of forces at the base of the aorta and disrupt aortic valve function. We report the case of a young man with a known non-coronary sinus of Valsalva aneurysm who presented following rupture of the aneurysm into the right atrium (RA).

Case Presentation:
A young man with partial trisomy 15 and severe global developmental delay suffered an embolic stroke at 14 years old. This led to a diagnosis of a non-coronary SVA. He was started on warfarin, but surgical intervention was not pursued. He was stable until 26 years of age when he presented with several weeks altered behavior, poor oral intake, dry cough, lower extremity edema, and lab derangements. A renal ultrasound ordered by the PCP showed new ascites and dilated hepatic veins. He was transferred directly to the AACHD clinic for further evaluation.

Exam was notable for new weight gain, lethargy, hyperdynamic precordium with an RV lift, tachycardia, prominent S3/S4 gallop, a new harsh III/VI continuous murmur loudest at the right upper sternal border with radiation throughout the precordium, lower extremity edema, ascites, and a pulsatile liver. An echocardiogram confirmed rupture of the non-coronary SVA with blood shunting under high-pressure into the RA. The aneurysm had increased in size and protruded into the RA causing severe obstruction of RV inflow (mean gradient increased to 14-15 mmHg). There was only trivial aortic regurgitation. The RA and RV were dilated with normal biventricular systolic function. A pericardial effusion and ascites were also noted. The patient was admitted and underwent surgery the following day. The surgeon encountered a large effusion upon entering the pericardium. Aneurysmal tissue within the right atrium was resected. A trimmed patch of CorMatrix was used to close the defect from the aortic side and then close the aneurysm tissue from the RA side to achieve a two-layer closure. The aortic valve was repaired by reattaching some of the leaflet to the aortic wall. The tricuspid valve also required repair since there was aneurysmal tissue adherent to it causing poor coaptation. He had a good surgical result and complete resolution of right heart failure. He was also able to discontinue warfarin.

Discussion:
This case represents a subacute presentation of a ruptured non-coronary SVA. As with our patient, about 50-70% of patients have a gradual onset of congestive heart failure symptoms after rupture. Aneurysms from the right sinus of Valsalva are most common (65-85%) with the majority of the rest originating from the non-coronary sinus (10-30%). Rupture is usually into a right heart chamber. Congenital aneurysms occur more frequently than acquired aneurysms, but are still rare among congenital lesions. Several syndromes (i.e. connective tissue disorders and chromosomal abnormalities) are associated with SVA, and we presume that our patient’s SVA was likely due to his partial trisomy 15.

The red flags in this case were the loud continuous murmur and prominent gallop rhythm. These findings represented a definite change from his baseline cardiac exam and were harbingers of a serious cardiovascular problem. Historically, cardiac catheterization with aortography was the gold standard for diagnosing SVA; however, advances in echocardiography have allowed this non-invasive modality to be the initial diagnostic test of choice. Transthoracic echo is usually sufficient, but transesophageal echo can be helpful. Diagnostic cardiac catheterization is now reserved for stable patients in whom coronaries need to be better visualized. Congestive heart failure is the primary cause of death, but endocarditis may play a role in about 8% of patients. Untreated ruptured SVA have demonstrated a mean survival period of 1-4 years in various series; therefore, early surgical intervention is needed.

Conclusion:
In summary, sinus of Valsalva aneurysms are rare but potentially life-threatening defects that are usually congenital in origin. Even large aneurysms may be silent until rupture or obstruction occurs, most likely in the 2nd or 3rd decade of life. A harsh continuous murmur, especially if new and accompanied by signs of congestive heart failure, should trigger concern for ruptured SVA. Transthoracic echocardiogram is the diagnostic imaging modality of choice. Surgical repair of ruptured sinus of Valsalva aneurysms can typically be undertaken with good long-term outcomes.
Infective Endocarditis Due to Granulicatella Adiacens

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

Background: Granulicatella adiacens is a newly nomenclatured, nutritionally variant streptococcus that is normal oral flora, recently implicated in endocarditis and systemic infections. Its contribution to human disease is likely underestimated due to difficulties in culturing it.

Clinical case: An otherwise healthy adolescent female presented to a university health clinic with new-onset epistaxis and dyspnea and was found to have a prominent murmur. Transesophageal echocardiogram (TEE) demonstrated a congenital bicuspid aortic valve with large vegetation, severe aortic insufficiency, sub-annular aortic root abscess and mitral valve vegetation with moderate regurgitation [image]. Blood cultures grew G. adiacens. IV vancomycin was started. Her NT-proBNP level was 6000. She underwent uncomplicated bioprosthetic aortic valve replacement and bovine patch repair of the aortic abscess and primary mitral valve repair. She was discharged on vancomycin but readmitted two weeks post-operatively with recurrent fever and new large mitral vegetation. Meropenem, ampicillin and gentamicin were started and she improved clinically. The mitral valve vegetation remained stable without mitral insufficiency on subsequent TEE.

Discussion: Echocardiographic imaging established the initial diagnosis of endocarditis and facilitated peri-operative medical therapy as well as surgical intervention. Endocarditis linked to G. adiacens is noted to be more severe and have higher rates of mortality than that caused by Streptococcus viridans or Enterococcus sp. A previous study showed 100% of G. adiacens isolates were susceptible to vancomycin, although in vitro testing does not predict in vivo response.

Conclusions: TEE effectively identified an aortic root abscess requiring surgical intervention. By demonstrating improvement in the mitral valve after adjustment in antibiotics, post-operative TEE obviated need for repeat sternotomy and high-risk reoperation.

Figure 1: TEE imaging demonstrating large aortic valve vegetation.
**Title: 0040 - CASE**

Don’t Ignore A Gut Feeling – A Case of Spontaneous Superior Mesenteric Artery Dissection Treated With A Percutaneous Stent Placement  
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**Abstract:** (Your abstract must use Normal style and must fit into the box. Do not enter author details)

**Introduction/Objective:** Isolated spontaneous dissection of the superior mesenteric artery (SMA) is a rare condition with poorly understood etiology and prevalence. Its presentation can vary from asymptomatic to ischemic bowel.

