Fellow in Training Research Abstracts Oral Competition

Indiana-ACC Poster Competition Abstract

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Please structure your case study abstract using the following headings: * Introduction/objective * Case presentation * Discussion * Conclusion

Title:

Fragmented QRS on the 12 Lead ECG: A Marker of Sudden Cardiac Death

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Background and Objectives: Sudden cardiac death (SCD) is associated with a low probability of survival. Fragmented QRS complex (fQRS) on a 12-lead ECG, which include various RSR' patterns, are shown to be markers of altered ventricular depolarization and substrate for ventricular arrhythmia. We postulated that the incidence of an fQRS in the SCD population would be relatively high compared to a population without cardiac disease and possibly be a predictor of SCD.

Methods: Demographics, ECG, and lab data of patients who presented with cardiac arrest (CA) were studied. Of these 166 had SCD. The patients with SCD were compared to 264 age and sex matched healthy controls (HC).

Results: The prevalence of fQRS was 57.2% and 22.3% in the SCD and HC groups respectively (SCD vs. HC, $p<0.0001$). The incidence of fQRS and/or Q waves was 60.2% and 23.5% in the SCD and HC groups, respectively (SCD vs. HC, $p<0.0001$). Patients with fQRS and SCD were more likely to have fQRS in multiple major lead territories compared to fQRS patients with the HC group (16.84% vs 1.69%, $p=0.0035$). Patients with fQRS were more likely to have SCD with an OR of 3.58 (95% CI: 2.15-5.96, $p<0.0001$) when compared with the HC group.

Conclusions: Compared to healthy controls fQRS on the 12-lead ECG has a relatively high incidence in patients who have experienced SCD and merits further investigation as a possible predictor of SCD alone or in conjunction with other ECG markers of abnormal depolarization or repolarization.

Indiana-ACC Poster Competition Abstract

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Title:

Left Bundle Branch Cardiomyopathy and His-Bundle Pacing

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Background: Left bundle branch block (LBBB) has been classically depicted as an electrical delay of conduction manifested by a mechanical dysynchrony of left ventricular contraction with a resultant decrease in systolic function. His-Bundle Pacing (HBP) addresses, in the most physiological sense, the underlying electrical abnormality. While there has yet to be a randomized control trial establishing LBBB as an etiology of left ventricular systolic dysfunction, there has been numerous studies establishing a relationship between left bundle branch and the development of a clinical cardiomyopathy in both humans and animal models. Labelled as Left Bundle Branch Cardiomyopathy, the response of this cardiomyopathy to His-Bundle Pacing is unknown.

Objective: To demonstrate the feasibility and impact of His-Bundle Pacing on Left Bundle Branch Cardiomyopathy

Methods: Five patients who met criteria for LBBB Cardiomyopathy were found and underwent HBP. HBP was performed using the SelectSecure pacing lead (model 3830, 69 cm, Medtronic Inc, Minneapolis, MN) as previously described. The lead was delivered through a fixed curve sheath (C315HIS, Medtronic Inc; Minneapolis, MN) or a deflectable sheath (C304, Medtronic Inc, Minneapolis, MN). His bundle (HB) electrograms were mapped with the pacing lead in unipolar 115 fashion and recorded with Medtronic pacing system analyzer (model 2290) and/or on a EP 6 recording system based on operator preference. Selective HBP (S-HBP) was defined as ventricular activation occurring solely over the His-Purkinje system: (1) His-Purkinje mediated cardiac activation and repolarization as evidenced by electrocardiographic concordance of QRS and T wave complexes; and (2) the paced-ventricular interval was almost identical to the His-ventricular interval. LBBB was defined as QRSd >140 ms in men (>130 ms in women) with mid-QRS notching in two contiguous leads. The lead was then fixed at the optimal position 120 by means of 4–5 clockwise rotations. The HB capture threshold was assessed and recorded at a pulse width of 1.0 ms.

Results: Three patients had selective HBP capture and two patients had non-selective HBP. The ejection fraction demonstrated at 108% increase, improving from 25% at baseline to 52%. The QRS duration decreased by 28%, narrowing from 152 ms at baseline to 109 ms at follow-up. NYHA classification improved by at least one for each patient. Every patient who underwent His-Bundle Pacing was characterized as a hyper-responder, with improvement in ejection fraction from <35% prior to HBP to >50% after implantation.

Conclusions: His-bundle pacing (HBP) is a viable strategy in treating LBBB-induced cardiomyopathy, addressing the underlying physiology rather than the mechanical manifestations of LBBB.

Indiana-ACC Poster Competition Abstract

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Fellow in Training Research Abstracts

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Title:

Parent and Adolescent Knowledge of Congenital Heart Disease Diagnosis and Associated Risk Factors

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Background: Understanding the diagnosis of congenital heart disease (CHD) and its associated risks is paramount for the guardian, but becomes more important to the individual adolescent as they transition into adulthood and become responsible for their own health care, including decisions about their reproductive health. This study examines parental and adolescent knowledge of the CHD diagnosis, need for spontaneous bacterial endocarditis (SBE) antibiotic prophylaxis, and risk of transmission to offspring if they were to become pregnant. Correlates of adolescent and parental knowledge were examined.