**Case Presentation:** A 62 year old female with history of multiple sclerosis, platelet storage pool deficiency and hypertension had been suffering from postprandial abdominal pain for over a year. She had a history of multiple abdominal surgeries including exploratory laparotomies for hemoperitoneum due to a ruptured ovarian cyst, small bowel obstruction due to adhesions, and an incarcerated internal hernia. After her last laparotomy, she started having episodes of cramping, dull, epigastric and right upper quadrant abdominal pain after eating a meal. This pain would usually last about an hour, and was severe enough for her to develop a food aversion. As a result, she ended up losing 20lb over next several months. To evaluate the cause of her symptoms, she underwent an abdominal computed tomography (CT) scan with contrast and this scan was initially reported to be normal. With ongoing abdominal pain, the CT scan was revisited and a possible superior mesenteric artery (SMA) dissection was discovered. Subsequently, abdominal magnetic resonance angiography (MRA) was ordered and a superior mesenteric artery dissection was confirmed. Her abdominal MRA was notable for a patent 8 mm diameter SMA at the ostium followed by a 3 cm long segment of stenosis (4 mm diameter). This stenosis was followed by a 3.3 cm long dissected segment with the false lumen supplying the first dominant jejuna artery. After non-invasive imaging, she was taken to our catheterization lab for a possible percutaneous stent placement. She underwent an abdominal angiogram, but her initial stent placement attempt was unsuccessful due to the severe angle into the artery from the femoral approach. She was brought back to catheterization lab and a left brachial artery cut down was performed for arterial access. A 6 Fr x 90 cm Raabe Flexor introducer sheath was used along with a Stabilizer Plus 0.014 X 300 cm wire to cross the lesion. A balloon angioplasty was performed with a 4.00 X 30 mm Apex Monorail balloon as the first intervention. A 7mm X 40 mm Precise Pro Rx self-expanding stent was deployed across the lesion with excellent results and good flow was established to all branches of the SMA. The patient tolerated the procedure well and was discharged on Aspirin and Clopidogrel daily. In follow up, she described complete resolution of her post-prandial abdominal symptoms.

**Discussion:** This case highlights a rare entity that is easy to miss but can lead to significant morbidity. Majority of SMA dissections present with acute mesenteric ischemia are associated with aortic dissection. In our patient, she had a subacute presentation with an isolated SMA dissection. Additionally, she did not have any of the frequently associated conditions such as connective tissue disorders, vasculitis, or arteritis that have been reported in the literature. Stress on the wall of the SMA at the inferior pancreatic edge may contribute to the usual origin of the dissection - 1.5 to 3 cm from the origin of the artery. Although rare, these patients may be referred to a cardiovascular specialist for further evaluation, and the key to management of these patients is to suspect the diagnosis based on the clinical history and exam. Once the diagnosis is confirmed with multimodality imaging, treatment of a SMA dissection with a percutaneous approach can be as successful as a surgical approach with significantly less morbidity.

**Conclusion:** SMA dissection can be seen in isolation and may lead to substantial symptoms. It should be considered in patients with appropriate history, and it can be successfully treated with a percutaneous approach.
Abstract: The relationship between left ventricle (LV) and left atrium (LA) can be characterized as interactive and dynamic. This is evidenced in studies characterizing ageing, elite athletic training and multiple disease states. LV afterload is determined largely by its elastic properties and downstream pressure and it increases with more severe LV diastolic dysfunction, especially with elevated LV filling pressures. In patients with severe aortic stenosis (AS), changes in the LV ventricle include reduced ventricular performance (ejection fraction) and LV remodeling (hypertrophy). Severe stenosis can also impair the diastolic function of the left ventricle. In these patients, the onset of diastolic dysfunction occurs from prolonged ventricular relaxation and decreased ventricular compliance caused by a thick non-compliant left ventricular and persistent increased afterload. Because the process of LV relaxation is more energy dependent than contraction, abnormalities of LV diastolic function occur earlier than systolic function. The symptoms of aortic stenosis may manifest due to this early dysfunction. As there is persistent and progressive pressure overload, systolic decompensation begins to occur. In AS, preserved LA function helps in maintaining optimal cardiac output despite the impaired LV relaxation and reduced LV compliance. Investigators have assessed LA contractile function through the measurement of left atrial ejection force (LAEF) and kinetic energy. The LAEF is a potentially useful index in assessing the atrial contribution to diastolic performance. It is the force expended by the LA during atrial systole to propel blood into the LV. It is assessed according to the Newtonian principle, in which force (in dynes) = mass × acceleration. Mass is defined by the product of the density of blood (i.e. 1.06 g/cm³) and the volume of blood passing through the mitral annulus during atrial contraction. By Manning’s method, the left atrial ejection force is calculated by $1/3 \times \text{mitral orifice area} \times (\text{peak A velocity})^2$.

Objective To find the relationship of left atrial ejection force (LAEF) and severe aortic stenosis (SAS).

Methods We evaluated left atrial systolic function using left atrial ejection force (LAEF) in 56 patients with severe aortic stenosis who underwent aortic valve replacement. Of these, 7 patients were excluded due to concomitant mitral annular calcification, 5 patients were excluded due to atrial fibrillation, 2 due to atrial ablation, 1 due to moderate mitral stenosis and 1 due to severe mitral regurgitation. Of the 40 included patients, 19 patients had normal diastolic function and 21 patients had grade 1 diastolic dysfunction as determined by standardized ASE guideline parameters. The ages of all patients ranged from 46 to 91 years of age. The LAEF was calculated as $1/3 \times \text{mitral valve area} \times (\text{peak velocity of A wave})^2$ using two-dimensional and pulsed-Doppler echocardiography according to Newton’s law of motion and hydrodynamics. Normal LAEF was calculated as 0.098 × age - 0.74 (kdynes) from the regression line. Because of this correlation, we used age-corrected LAEF (%LAEF) that was calculated as measured LAEF / normal LAEF × 100. We looked at (1) LAEF before and after aortic valve replacement in patients with normal diastology and grade 1 diastolic dysfunction, (2) The age corrected LAEF before and after aortic valve replacement in patients with normal diastology and grade 1 diastolic dysfunction, (3) Change in LAEF and NYHA class after aortic valve replacement and (4) Change in age corrected LAEF and NYHA class after aortic valve replacement.

Results After aortic valve replacement, the grade 1 diastolic dysfunction group had a mean decrease in LAEF of 0.15 ± 0.94 and the normal group had a mean increase in LAEF of 0.28±0.26 (n.s., p = 0.06). After aortic valve replacement, the grade 1 diastolic dysfunction group had a mean decrease in age corrected LAEF of 3.05 ± 14.52 and the normal group had a mean increase in age corrected LAEF of 4.36±4.78 (significant, p = 0.037).