Methods: A sample of dyads of mother/adolescent pairs (N=77) who could independently complete an online questionnaire (adolescent age range 14-21 years) were recruited from university associated cardiology outpatient clinics. Participants completed an on-line survey that included age, ethnicity, level of education, knowledge of their cardiac lesion, indications for prophylactic antibiotics, and risk of transmission of cardiac lesion to offspring. This survey also queried adolescents and mothers about frequency of conversations with health care providers and communications between mothers and their daughters as they relate to various health and reproductive issues. Conversation variables were a summed number about specific topics discussed with provider(s) (range = 0-5) and parent (range = 0-6). A study cardiologist reviewed the patients' medical record to identify the cardiac diagnosis and level of complexity, indication for prophylactic antibiotics, and risk of transmission of cardiac lesion to offspring.

Results: The mean ages of daughters and mothers were 16.7 years (SD=2.1) and 44.8 (SD=6.3), respectively. The majority of mothers (N=75, 97.4%) had at least a high school education or greater. Less than half of the adolescents (n=36, 46.8%) surveyed were able to completely identify their cardiac lesion, compared to 75% (N=58) of mothers who could correctly identify the cardiac diagnosis in its entirety. Adolescents who correctly identified their diagnosis were older (17.2 [SD= 2.0] years versus 16.4 [SD= 2.1] years, NS), more likely to have graduated from high school or to be attending college (X^2=6.134, $p<0.05$), had a mother who could correctly identify their lesion (95.8 % versus 79.3 %, $p<0.05$), and reported more reproductive health care conversations with their health care provider (mean=1.7 [SD=1.7] versus mean=1.0 [SD=1.4], NS) and their mother (4.6 [SD=1.8] versus 3.1 [SD=1.9], $p< 0.001$). Most mothers (N=52, 67.5%) could identify the need for SBE prophylaxis whereas 46 (59.7%) of the adolescents could. In 51% of the dyad pairs, both the mother and daughter were able to correctly identify the need for antibiotic prophylaxis (N=37); the mother and daughter were both incorrect 26% (N=19) of the time. Age and level of education were not significantly associated with adolescents' ability to correctly identify their need for SBE prophylaxis and there was no association with self-reported discussions with health care providers about SBE prophylaxis. The ability to correctly identify the risk of transmission of a congenital heart defect was the most difficult for mothers and daughters: 51.9% (N=40) of the mothers and 35.1% (N=27) of the daughters could identify correctly their risk of transmission. The adolescents who correctly identified risk of fetal transmission reported more reproductive health care conversations with their health care provider (2.4 [SD=1.7] versus 0.8 [SD=1.2], $p<0.001$) and more conversations with mother (4.9 [SD=1.5] versus 3.5 [SD=2.0], $p<.01$).

Conclusion: Many adolescents with CHD were unable to identify their cardiac lesion, the need for SBE prophylaxis, or their risk of transmitting their lesion to a baby. However, mothers were better in self-reporting about their daughters' lesion, need for SBE prophylaxis, and risk of transmission. This raises concerns that as adolescents transition to other adult providers and become independent of their parents, they may not accurately report or understand their needs as they relate to their specific cardiac lesion. The association between adolescents who correctly identified information relating to their CHD and more frequent reproductive health care conversations, suggests that more frequent conversations between adolescents and providers and their mothers may be beneficial in imparting correct health information to adolescents. As more patients with congenital heart disease survive to adulthood and transition to adult providers, it is important to have direct conversations with these patients about their diagnosis, risk of fetal transmission, risk of pregnancy, and options for contraception early and during the teen years. Providers should also encourage mothers to have these conversations with their daughters, as we have demonstrated this is associated with more knowledge regarding these topics in the adolescent patient.

Indiana-ACC Poster Competition Abstract

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Title:

Safety and Feasibility of Rotational Atherectomy via Transradial Approach in Severe Coronary Calcification.

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Background

Severe coronary artery calcification is associated with worse clinical outcomes following PCI. Atherectomy strategies have been used to improve overall procedural success, facilitate stent delivery, promote maximal stent expansion and improve long term outcomes. Prior studies comparing transfemoral (TF) versus transradial (TR) approach have shown lower rates of vascular complications with TR PCI, but often with increased fluoroscopy time and contrast volume. We compared TF versus TR approach for rotational atherectomy (RA) with PCI at a single institution.

Methods

Between January 1, 2009 and May 31, 2017, we retrospectively reviewed clinical and procedural variables for 157 consecutive patients undergoing RA with PCI (133 TF and 24 TR). This represented 1.9% (157/8456) of all PCIs performed in our laboratory. Baseline patient characteristics, procedural variables, MACE rates (defined as cardiac death, myocardial infarction and target vessel revascularization) and vascular complications were compared. No patients crossed over between groups.