After aortic valve replacement, patients with improved NYHA class were found to have an increased LAEF of 0.22 ± 0.65, those with worsened NYHA class had a decreased LAEF of 0.68 ± 0.97 and those without NYHA change had an increase in LAEF of 0.24±0.47 (significant, p < 0.01). After aortic valve replacement, patients with improved NYHA class were found to have an increase in age corrected LAEF of 3.81 ± 10.56, those with worsened NYHA class had a decrease in age corrected LAEF of 12.33 ± 14.54 and those without NYHA change had an increase in age corrected LAEF of 3.58 ± 6.64 (significant, p ≤ 0.001).

Conclusion We conclude that after aortic valve replacement, the grade 1 diastolic dysfunction group did not have a significant decrease in LAEF and the normal group did not have a significant increase in LAEF. We also conclude that after aortic valve replacement, the grade 1 diastolic dysfunction group had significant decrease in age corrected LAEF and the normal group had a significant increase in age corrected LAEF. Also, after aortic valve replacement, the LAEF and age corrected LAEF have significant correlation with the NYHA class. These results suggest that in patients with severe aortic stenosis, the pathological abnormalities extend not only to the left ventricle but also to the left atrial muscle. After replacement, there is significant change in the LA mechanics.
The Unrelenting Pericardial Cyst: A Common Presentation With an Uncommon Outcome.
Heath Wilt\textsuperscript{a}, \textsuperscript{b} University of Cincinnati, Cincinnati, OH, USA

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<th>Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)</th>
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<td><strong>Background:</strong> Pericardial cysts are a rare entity, occurring in one case per 100,000; most of which are congenital in etiology, and only one-third recur following surgical excision or drainage. This identifies a unique case of a recurrent, inflammatory pericardial cyst following prior coronary artery bypass grafting.</td>
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<td><strong>Case Presentation:</strong> 53 year old female presented to the ED for complaints of dyspnea over two days associated with intermittent pleuritic chest pain without provocation. She denied fever, chills, palpitations, or resting shortness of breath. Past medical history was significant for bioprosthetic aortic valve secondary to endocarditis resulting from intravenous drug use. She underwent 3 vessel CABG at the time of aortic valve disease for coronary atherosclerosis noted by angiography preoperatively, but does not prior history of acute coronary syndrome. Upon arrival, she mildly tachycardic with stable blood pressure and SpO2 of 100% on room air. Preliminary blood work revealed normal renal functioning, no leukocytosis or anemia. There was mild transaminitis, with AST and ALT at 92 and 91 u/L, respectively. INR was 1.2. Troponin-I was less than 0.01 ng/ml. Presenting ECG illustrated normal sinus rhythm with evidence of left ventricular hypertrophy with nonspecific inferolateral ST segments abnormality. Q waves in leads II and aVF. A contrasted CT of the chest did not reveal evidence of a pulmonary embolus, however it did show an 8.2 cm x 5.2 cm x 1.1 cm pericardial cyst adjacent to the right atrium and ventricle, with mass effect (figure 1). Her prior cardiac surgeon was contacted, reporting a similar mass identified 10 months after her bypass requiring drainage by interventional radiology. She was admitted to the ICU given concerns for mass effect upon the right ventricle. On the evening of the first hospital day, the patient developed transient heart block, but remained hemodynamically stable. Given the structure size and concern for heart block, the patient underwent drainage of the cyst by interventional radiology on hospital day #1, and a subcostal pigtail catheter was left to dependent drainage for several days. Analysis of the fluid collection was consistent with chronic inflammatory changes. Chest pain continued throughout the hospital course, and given her prior bypass, she underwent angiography on the HD #3 illustrating unchanged native coronary disease with patent vein grafts to right coronary artery, second diagonal, and left internal mammary artery to mid-left anterior descending artery. The patient underwent cardiac MR which confirmed anatomical placement within the pericardium (figure 2). There was no evidence of infiltrative disease or extra cardiac foreign body. Delayed gadolinium enhancement along the myocardial wall adjacent to the pericardial cyst suggested myopericarditis. An infectious diseases workup continued. On hospital day #10, the patient underwent urgent drainage of pericardial cyst via right anterior thoracotomy given rapid accumulation following drain removal (figures 3, 4). Serous fluid with fibrous debris was noted. Immediately post-operatively, she developed ST elevation myocardial infarction with troponin-I peaking at 151 ng/mL. Emergent angiography allowed for aspiration thrombectomy and PFTE stent placement to her SVG to RCA graft, perforated intraoperatively. The patient eventually was discharged on hospital day #16. Fluid and tissue specimens were consistent with chronic inflammatory change. She returned six months septic with confirmed bacterial and fungal endocarditis with embolic phenomena confirmed by repeat CT, also revealing the cyst recurrence. The patient eventually opted to transition to hospice care after discussion with her family.</td>
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<td><strong>Discussion:</strong> Pericardial cysts are quite uncommon, occurring in around one case per 100,000 people. An estimated 60-70% are congenital. Of these, an estimated three-quarters are left unrecognized, with the remainder often being found incidentally. It is rare for these structures to exceed 5 cm in their largest dimension. Acquired pericardial cysts are often inflammatory in nature, as suggested in our case by pathologic review. Causes of pericardial cysts are numerous in report: bacterial infections (particularly tuberculosis), Echinococcal infections, rheumatologic conditions, trauma, and cardiac surgery. They typically arise in the right anterior cardiophrenic angle, as with our patient. CT and MRI can help distinguish anatomy and separate from other masses of the chest cavity. Clinical presentation can be varied and half of all cases are asymptomatic. Common complaints include chest pain and dyspnea, although complications include cyst rupture, tamponade, pulmonary and bronchial obstruction, arrhythmia, and RV outflow obstruction or death. Treatment of choice for all cysts include percutaneous aspiration, with some resources advocating ethanol sclerosis. Surgery is an alternate option. Our patient’s cyst was likely from prior surgery, although her case is unique given the frequency and rapidity of recurrence, as well as size (8.2 cm). Cyst recurrence has been reported to be exceedingly rare with any intervention; this case reports a third recurrence despite previous intervention with both percutaneous aspiration as well as surgical excision. Confirmation of a pericardial cyst can be best verified via use of cardiac MR, and specific identification should be pursued to prevent known, albeit rare, consequences with significant morbidity and mortality.</td>
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Title: Not All ST segment Changes Are Myocardial Injury: Hypercalcemia-induced ST-segment elevation

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Abstract: Not All ST segment Changes Are Myocardial Injury: Hypercalcemia-induced ST-segment elevation

Introduction

Acute ST-segment changes on surface electrocardiogram (EKG) could represent underlying myocardial ischemia and are evaluated immediately. However, there are many other conditions that can affect ST segment. In this case report, we would like to present an uncommon presentation of a common electrolyte abnormality causing ST-segment elevation.