Results

There was no significant difference in procedural variables between groups; fluoroscopy time (TR 23.2 +/- 10.5 vs TF 26.8 +/- 11.7 min), contrast volume (TR 191 +/- 49 vs TF 211 +/- 88 mL), number of lesions treated (TR 1.7 +/- 0.8 vs TF 1.6 +/- 0.7) and total stent length for atherectomy vessel (TR 42.1 +/- 23.7 vs TF 38.1 +/- 20.1 mm); p values 0.2-0.6. Baseline patient demographics and vessel distribution were comparable. At 30 days, there was no significant difference in vascular (TR 0 vs TF 8 events, p = 0.6) or MACE rates (TR 0 vs TF 3 events, p = 1.0). Similarly, there was no statistical difference in 1 year vascular (TR 0 vs TF 15 events, p = 0.13) or MACE rates (TR 2 vs TF 20 events, p = 0.5). There was a trend towards a lower combined event rate in the transradial group at one year that was not statistically significant (TR 8.3% vs TF 26.3%, p = 0.068).

Conclusions

TR access is a safe and reasonable approach for RA with adjunctive PCI. Similar success rates, procedural variables, MACE and vascular complication rates were seen at 30 days and 1 year when comparing TR to TF. There was a trend for higher rates of vascular complications and combined event rate in the TF approach that did not reach statistical significance. Importantly, TR approach was not associated with increased fluoroscopy time or contrast volume in our study, despite similar number of lesions treated. These findings support that a “radial first” approach for rotational atherectomy is reasonable.

Indiana-ACC Poster Competition Abstract

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Fellow in Training
Case Abstracts

Indiana-ACC Poster Competition Abstract

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Title:

Using Cardiac Implantable Electronic Device to Noninvasively Perform Electrophysiology Study to Reduce Procedural Risk in a Patient with Right Atrial Lead Thrombus and Pulmonary Embolism

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction/objective: Electrophysiology study (EPS) and radiofrequency ablation are generally performed invasively using multiple catheters. Some patients with cardiac implantable electronic devices (CIEDs) may form mobile thrombus on their leads, and EPS carries an increased procedural risk of thrombus dislodgment. Using a patient's cardiac implantable electronic device to perform EPS allows a diagnosis to be made noninvasively and reduces the risk of thrombus dislodgement.

Case presentation: A 69-year-old female with medical history significant for sinus node dysfunction, dual-chamber (Medtronic) pacemaker in situ, and recently discovered pulmonary emboli and right atrial lead thrombus (Figure 1A) being treated with apixaban developed incessant, symptomatic supraventricular tachycardia (Figure 2A-B).

Discussion: Due to the incessant nature of her tachycardia and repeated emergency department visits and hospitalizations, a decision was made to proceed with electrophysiology study and ablation. In order to minimize the number of catheters within the heart, avoid dislodging patient's right atrial lead thrombus, and prevent interruption of anticoagulation therapy, electrophysiology study was performed noninvasively utilizing the patient's dual-chamber pacemaker.

EPS demonstrated 1:1 conduction over the slow pathway at pacing cycle lengths below 420 ms. Atrial extrastimulus pacing consistently demonstrated atrioventricular nodal echoes (Figure 3A). Ventricular burst pacing induced tachycardia with a V-A-V initiation (Figure 3B). The tachycardia exhibited a cycle length of 370 ms, a ventriculo-atrial time of 80 ms to the atrial electrode, and V-V intervals changes preceded and predicted A-A intervals. Ventricular overdrive pacing was performed through the device demonstrated a V-A-V response (Figure 3C). While performing a noninvasive EPS through the patient's CIED presented several limitations, the V-A-V response and V-V intervals predicting A-A intervals ruled out atrial tachycardia, and the delta VA interval of 180 ms during pacing versus during tachycardia and sustained conduction over a slow pathway favored atrioventricular nodal reentry (Figure 3C).

AV nodal slow pathway modification was undertaken with a 4 mm non-irrigated ablation catheter. This single catheter was advanced inside the heart only during the ablation to minimize the chance of dislodging thrombus (Figure 4). When junctional beats were elicited during power RF power delivery, artifacts made observation of retrograde conduction difficult and the patient's pacemaker was set to AAI mode to provide atrial pacing and monitor atrioventricular conduction (Figure 3D). Post ablation, repeat stimulation protocols with provocative medication testing using epinephrine revealed no evidence of sustained conduction over a slow pathway or inducible tachycardia. Ventricular burst pacing demonstrated intact VA conduction which was unchanged from baseline values. Post-operative echocardiography re-demonstrated the right atrial lead thrombus without change (Figure 1B).

Conclusion: In high risk patients with CIED in situ presenting with symptomatic supraventricular tachycardia, EPS can be performed noninvasively through the CIED to minimize intracardiac catheter manipulation and procedure time.

Indiana-ACC Poster Competition Abstract

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Title:

Left Ventricular Assist Device Outflow Graft Obstruction Treated with Percutaneous Trans-Catheter Stenting

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

INTRODUCTION

The prevalence of congestive heart failure (CHF) is estimated to be on the rise, affecting nearly 6.5 million Americans, a 50% increase when compared to estimates from nearly 20 years ago.[i-ii] Of those patients with advanced disease, the one-year mortality may exceed 50 percent.[iii] With the rapidly growing prevalence and high mortality rate, left ventricular assist devices (LVAD) have become a viable therapeutic option for advanced CHF given the limited number of organs for heart transplantation. However, LVADs carry risks of morbidity including infection, stroke, bleeding and device malfunction. Device malfunction often results from device thrombosis, malpositioining or outflow graft obstruction.[iv] Treatment typically includes surgical correction or urgent transplantation; however, we present a case of outflow graft obstruction treated with percutaneous trans-catheter stenting.