Case Presentation

An 83 year-old male veteran with history of coronary artery disease (CAD), ischemic cardiomyopathy with left ventricular ejection fraction of 15%, and many other comorbidities including Multiple Myeloma, was transferred from another hospital to Veterans Affairs Medical Center. He complained of progressive shortness of breath and increasing leg swelling. His symptoms were worsened with exertion and associated with lightheadedness. He recently gained 10 pounds. He denied neither chest pain nor pressure. On exam, blood pressure was 99/66 mmHg, Heart rate was 74 /min and he was saturating 94% on 2 liter of nasal cannula oxygen. Inspiratory rales and crackles were heard on lung exam, significant jugular venous distension and bilateral 2+ pitting pedal edema were noted.

Presenting EKG showed sinus rhythm, heart rate of 90 /min, normal axis and premature ventricular contractions (PVC). Voltage criterion of Left ventricular hypertrophy (LVH) was met. QT interval was 371, and the QTc interval was 418. The most notable finding was ST segment elevation in anterior leads (V1-3) and ST segment depression in lateral leads for which Cardiology was consulted urgently. He repeatedly denied chest pain and his cardiac enzymes were within normal limits. Further evaluation showed anemia, acute renal insufficiency, hyperkalemia and hypercalcemia. We checked his previous EKG recordings and found that ST changes are inconsistent, some of the previous EKG tracings showed ST elevation but some did not.

Patient was immediately treated for acute decompensated heart failure. Emergency cardiac catheterization was deferred since his EKG changes could be explained by significant hypercalcemia, patient had no chest pain and cardiac enzymes were not elevated. Patient was treated with intravenous fluids, diuretics, and Zoledronic acid. ST segment changes resolved on repeat EKG after normalization of his calcium level. Subsequent cardiac enzymes were also negative.

Discussion

Acute myocardial ischemia, the leading cause of death in the world, can present with ST-segment changes. But not all the ST-segment changes are from myocardial ischemia. ST-segment on surface EKG represents ventricular repolarization phase and can be affected by conditions other than myocardial injury. For example, pulmonary embolism, pericarditis, subarachnoid hemorrhage, etc. Reperfusion therapies will have no benefit or may even lead to adverse outcomes if there is no underlying myocardial injury. Incidents have been reported regarding increased morbidity and mortality from unnecessary invasive revascularization procedures. We should be aware and constantly remind ourselves of those conditions.

Calcium is a dominant myocardial cation, and extreme changes of serum calcium will effect myocardial action potential repolarization by itself or affecting other cations transfer across the myocardial cell membrane. In medical literature, EKG features of hypercalcemia are described as: absent or shortened ST segment, shortened QT segment, and lengthened T wave duration. In fact, there had been reported that hypercalcemia may not only lead to QT shortening, but also ST elevation, mostly localized to anterior leads, V1-3. [4] The exact mechanism is not yet fully understood.

Conclusion

This case serves as a reminder that not all ST changes are due to myocardial injury. History taking and physical examination are the most important initial evaluation. Lastly, it is very beneficial to compare prior EKG records with the current EKG before deciding for emergency cardiac catheterization.
Duchenne muscular dystrophy (DMD) is a genetic, X-linked recessive disease. The clinical course in DMD consists of progressive skeletal muscle weakness wasting and weakness. Cardiac involvement in DMD is characterized by myocardial fibrosis which leads to dilated cardiomyopathy, progressive heart failure, and arrhythmias. Earlier detection of cardiac involvement and appropriate treatment thereof holds potential to improve outcomes.

Objective: Cardiovascular magnetic resonance (CMR) imaging for detecting disease progression in DMD patients is based on late gadolinium enhancement (LGE) imaging. LGE-CMR is clinically limited because it is by nature a late finding, and unable to detect diffuse myocardial fibrosis. CMR extracellular volume (ECV) quantification using T1 mapping is a histologically validated non-invasive marker of diffuse fibrosis. The primary aim is to determine ECV in DMD population. The secondary aim is to correlate ECV with additional metrics of left ventricular function.

Methods and Results: The study was a retrospective review of DMD subjects who have undergone CMR imaging at a single institution. Cardiac MR T1 relaxation maps pre- and post-contrast were combined to create ECV maps. Clinical parameters including ventricular volume and mass measurements, ejection fractions, presence and location of late gadolinium enhancement will also be collected. Preliminary data in 16 patients with DMD global myocardial ECV was significantly higher in the DMD group (29 ± 5%) compared with a control group of published normal values (24 ± 2%, p = 0.0003).

Conclusions: CMR T1 mapping technique is a feasible method for quantification of ECV in patients with DMD. Preliminary results demonstrate that global ECV fraction is significantly higher in the DMD population. Future directions hope to compare ECV as marker of cardiac involvement in DMD patients with other clinical markers of cardiac function.
Multiple Spontaneous Coronary Artery Dissections in an Asymptomatic Patient
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Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)
Introduction: Spontaneous Coronary Artery Dissection (SCAD) is a very cause of acute coronary syndrome (ACS) with the reported prevalence of 0.1-1.1% in the angiographic series. It was first reported by Pretty et al. in an autopsy of a young woman in 1931. Though SCAD is a rare cause of myocardial ischemia, multivessel SCAD is much less frequent than single vessel involvement and ACS remains the most common clinical presentation. Prognosis and treatment depends mainly on the anatomical location of the involved arteries and patient’s condition.

Case: A 38 year old male with a history of Type I Diabetes Mellitus for past 30 years, chronic kidney disease, hypertension, hyperlipidemia, and seizure disorder referred to cardiology clinic for preoperative kidney/pancreas transplant work up after nuclear stress test revealed apical ischemia. Given stress test findings, he underwent coronary angiography which showed multiple coronary artery dissections involving proximal Right Coronary Artery (RCA), mid Left Anterior Descending artery (LAD), and proximal to mid diagonal branch of left anterior descending artery. When asked more in details, patient reported symptoms of chronic fatigue and bilateral lower extremity edema in the months leading up to the angiogram. However, he denied any history of chest pain, shortness of breath, dyspnea, pre-syncpe, and syncope. He was brought back to the cardiac catheterization lab two weeks later, and angiography documented identical findings as listed above. IntraVascular UltraSound (IVUS) of the left anterior descending artery revealed a 20 mm long dissection. This portion of the LAD was subsequently stented with a 23 mm bare metal stent, and a post-stent IVUS study revealed no residual dissection. Patient tolerated procedure well and was discharged on aspirin, clopidogrel, metoprolol and atorvastatin. He continued to remain asymptomatic in the follow up visits of one and three months post intervention.