CASE PRESENTATION

A 74-year-old male with a past medical history of coronary artery disease status post coronary artery bypass surgery, ventricular tachycardia and atrial fibrillation s/p AV node ablation underwent Heartware (TM) LVAD implantation in 2014 as destination therapy. He did relatively well for approximately 4 years until he developed pre-syncopal symptoms associated with low flow LVAD alarms. Baseline speed was at 2700 RPMs with flows typically ~ 3 LPM, but upon presentation was found to have flows of <1LPM. Exam revealed signs of low output without hypovolemia. Laboratory work was notable for a therapeutic INR and acceptable LDH level of 264 U/L with a hemoglobin of 14 g/dL. An echocardiogram revealed increased ventricular dilatation from baseline, no pericardial effusion and appropriately positioned inflow cannula. CTA of the chest revealed significant obstruction at the outflow graft consistent with thrombus.

The patient was brought to the cath lab and femoral arterial access was obtained with a micro-puncture technique. After angiographic confirmation, the micro-puncture sheath was upsized to an 8-french short sheath. Selective angiography of the outflow graft was performed using a 6-french multipurpose diagnostic catheter and confirmed severe stenosis with a 70 mm gradient. A SupraCore wire was advanced across the stenosis. The 6-french catheter was exchanged for an 8-french multipurpose guide catheter and the stenosis was ballooned with a 5.0 x 40mm balloon expandable catheter. This was withdrawn and a 12.0 x 40mm Protégé self-expanding stent was deployed across the stenotic segment and then post dilated with a 10.0 x 40mm balloon catheter. Final angiography revealed no residual stenosis. Prior to angioplasty, the patient's blood pressure was 83/57 with a mean of 73 and post procedure, His blood pressure was 184/100 with a mean of 130. The patient was discharged the following day.

DISCUSSION

This illustrates a case of LVAD outflow graft obstruction that was successfully treated with a percutaneous approach. Outflow graft obstruction can decrease systemic perfusion resulting in multiorgan failure. This typically manifests with decrease mean arterial pressure, increased LVAD power and requires an increase in speed to compensate. Diagnosis relies on high clinical suspicion and CT imaging to verify obstruction within the outflow graft.

Previous case reports describe percutaneous techniques to remedy other LVAD complications. Parikh et al described a case of severe aortic insufficiency after implantation of a LVAD that was treated with percutaneous aortic valve closure to prevent regurgitant flow.[v] Additionally, Potapov et al evaluated recovery of myocardial function in an LVAD patient by performing a percutaneous balloon occlusion of the outflow graft.[vi] These percutaneous advancements naturally progressed to Retzer et al's description of the first successful percutaneous treatment of LVAD outflow graft stenosis.[vii] Our case adds to the growing body of literature that confirms the feasibility of treating LVAD outflow graft stenosis with a percutaneous approach.

Indiana-ACC Poster Competition Abstract

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Title:

A fatal case of flecainide toxicity: the role of a potential pharmacokinetic drug interaction with paroxetine.

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction/Objective

Flecainide is a Vaughan-Williams Class I-C, lipophilic antiarrhythmic agent that blocks sodium channels, thereby inhibiting phase 0 depolarization. Flecainide is metabolized in the liver by the CYP2D6 enzyme, has a long half-life and high oral bioavailability. Thus, inhibitors of CYP2D6 can result in increased circulating levels of flecainide. We present a rare case of fatal refractory cardiogenic shock due to flecainide toxicity from a possible drug interaction with paroxetine.

Case Presentation

62 year old African American female with a history of alcohol abuse, depression, typical right atrial flutter status-post ablation, and paroxysmal atrial fibrillation (AF) was admitted for electrolyte abnormalities and ECG abnormalities with concomitant symptoms of malaise and lightheadedness. In the month prior, she was started on flecainide for episodes of symptomatic AF. Due to continued AF episodes, the flecainide dose was gradually titrated up to 150 mg po BID. Her medications also included paroxetine 30 mg daily and diltazem ER 180 mg daily. Her ECG on admission showed a RBBB and a prolonged corrected QT interval (both were new). Her electrolytes were corrected, her QTc shortened. On day 2, her paroxetine was discontinued and replaced with sertraline and flecainide dose was increased to 200mg po BID. On day 5, her telemetry showed intermittent episodes of sinus bradycardia with competing accelerated junctional rhythm, RBBB, intermittent PVCs and episodes of NSVT. Later that day, the patient became hypotensive, had significant QRS widening and developed rapid, progressive shock refractory to multiple vasopressors and inotropic agents. Rhythm was unchanged. Initial metabolic panels, CT scans of head, chest, abdomen, and pelvis were unremarkable. A stat transthoracic echocardiogram revealed significant inter- and intraventricular dyssynchrony, with severe right ventricular dilation and severe tricuspid regurgitation. After careful review of laboratory and imaging studies ruled out alternative etiologies, a working diagnosis of flecainide toxicity was made. Toxicology was consulted; the patient received sodium bicarbonate (4 doses of 50 mEq of 8.4%) resulting in transient narrowing of the QRS complex. A sodium bicarbonate infusion was started with no further response. Thereafter, a 20% Intralipid fat emulsion infusion was initiated. Nephrology made plans to place emergent dialysis access and VA-ECMO was being considered for refractory shock. However, at this juncture, per family wishes, the decision made to withdraw care and patient passed shortly afterwards. The flecainide level results (3.84 mcg/ml [ref. range 0.2 -1.00 mcg/ml, toxic > 1.5 mcg/ml]) returned after she passed away. Drug interaction with paroxetine was felt to have played a significant role in the elevated serum flecainide levels.

Discussion

Awareness of flecainide toxicity and recognition of signs of toxicity, namely development of QRS widening and new bundle branch block, is paramount in avoiding life-threatening results. Severe QRS widening and ventricular dysfunction associated with hemodynamic collapse should prompt evaluation for ECMO support. Flecainide toxicity has high mortality due to rapid onset of shock, unfavorable pharmacokinetics, poor clearance, and poor efficacy of temporaizing measures including sodium bicarbonate and lipid emulsion infusions. Flecainide toxicity is rare; nearly all reported cases are secondary to overdose. Only one case of delirium that was felt to be related to flecainide toxicity (plasma level 1.3 ug/ml) resulting from a pharmacokinetic drug interaction with paroxetine by Taso Y. et. al. has been reported. Based on the clinical course of our patient a metabolic drug interaction between flecainide and paroxetine was felt to be at least a partial cause for this toxicity and resultant shock.

Conclusion

Flecainide toxicity is rare, but can be fatal and has limited management options. Paroxetine inhibits the CYP2D6 enzyme and can cause elevated circulating levels of flecainide. Signs of flecainide toxicity include cardiac conduction slowing resulting in QRS widening and bundle branch block. Because of the risk of toxicity, flecainide dosing adjustments should be made and plasma concentrations should be monitored closely when prescribed with CYP2D6 inhibitors such as paroxetine.

Indiana-ACC Poster Competition Abstract

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Title:

A Rare Case of Acute Simultaneous Cardio-Cerebral Infarction with Review of Mechanism of Onset and Optimal Management

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction:
Acute myocardial infarction (AMI) and acute ischeic stroke (AIS) are medical emergencies where timely recognition and prompt treatment have been shown to successfully decrease morbidity and mortality. Each have a short therapeutic window in which optimal results can be achieved. Sometimes, one may precede the other. Rarely, they occur simultaneously. Patients presenting with acute cardio-cerebral infarction (CCI) require complex and urgent decisions. This report examines a case of a patient who presented with acute CCI complicated by cardiogenic shock and her management.

Objective:
1. Discuss the mechanisms that can lead to simultaneous CCI.
2. Evaluate management of simultaneous CCI and review consensus guidelines.

Case Presentation:
A 58 year old female presented to a community hospital after being found down. ECG showed ST elevations in the anterolateral leads. During consent, patient was found to be altered and displayed a left-sided weakness. Catheterization was deferred and CT angiography was performed showing acute thrombosis of her basilar artery. She was outside the window for thrombolysis with tPA and was transferred to a tertiary care center for mechanical thrombectomy. Post-thrombectomy, she had limited neurologic function. ECG showed continued ST elevations concerning for an anterior STEMI. Cath lab activation was deferred due to neurology stating any anticoagulation or antiplatelet therapy was contraindicated given recent ACI and limited neurologic function. Echo showed reduced systolic function with EF 24% and global hypokinesis concerning for Takotsubo cardiomyopathy. Contrast revealed a large, sessile echogenic mass concerning for apical thrombus. After thrombus discovery, patient was cleared for a heparin drip. She regained the ability to follow commands. Unfortunately, she subsequently went into cardiogenic shock requiring multiple vasopressors and ultimately an intra-aortic balloon pump (IABP) by hospital day 2. Diagnostic catheterization was performed at that time showing 100% occlusion of proximal LAD along with 40% stenosis of proximal LCx and 40% stenosis of the mid RCA. Vasopressors were weaned but patient remained dependent on IABP. She was not an advanced heart failure candidate due to recent stroke. There was discussion to assess viability of her LAD territory and perform a PCI of her occluded LAD to increase her cardiac function. Unfortunately, on hospital day 8, patient developed hemorrhagic conversion of her ischemic stroke and ultimately was switched to comfort care after which she passed away.

Discussion:
Only case reports of simultaneous CCI have been documented as most instances are metachronous with one infarction preceding the other. Various mechanisms have been identified that could cause a simultaneous CCI. One mechanisms that could have led to this patient's presentation include AMI causing akinesis of the myocardium leading to acute thrombus formation and subsequent embolizaiton to cause AIS. Another is chronic heart failure leading to LV thrombus formation that could have dislodged and embolized to both coronary and cerebral arteries. A third would be an AIS causing an adrenergic surge leading to catecholamine-induced stunning and Takotsubo síndrome. This patient had not seen a physician in years and had multiple episodes of chest pain the year prior to presentation. She may have developed heart failure and LV thrombus with simultaneous embolization. Concerning management of simultaneous CCI, thrombolytic therapy is indicated in both AMI and AIS but the dosing and length of infusion are different for each. Mechanical intervention with either percutaneous coronary intervention (PCI) or mechanical thrombectomy of the cerebral arteries are high risk given the inherent instability of the patient. Therapy post-procedure can be difficult as well as dual anti-platelet therapy increases risk of bleeding after mechanical thrombectomy and tPA increases bleeding complications with PCI. Literature shows multiple case reports with different interventions and outcomes ranging from death to mild disability. A consensus statement from the American Heart Association and American Stroke Association advocates IV-tPA for AIS treatment followed by PCI of the coronary arteries if indicated (Class IIa, Level C) though no comment is made on DAPT post-procedure.