Discussion: Coronary artery dissections may occur secondary to aortic dissection, Marfan syndrome, blunt chest trauma, coronary artery bypass surgery, or cardiac catheterization. A spontaneous coronary artery dissection occurs in the absence of these contributing factors. According to most case series, spontaneous coronary artery dissections are seen on approximately 0.2% of coronary angiograms. More than 300 patients with spontaneous coronary artery dissection have been documented in the literature. More than 70% of coronary dissections occur in women, and 30% of those cases occur in the peripartum period. Female patients presenting with SCAD have increased incidence of left coronary artery dissection and absence of Coronary Atherosclerosis Disease (CAD). In contrast, in male SCAD patients, the right coronary artery is often affected and there is coexisting CAD. 20% of coronary artery dissection cases are reported to involve multiple vessels as in the case of our patient. The pathophysiology of SCAD is unclear. The speculated etiologies of this phenomenon include the following: atherosclerosis, peripartum period, oral contraceptive use, connective tissue disorders (such as Type IV Ehlers-Danlos Syndrome, Marfan Syndrome, and Idiopathic Cystic Medial Degeneration), vasculitis (such as Lupus, Polyarteritis Nodosa), exercise, and prolonged sneezing. The location and extent of the SCAD lesion usually guides therapy. Initial treatment is not standardized but current tendency is towards coronary artery bypass surgery for left main lesions, percutaneous coronary artery intervention for proximal involvement of LAD, left circumflex and RCA, and medical management for more distal lesions.

Conclusion: Spontaneous coronary artery dissection should be considered in young patients presenting with myocardial ischemia or infarction. Contrary to prior belief, SCAD is not only the disease of younger women in the peripartum period or those carrying inflammatory disease, and can be seen in male or middle-aged females. Although some controversy still exists as to what is the appropriate treatment for patients with SCAD, a large majority of patients who survive the acute phase are free from events at follow up.
Case Report:

A 55 year old obese male with multiple risk factors and alcohol abuse presented for worsening exertional dyspnea. His electrocardiogram (EKG) showed sinus rhythm with left atrial (LA) enlargement. An echocardiogram revealed dilated left ventricle (LV) and LA with severely reduced LV systolic function (ejection fraction 15-20%), grade III diastolic dysfunction and elevated LA filling pressure. Patient was treated medically and was referred for coronary angiography to rule out ischemic etiology. Coronary angiogram showed a left dominant system with non-obstructive coronary artery disease and elevated LV end diastolic pressure of 33 mmHg. Interestingly phasic compression (inside-out) of proximal left circumflex (LCX) was noticed during ventricular diastole. The phasic compression of proximal LCX correlated with atrial depolarization on the EKG suggestive of LA contraction being responsible for this phenomenon. To our knowledge, this is the first case report describing dilated LA causing compression of LCX during atrial systole.

Discussion:

The clinical significance of this finding is poorly understood. Previously, this phenomenon was reported in a heart transplant recipient where diastolic compression of the diagonal artery was noted. The patient had no ischemic symptoms and was undergoing routine left and right cardiac catheterization. He was successfully treated with intracoronary stenting once ischemia was demonstrated in the corresponding coronary territory on stress myocardial perfusion imaging. Our patient complained of dyspnea and the diastolic compression of the left circumflex was believed to be secondary to elevated filling pressures as reflected by increased EDP. The compression of the left circumflex artery is probably due to its anatomical relationship with the left atrium. The left atrium contracts to fill a non-compliant ventricle and in the process compresses the left circumflex. The patient was administered decongestive therapy and on follow-up was asymptomatic.
An interesting case of Scimitar syndrome
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Abstract: (Your abstract must use Normal style and must fit into the box. Do not enter author details)

Introduction: An 18 year old girl, diagnosed with Scimitar syndrome during infancy, and was lost to follow up. She presented at 16 years of age with hemoptysis, and was found to have a very unusual vascular anatomy during cardiac catheterization.

Case presentation: An 18 year old girl diagnosed with Scimitar syndrome, dextroposition of heart and hypoplastic right lung in infancy and was lost to follow up. She presented to Emergency room complaining of hemoptysis for 2 days. During this visit, she also complained of gradually worsening shortness of breath, exertional chest pain and orthopnea. Physical examination showed slightly decreased breath sounds on right compared to left with no added sounds and normal cardiac exam. The coagulation screen was normal and haemoglobin level was 13.2. A CT angiogram of chest was significant for hypoplastic right lung with a severely hypoplastic right pulmonary artery, and a large vertical pulmonary vein drains right lung, coursing inferiorly, with dilated suprahepatic inferior vena cava and right and middle hepatic veins suggestive of communication with hepatic venous system. A large arterial branch was seen arising from the suprarenal aorta and extending into the hypoplastic right lung likely represents aortopulmonary collateral. The mediastinum was shifted to right with dextroposition of heart and absent coronary sinus. The transthoracic echocardiogram showed dextroposition of the heart with hypoplastic right pulmonary artery. The right pulmonary veins were not draining to left atrium and the right lower lobe vein was directed to the right pulmonary artery. The unusual vascular anatomy led to cardiac catheterization, which showed normal RV pressures, Qp:Qs of 1.47:1, pulmonary vascular resistance of 0.99 units/m². The angiograms showed hypoplastic right pulmonary artery with poor distal arborization and no demonstrable forward flow but retrograde flow in to left pulmonary artery. Angiogram of descending aorta revealed large collateral from the descending aorta supplying the right lower and middle lobe and drained in to a vertical venous channel communicating with the right pulmonary artery. The balloon occlusion of the right pulmonary artery led to increase in pressure by 20-25 mmHg in the aortopulmonary collateral. The lung perfusion scan showed mild heterogeneous activity in the left lung and non visualization of the right lung.

On exercise stress test, she had normal cardiac response to maximal exercise with significant cardiac limitation based on her maximum oxygen consumption (69% of predicted) and below average maximum ventilation (40% of predicted). Her hemoptysis resolved and her symptoms improved. Since she improved, we decided to not intervene at that time, with the plan that if she developed any cardio-respiratory compromise, we would consider surgical resection of the right lower lobe. She has severe persistent asthma with severe airway obstruction and significant exercise induced asthma on bronchodilators and inhaled steroids. At her last visit, 18 month later, she is asymptomatic from cardiac perspective.