Conclusion:
Simultaneous CCI is a complex and difficult medical emergency that requires prompt recognition and collaboration between cardiologists and neurologists. Current recommendations are to administer IV-tPA for AIS followed by PCI of coronary arteries for AMI.

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Please structure your case study abstract using the following headings: * Introduction/objective * Case presentation * Discussion * Conclusion

Title:

An Uncommon Cause of Fulminant Myocarditis: Influenza B.

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction
Influenza remains a frequent cause of hospitalization and death during the winter season. Since early in March of 2018, influenza B has been more frequently reported than influenza A. While the incidence of myocarditis from influenza is rare (from 0-11%), the severity of illness can include fulminant myocarditis. In cases of fulminant myocarditis, influenza A is more frequently reported in adults. We describe a case of fulminant myocarditis from influenza B.

Case Description
A 52-year old female with a past medical history of hypertension presented to urgent care with productive cough, fever, chills and myalgias. A diagnosis of acute bronchitis was made, but despite treatment with azithromycin, her symptoms progressively worsened over of the next 2 weeks. Subsequent admission to the hospital for profound dyspnea and chest pain revealed an electrocardiogram showing diffuse ST elevation with PR depression and with transthoracic echocardiogram revealing normal biventricular size with severe biventricular dysfunction and a small pericardial effusion. Progressive hypotension and multi-organ failure ensued, and her cardiogenic shock deteriorated further despite temporary circulatory support with an Impella 2.5, requiring eventual placement of venoarterial extracorporeal membrane oxygenation (VA ECMO) support. Admission labs revealed a positive influenza B PCR, consistent with a diagnosis of influenza B myopericarditis. She was also treated with IV Peramivir. After VA ECMO support, continuous venovenous hemodialysis and intensive supportive care for a total of 10 days, her biventricular failure completely recovered, and she tolerated decannulation of VA ECMO support.

Discussion
Myocarditis remains a common, often unrecognized cause of dilated cardiomyopathy worldwide. Fulminant myocarditis is characterized by new, profound ventricular dysfunction causing significant hemodynamic instability, often requiring mechanical circulatory support. Lymphocytic myocarditis from viruses remains the most common cause, which typically includes Parvovirus B19, Human Herpes Virus 6 and Coxsackie. Influenza myocarditis is an uncommon cause of fulminant myocarditis with influenza A being the usual culprit, with very few case reports of influenza B causing fulminant myocarditis. In a 2011 review of cases of myocarditis, 3/30 (10%) of the cases were from influenza B. A pathology review published in the Journal of Infectious Disease looking at Center for Disease Control reported deaths from dying influenza B between 2000 and 2010, myocardial injury was seen in 20/29 of cases, with 76% under the age of 18 years old.

Conclusion
Our case is an example of an uncommon, but important cause of fulminant myocarditis with very few case reports in adults. However, with an increasing prevalence of influenza B, particularly later in influenza season, heightened awareness of the potential sequelae are important to institute early supportive treatment.

Indiana-ACC Poster Competition Abstract

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Title:

***Fusobacterium species* Pyopericardium Originating from Odontogenic Source**

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction

Bacterial pericarditis, though a rare clinical entity, is rapidly fatal if not recognized quickly[1]. Infection usually arises following instrumentation of the pericardium or from trauma, though it can also spread from any previous supra-diaphragmatic infection[1]. Typical organisms involved include staphylococcus and streptococcus species in addition to oral flora[1]. Here, however, we report a rare care of pyopericardium caused by *fusobacterium species* spreading from a retropharyngeal abscess.

Case Presentation

21 year-old male with recent dental work presented with subacute symptomatology of fevers and emesis then acute chest pain, dyspnea, tachycardia, hypotension, and significant laboratory derangements. Bedside echocardiogram confirmed tamponade and ECG consistent with pericardial disease pattern. Urgent pericardiocentesis yielded 860mL gross purulence requiring subsequent pericardial window. Cultures grew fusophorum nucleatum, fusophorum necrophorum, and streptococcus species. CT imaging revealed subcutaneous emphysema in the head and neck region requiring drainage. Antibiotic treatment and drainage ongoing with ampicillin-sulbactam.

Discussion

Though pericarditis is the most common form of pericardial disease, with 90% of cases classified as “idiopathic” and often presumed viral, purulent pericarditis comprises <1% of cases in Western Europe and North America[2]. Worldwide, the most common pathogen identified is Mycobacterium tuberculosis, though this is less common in Western Europe and North America. Here, we present a case of pericarditis with pyopericardium from an atypical pathogen attributed to an odontogenic source due to the patient’s history on presentation.

Conclusion

High index of suspicion should be held in individuals for atypical pathogens in light of the degree of severity of pyopericardium that can develop.