Discussion: Scimitar syndrome is a rare congenital anomaly, characterized by a combination of partial or complete pulmonary venous return from the right lung to IVC, either above or below the diaphragm along with hypoplasia of the right lung and sometimes a systemic arterial supply to the right lung. About 19 to 31% patients with Scimitar syndrome may have an associated cardiac anomaly. Our patient was diagnosed with this syndrome in infancy, but we also discovered an unusual vascular drainage of right lung, which was in addition to her typical findings of Scimitar syndrome. The unusual vascular finding consisted of an abnormal large caliber vein that drained the right lower lobe of hypoplastic lung and into the right pulmonary artery. Despite the given anatomy, her right ventricular pressures were within normal limits demonstrated by cardiac catheterization and there was no evidence of pulmonary hypertension.

Conclusion: Our case demonstrates that even if a patient falls into a defined cardiac anomaly or syndrome, associated findings should always be sought as the exact vascular anatomy may differ in each patient. Also, for proper delineation of any structural anomaly of the heart, more than one diagnostic modality is often required to evaluate and consider potential treatment options.

![Picture1](image-url)
Three Dimensional Imaging of the Patent Ductus Arteriosus using Optical Coherence Tomography
Guruprasad Mahadevaiah¹, James Hill¹, Marco Costa², Hiram Bazerra², Christopher Snyder¹, Michael Jenkins², ¹Rainbow Babies and Children’s Hospital, Cleveland, OH, USA, ²Case Western Reserve University, Cleveland, OH, USA

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

Background: Patent Ductus Arteriosus (PDA) accounts for 5-10% of all congenital heart disease. Echocardiography is the primary imaging technique for evaluation of PDA. Angiography is used to assess ductal anatomy during interventional closure of the ductus arteriosus. Optical coherence tomography (OCT) is an optical imaging modality using light waves analogous to ultrasound using sound waves. OCT uses infrared light to provide high resolution (10-15μm) images of vessel wall anatomy and is used for coronary imaging.

Objective: We are evaluating the use of optical coherence tomography (OCT) to image the PDA, given the potential benefit of superior resolution and lower radiation.

Methods: Standard angiograms were performed on two patients with patent ductus arteriosus prior to device occlusion. OCT was then used to obtain high-resolution three-dimensional vessel reconstructions. Devices were chosen based on angiographic measurements.

Results: Angiograms and OCT are shown in Figure 1 below. OCT imaging gave a more complete assessment of ductal anatomy and would have led to choosing a larger device than what was chosen based on angiography. In the first case, the device was too small and easily pulled through the ductus necessitating upsizing. The device that was eventually delivered was what would have been chosen had we used OCT measurements. In the second case, coil diameter was chosen to be approximately twice the minimal ductal diameter by angiography, and ended up with successful closure despite being only 1.3x the vessel diameter. Had OCT measurements been used, a larger coil would have been chosen.

Conclusion: We found OCT imaging of the PDA to be feasible, and only used a small amount of additional radiation and contrast. The three-dimensional OCT reconstructions provided additional anatomic information that could potentially improve device selection. In addition, once the technique is perfected, little or no angiography or fluoroscopy will be required to perform imaging runs, and only a small injection of contrast appears to be sufficient for vessel imaging. More work needs to be done to comprehensively evaluate this modality, but if OCT is shown to be comparable or superior, it may potentially replace standard angiography by improving accuracy and decreasing overall radiation use in selected interventional procedures such as this one.
Title: 0049 - CASE
The Hidden Source, an interesting case of Non bacterial Thrombotic Endocarditis in a patient with Granulomatosis with Polyangiitis
Diego Alcivar Franco1, Jonatham Kanam1, Kevin Silver1, 1Summa 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 Granulomatosis with Polyangiitis as a cause of unexplained non bacterial thrombotic endocarditis
2. Review the pathophysiology, diagnosis, and management of cardiac involvement of Granulomatosis with Polyangiitis

Case Presentation
The patient is a 20 y/o Amish female who presents with past medical history significant for being treated since spring of 2014 for recurrent upper airway infections. She had been prescribed several courses of antibiotics but has failed every one. Before admission she was seen in the ED with urinary retention and was sent home on antispasmodics and a foley catheter. Days later is admitted with hemoptysis, unquantified fever and weakness. VS: T 36.7C, HR 127, BP 115/67mmHg, RR 22, O2sats on room air 91%. CT angiogram done showed no PE but presented cavitary lesions within the apices of the lungs. Moments after admission suffered a polymorphic ventricular tachycardia event and was successfully resuscitated and intubated. Her EKG prior to arrest demonstrated a sinus tachycardia, inferolateral T-wave abnormality, and QTc of 428. Initial labs: WBC 27.3, hemoglobin 11.2, and platelets 616,000, lactate 1.6, creatinine 0.56, Sodium 135, Potassium 4.6, bicarbonate 25, ESR 65, CRP 150, LFTs marginally elevated, Urinalysis showed hematuria and Troponin 6.9 and increased to 34.8. Transthoracic echocardiogram showed EF 28% with global left ventricular systolic dysfunction with great regional variability. Basal, anterior and septal walls were akinetic, apical inferolateral and anteroseptal walls were hypokinetic. Mild MR with no clear vegetation and a small pericardial effusion. The patient was placed in isolation given concern for TB, puncultures were negative and septic shock pattern was decreasing and cardiogenic shock pattern increasing. LHC was done, EF 20%. LVEDP 34mmHg, Severe triple vessel CAD. Occluded distal first diagonal artery, distal first obtuse marginal artery, mid to distal ramus intermedius and occluded mit posterior descending artery. Wall motion abnormalities correlated with occluded vessels and TTE. Later labs ANA 1:40, cANCA 1:80, IgG 1440.0, IgM 100.0, IgA 269.0, MPO negative pANCA negative. Granulomatosis with Polyangiitis was diagnosed and pulse steroids were given with significant improvement. After extubation patient presented Vfib arrest, cardioverted and assessed with TEE which found a vegetation in the atrial valve of 1.5 x 1cm. 2 Weeks into hospitalization and given improvement in clinical picture a follow up TEE after steroid and rituximab therapy showed decrease in vegetation to 1.2 x 0.7 cm. Patient was discharged with follow up with Rheumatology and Cardiology.