Indiana-ACC Poster Competition Abstract

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Title:

The Silence of Ischemic Mitral Regurgitation

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction: Mitral regurgitation (MR) following myocardial infarction is often unrecognized and clinically silent. This condition tends to have a worse prognosis with increased risk of heart failure and death. It is also more common than acute MR secondary to a ruptured papillary muscle, which is rare and often fatal.
Objective: Review the clinical examination and management of ischemic mitral regurgitation.
Case presentation: A 60-year-old woman with a history of CAD status post PCI of her RCA for acute thrombosis one year ago presented to the ED because of progressive dyspnea for four weeks, accompanied by weight gain and lower extremity swelling. The physical exam revealed the patient was hemodynamically stable but with bilateral rales, 2/6 holosystolic murmur at the apex, and pitting edema. Treatment was initiated with intravenous diuresis. A doppler echocardiogram showed a severe MR jet with posterior wall remodeling. The patient subsequently underwent a right and left heart catheterization, which revealed a large V-wave on the PCWP tracing and 100 percent chronic occlusion of the mid RCA. After continued medical therapy, a transesophageal echocardiogram was obtained which revealed a mitral regurgitant volume of 68 mL with a tethered posterior leaflet. The patient remained nearly symptom-free on discharge and given appropriate follow up to discuss mitral valve surgery.
Discussion: This case illustrates the importance of doppler echocardiography in ischemic MR and the prudence for clinicians to confirm the mechanism. Prevalence is not well defined because of the heterogeneity of MR. TEE is helpful in excluding organic leaflet pathology. This patient's MR was discovered in the chronic phases of infarction and was likely due to remodeling of the posterior wall after the RCA infarct. Risk factors for ischemic MR include advanced age, female gender, and patients are more likely to present with heart failure. Cardiac auscultation cannot be relied upon to diagnose or assess the severity of ischemic MR. Primary surgical approaches for ischemic MR include MV annuloplasty, repair, or replacement +/- at the time of coronary bypass. A lack of randomized controlled trials has yet to identify the best treatment.
Conclusion: Recognition of ischemic MR is critical as it often presents silently in the setting of heart failure and is associated with an increased risk of cardiovascular mortality. The treatment of choice is often guided by the experience of each center and alternative treatments may need to target the role that left ventricular remodeling plays in ischemic MR.

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Title:

Putting Out the Fire: Epicardial Ablation for the Management of Symptomatic Brugada Syndrome

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction: An inherited ion channelopathy characterized by ventricular tachyarrhythmias and increased risk of sudden cardiac death, Brugada syndrome (BrS) is a challenging condition with limited therapeutic options. Implantable cardioverter defibrillator (ICD) implantation remains the mainstay therapeutic with the greatest supportive evidence. However, catheter ablation strategies, while still considered experimental treatment, have been reported more recently for the management of symptomatic BrS.

Objective: We aim to present the experience and success with catheter ablation strategies for symptomatic BrS at our institution via two clinical cases.

Case Presentations:
A 25-year-old man with a history of BrS status post subcutaneous-ICD (S-ICD) placement presented with ventricular tachycardia (VT) storm. Prior to hospital arrival, the patient suffered loss of consciousness and VT cardiac arrest, receiving twenty-six shocks from his device. Patient ultimately underwent an epicardial catheter ablation of the right ventricular outflow tract (RVOT) and right ventricle (RV). VT was no longer inducible at the end of the ablation case. One week later, the patient had his S-ICD removed and received a dual-chamber ICD with an extra-coil in the coronary sinus to reduce defibrillation threshold. At discharge the patient was started on quinidine 300mg BID and found to be positive for the *SCN5A* gene mutation. Since that time, patient has been followed closely for over 1.5 years without any recurrence of atrial or ventricular arrhythmias. He remains asymptomatic and has been able to return to work.

A 37 year-old-man with a personal history of BrS along with a family history of sudden death and BrS drug-induced phenotype presented with syncope and multiple appropriate ICD shocks for ventricular fibrillation (VF). Quinidine was initially prescribed and the patient was without recurrence of ventricular tachyarrhythmias or syncope events while on quinidine. However, the patient soon developed profuse diarrhea attributed to quinidine therapy. With the hopes of eliminating quinidine therapy, the patient pursued RV and RVOT epicardial ablation. Extensive ablation of fractionated and late activation areas throughout the epicardial RV was successfully completed. Two-month follow up reveals no recurrence of ventricular arrhythmias, syncope, or ICD shocks, and the patient remains off quinidine therapy.

Discussion: BrS is diagnosed based off clinical features along with the electrocardiogram (ECG). Majority of patients with BrS initially present with syncope or pre-syncope, yet some patients may be asymptomatic. The final diagnosing key is ECG changes in the right pericardial leads whether fleeting or constant changes. There are three types of BrS with only type I being diagnostic for BrS, whereas type II and III are suggestive not diagnostic. Type I shows a coved-type ST-segment elevation greater than 2mm, followed by a descending negative T wave in at least one right precordial leads (V1-V3). Since the identifying BrS, there have been 19 genetic variants and over 300 mutations associated with the syndrome, the most common mutation located on the *SCN5A* gene. *SCN5A* is the loss of function at the cardiac sodium channel and 20-25% of patients with BrS have this mutation, classifying as BrS type I. Effective treatment for BrS has been proven primarily only with ICDs, despite the disadvantages of ICDs (multiple device replacements, inappropriate shocks, etc). There are currently two drugs, isoproterenol and quinidine, that have been useful in the treatment for BrS. More recently catheter ablation of ventricular ectopy has been thought to treat BrS after demonstrating that VF may be triggered similar to ventricular ectopy.

Conclusion: Catheter ablation management strategy for symptomatic BrS was first reported in a series of 3 patients in 2003. Since then, multiple case reports and case series have been published on this topic. While ablation therapy in this population remains inconclusive and is still considered experimental, the limited evidence thus far suggests that ablation is a reasonable strategy for some BrS patients.

Indiana-ACC Poster Competition Abstract

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Title:

Fulminant heart failure in AL amyloidosis: from bland cardiac work-up to cardiogenic shock in 7 days

Abstract: (Your abstract must use Normal style and must fit into the box. You may not alter the size of this)

Introduction: Estimated AL amyloidosis incidence is 9 cases per million per year, and 50-70% of patients have cardiac involvement (1). One year mortality in AL cardiac amyloidosis (CA) is about 45%, which is significantly worse than in ATTR CA (2,3). Classic CA is characterized by restrictive physiology and conduction abnormalities. Echo findings include increased LV wall thickness, diastolic dysfunction, and depressed longitudinal and radial strain with apical sparing (3). A recent single-center study of 46 CA patients referred to CCU for acute heart failure showed that majority of deaths occurred in patients who presented with cardiogenic shock and those with AL amyloidosis (4). Most died within 3-9 days of being started on catecholamine infusion. Mean baseline EF of AL CA patients admitted with cardiogenic shock was 41+/-13%, and mean time between AL CA diagnosis and admission was 224+/- 557 days. Most case reports (5-8) of fulminant heart failure in AL CA describe patients with suggestive echo/EKG findings on initial presentation, such as myocardial hypertrophy and diastolic dysfunction. The following case demonstrates rapid progression to cardiogenic shock in a patient whose initial work-up was reassuring and whose new diagnosis of cardiac amyloidosis became a surprise.

Case presentation: 77 year old male with ESRD on hemodialysis for 8 years, diabetes, hypertension was admitted for gradually worsening dyspnea for 1 week. Patient had excellent functional status prior to the onset of symptoms. His troponin I and BNP were mildly elevated. TTE was remarkable for elevated RVSP 40-45 mmHg. CT chest was negative for PE. Stress SPECT showed a large, mostly fixed perfusion defect in the left circumflex distribution. Coronary angiography showed luminal irregularities in the left circumflex and otherwise normal coronaries. LVEDP was 26. The same evening, patient became increasingly dyspneic, suffered PEA arrest, and was transferred to ICU on BiPAP and in shock. TTE showed severe global LV hypokinesis. Within 24 hours, he developed new persistent chest pain and sustained monomorphic VT. He was started on IV amiodarone and was intubated for respiratory distress. Telemetry showed intermittent idioventricular rhythm and atrial fibrillation with slow ventricular rate. Repeat heart catheterization showed no new coronary lesions and confirmed low cardiac index. IABP was placed. Patient received high dose steroids for shock and for empiric treatment of myocarditis. EMB showed extensive amyloidosis and myocyte hypertrophy; no evidence of myocarditis was seen. Infectious myocarditis work-up was negative. SPEP with immunofixation showed monoclonal IgG lambda protein. Over the next 3 days, pressors were nearly completely weaned, and repeat TTE showed EF 53%. IABP was removed. Later that evening, patient decompensated and suffered PEA arrest, shortly followed by asystole. Blood cultures drawn the night prior grew S. aureus. EMB liquid chromatography and tandem mass spectrometry identified AL-type protein after the patient's demise.

Discussion: This case illustrates precipitous heart failure in a newly diagnosed AL cardiac amyloidosis (CA). Clinical course was most consistent with myocarditis, which is unusual for patients with CA. It is also unusual that, prior to patient's decompensation, there was little evidence of extensive infiltrative process in the heart. Two case reports of CA with myocarditis-like presentation describe a variant of AL amyloidosis that caused myocardial injury due to amyloid deposition in the small intramural blood vessels (9,10). This variant of AL CA may be more common than we realize. Interestingly, our patient seemed to at least partially recover from cardiogenic shock before succumbing to hospital-acquired septicemia. Atypical stress cardiomyopathy after cardiac arrest could have contributed to the severity and partial reversibility of his heart failure. While no other imaging modality was available at the time, additional information from strain or cardiac MRI could certainly have aided in the diagnosis. Multiple case reports describe fulminant heart failure in AL amyloidosis (5-10), but little is known about how quickly objective markers of heart failure and myocardial injury evolve from the onset of symptoms in such cases. Our patient's trajectory underscores how difficult cardiac amyloidosis can be to diagnose and how easily one can be misled by a reassuring initial cardiac work-up.

Conclusion: Cardiac amyloidosis, especially AL form, can present as fulminant heart failure preceded by subacute, gradually worsening exertional dyspnea. Clinicians should pay close attention to systemic symptoms and signs of amyloidosis if clinical heart failure is suspected, but initial work-up does not reveal obvious cardiomyopathy or significant coronary artery disease.

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