Discussion
Granulomatosis with Polyangiitis mostly occurs in older adults, although it has been reported in all ages, that presents with constitutional symptoms including fever, malaise, arthralgias and weight loss, but its characterizes to by its involvement of the upper and lower airways accompanied by renal impairment. Skin and multiple organ systems can involved including the eyes, nervous system and less commonly the heart, GI and GU tract, parotid glands, liver, thyroid and breast. Our interest in this case brings to discussion the involvement of the heart, which in the literature is described to cause myocarditis, pericarditis and tamponade; but along our research we have found that there have been a few case reports on granulomatus endocarditis or non bacterial thrombotic endocarditis induced by this vasculitis. It’s important to consideration for this disease under the differential diagnosis, the process behind diagnosis (ACR, CHCC and EMA criteria) together with the duration and management with steroids and immunosuppressive medications such as cyclophosphamide and rituximab.
Coronary artery spasm (CAS), also described as variant angina or Prinzmetal angina, is characterized by transient periods of angina associated with ST-segment elevation on the electrocardiogram (EKG), generally occurring in the absence of obstructive coronary artery disease (CAD). Despite a similar presentation to angina pectoris associated with CAD, CAS is a different entity with a better prognosis.

Case Presentation
A 80-year-old African American female with a history of CAD and percutaneous coronary intervention (PCI) to the distal left anterior descending artery (LAD) with a drug eluting stent in the setting of an non-ST elevation myocardial infarction 6 months ago with preserved ejection fraction, presented with midsternal chest pain radiating down the left arm for approximately one and a half hours. She had stable vitals, an unremarkable cardiac exam, an initial troponin of 0.09 that trended downward, and a negative urine drug screen. After sublingual nitroglycerine, her chest pain resolved and she was managed with aspirin, heparin drip and admitted to the cardiology service. An EKG showed diffuse T wave inversions. She underwent angiography showing a normal left coronary artery system with patent LAD stent, however during the catheter exchange for engagement of right coronary artery (RCA), she became hypotensive to a systolic blood pressure of 60 with ST elevation in the inferior leads with quick insertion of an intra-aortic balloon pump to improve hemodynamics. Re-injection of the left coronary system showed severe CAS of the left anterior descending artery as well as the circumflex artery that resolved with intracoronary nitroglycerine. The RCA was found to be completely occluded requiring use of a guidewire and balloon dilation of the ostial vessel to enable flow. A thrombectomy catheter was used to assess for thrombus, which was not seen upon manual aspiration. Intracoronary nitroglycerine was given into the RCA that also resulted in resolution of the CAS. The patient's blood pressure stabilized, a low dose nitroglycerine drip was initiated, and she was admitted to the cardiovascular intensive care unit. The next morning and the next evening, the patient had the same chest pain with ST elevation in the inferior leads that resolved with sublingual nitroglycerine. She was transitioned from an IV nitroglycerine drip to oral isosorbide mononitrate and amlodipine which resulted in no further episodes of chest pain and discharge from the hospital.

Discussion
CAS was first described by Prinzmetal as a “variant form of angina pectoris” characterized by ST segment elevation on the EKG and occurring at rest rather than with exertion, lasting 5 to 15 minutes often from midnight to early morning. It is usually caused by a focal spasm of a coronary artery, however spasm can occur in more than one site and diffuse spasm involving multiple coronary arteries has also been described. Spasm can occur at a site of minor or major stenosis or in angiographically normal coronary arteries. Vascular smooth muscle hyper-reactivity, vasoconstrictor stimuli/autonomic tone, endothelial dysfunction, and microvascular dysfunction are involved in the pathogenesis of CAS with vascular smooth muscle hyper-reactivity thought to be the key abnormality. With the exception of smoking, CAD risk factors such as hypertension and hyperlipidemia do not seem to show a significant association with CAS. Active smoking is associated with approximately 75% of cases of CAS. While the prevalence of CAD increases with age, CAS is more common in individuals less than 50 years of age. Other risk factors for CAS include medications such as ephedrine based products, cocaine, marijuana, alcohol, butane, sumatriptan, and amphetamines. There also seems to be an ethnic difference suggesting a genetic component with a higher prevalence of CAS in Japanese cohorts. The diagnosis is confirmed when transient ST-segment elevation is seen on an EKG in the setting of chest pain and subsequent angiography reveals no high grade stenosis with spastic coronary arteries. An ambulatory EKG (Holter) monitor can detect these periods of transient ST-elevation if spasm has resolved at time of angiography. Intracoronary acetylcholine provocation testing in patients with less than 50% coronary stenoses discovered CAS in 33.4% of cases with only a 1% risk of minor complications. Calcium channel blockers and long-acting nitrates are the treatments of choice; in addition, smoking cessation should be emphasized. For patients with refractory focal CAS, PCI can be helpful. Non-selective beta blocker should be avoided as they can cause spasm. Rho-kinase inhibition, which attenuates the vasoconstrictive response to acetylcholine, has been effective, though in small studies. Myocardial infarction and arrhythmias occur in approximately 25% of untreated patients, however long term prognosis exceeds 80%. A literature review resulted in only a few case reports describing cardiogenic shock from CAS.

Conclusion
CAS is characterized by transient episodes of chest pain associated with ST-segment elevation on the EKG and angiography excluding significant fixed obstructive disease. This case was an atypical presentation of CAS in an elderly non-smoking female with diffuse multivessel coronary involvement requiring use of an intra-aortic balloon pump to manage cardiogenic shock.
**Title: 0051 - CASE**

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<th>Spontaneous Left main and Right Coronary artery spasm in a patient with Vasospastic Angina.</th>
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<td>Analkumar Parikh¹, Rey Arcenas¹, Ajay Agarwal², ¹Kettering Medical Center, Kettering, Ohio, USA, ²VA Medical Center, Dayton, Ohio, USA</td>
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**Abstract:** (Your abstract must use Normal style and must fit into the box. Do not enter author details)

**Introduction:** Coronary artery spasm causing supply and demand mismatch can mimic coronary ischemia resulting in unnecessary interventions. Among the few reported cases of left main coronary artery spasm, most descriptions are due to catheter based irritation of vessel. However, bilateral spontaneous coronary artery vasospasm of the right and left main coronary artery is an exceptionally rare occurrence in a single patient and during the same procedure. We describe a patient who had a strong clinical history consistent with vasospastic angina that demonstrated spasm of left main and proximal right coronary artery.

**Case Description:** The patient was a 43-year-old male who was admitted to the psychiatry ward from an outside hospital for further management of homicidal and suicidal ideation. Patient developed chest pain 3 days post admission. On further questions he gave a history of frequent and recurrent chest pain symptoms over the last 1 year. These episodes were associated with substernal chest pain which had tightness like quality. Physical examination was otherwise normal. His past medical history was significant for hypertension, hyperlipidemia, tobacco use, polysubstance abuse. On initial evaluation the EKG showed sinus bradycardia without acute ST changes. The laboratory evaluation was within normal limits including the initial cardiac enzymes. He underwent a myocardial perfusion imaging study which showed moderate reversible ischemia in anterolateral region, suggestive of significant ischemia. Echocardiographic examination was without wall motion abnormalities. Subsequently, coronary angiography was performed, and the initial left coronary injections revealed significant left main coronary narrowing. The left anterior descending and circumflex coronary arteries were without any significant lesions. The right coronary artery angiogram revealed presence of severe spasm in the proximal segment with appropriate response to 200 mcg intra-coronary nitroglycerin. Subsequently, the left main coronary artery was reengaged and another dose of 200 mcg of nitroglycerin was given intra-coronary. On the repeat left coronary angiograms the left main stenosis due to coronary vasospasm was resolved. The patient was started on calcium channel blockers, counseled on smoking cessation.

**Discussion:** Coronary artery spasm is typically associated with other substances including alcohol, marijuana, amphetamine, chemotherapeutic agents, and antimigraine therapy. Our patient suffered from severe episodes of chest pain, thought to be caused by inadequate coronary blood flow. At angiography he appeared to have severe stenosis of left main and right coronary artery which improved with intracoronary nitroglycerin injection. The cause of the coronary artery spasm is uncertain: a result of injection of contrast medium due to mechanical irritation by tip of the catheter or a spontaneous phenomena. The obvious clinical differentiation of coronary spasm from fixed obstructive disease has significant clinical implications. Upon literature review we did not find any reports of bilateral right and left main coronary artery spasm. In this report we describe a patient in whom left main and right coronary artery spasm was relieved only following administration of intracoronary nitroglycerin. This case demonstrates one of many challenges to interventional cardiologist for which there are no other evidence based guidelines or randomized trials. The vasospasm of the coronary arteries maybe spontaneous or iatrogenic.(catheter induced). Vasospasm much be considered in differential diagnosis particularly when angiography reveals no significant atherosclerosis disease in other coronary arteries. Current anti-vasospastic therapy of choice are calcium channel blockers and/or nitrates. Beta-blockers are contraindicated as beta blockade may cause unopposed alpha-adrenergic stimulation and increased vasoconstriction.
Title: 0053 - CASE

Infected Endocarditis with a Large Pericardial Effusion
Timothy Byrnes¹, Roger Chaffee⁰, ¹Summa Health System, Akron, Ohio, USA

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

Introduction/Objective:
Infected endocarditis can infrequently present without any cardiac lesion and rarely with large pericardial effusions.

Case Presentation:
29 year old caucasian male presents with chief complaint of sudden onset of chest pain that awoke him from sleep at 0500. Chest pain is retrosternal, waxes and wanes since onset, exacerbated by deep inspiration and supine position, relieved by sitting forward. Chest pain associated with diaphoresis, fever, and shortness of breathe. Denies any prior similar episodes, recent sick contacts, or illnesses.

Past Medical History: left buttock MSSA abscess 1.5 years ago

Social History: + tobacco (1 pack/day), + alcohol (4 beers/day), no illicit drugs, single, lives with mother, server at restaurant

Vital signs: 103.0 ° F, HR = 124, RR = 18, BP = 143/81, SpO2 = 99%RA, weight = 152 kg

Physical exam:
General: uncomfortable, AAO x 3

Neck: supple, no JVD

CV: RRR, + S1 and S2, no M/R/G, no S3 or S4

Lungs: CTA b/l, no W/R/R/C

Abdomen: + BS, soft, NT/ND

Extremities: pulse 2+ b/l distal UE and LE, no peripheral edema

Skin: 1 cm elliptical open wound with surrounding erythema of left buttock without draining purulence

Labs: BMP significant for glucose of 202 mg/dL. CBC significant for WBC of 16.0 mg/dL with 39% bands. Troponin = 3.93 ng/mL. C-reactive protein = 90 mg/L. HbA1c = 6.5%.

Imaging: CXR = no acute process. EKG = sinus tachycardia, diffuse ST-segment elevation and PR-segment depression.

Patient admitted for acute myopericaditis to telemetry with ibuprofen 600 mg every 8 hours and colchicine 0.6 mg/dL. Transthoracic echocardiogram found LVEF of 57% with concentric LVH and small pericardial effusion. Blood cultures grew gram positive cocci in clusters 2 out of 2 samples. On questioning patient complained of ‘pimple’ of left buttock that he drained 2 days prior to admission. On exam had 1.5 cm elliptical open wound with surrounding erythema without purulent drainage and started on vancomycin 1.5 gm IV every 8 hours. The following day blood cultures grew MSSA on 2 out of 2 samples and vancomycin discontinued and nafcillin was started. CT chest and abdomen with IV contrast found multiple ill-defined nodular densities bilateral, small bilateral pleural effusions, and large pericardial effusion. The following day repeat blood cultures negative. Three days later transesophageal echocardiogram found LVEF of 60%, mild LVH, aneurysmal atrial septum without PFO, all valves structurally and functionally normal, and a large echolucent pericardial effusion with possible RV apical diastolic collapse. The following day a subxiphoid pericardial window and drain with removal of 1000 mL of serous fluid was performed. Pericardial fluid and tissue cultures negative. Pathology studies consistent with fibrinous pericarditis with fibrosis and inflammatory pericardial effusion without malignancy. The following day a PICC line placed, pericardial drain removed, and patient discharged home with IV ertapenem for 6 weeks.

Discussion:
Infected endocarditis can present without any cardiac lesions in ~25% of cases and with large pericardial effusions ~2% of cases. Typically patients that present with large pericardial effusions are younger and have a history of IV drug abuse. Although the pericardial fluid and tissue studies did not reveal an infectious source, it has been shown in the previous that this is not uncommon although the reason is unknown. It is possible in this setting that the MSSA could have cleared with antimicrobial treatment, as pericardial drainage was performed 7 days after the initiation of therapy. As this patient has possible complicated right sided IE it was felt to be prudent to treat with 6 weeks of IV antibiotics.

Conclusion:
Infected endocarditis can present without cardiac lesions and rarely with pericardial effusions. Pericardial effusions in the setting of IE typically do not grow microbacteria. Treatment with 6 weeks of IV antibiotics is recommended for left sided and complicated (embolic phenomenon or heart failure) right sided IE.