Indiana-ACC 2014 Poster Competition Abstracts

CV Team Abstracts

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:

Real World Experience with Metric #37: Retrospective analysis of Post PCI bleeding rates in a state wide healthcare system.

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

Metric #37, Risk Adjusted Bleeding, defines bleeding as: Any of the following occurring within 72 h after PCI or before hospital discharge (whichever occurs first): site-reported arterial access site bleeding, which may be either external or a hematoma >10 cm for femoral access, >5 cm for brachial access, or >2 cm for radial access; retroperitoneal, gastrointestinal, or genitourinary bleeding; intracranial hemorrhage; cardiac tamponade; post-procedure hemoglobin decrease of 3 g/dl in patients with a pre-procedure hemoglobin level <=16 g/dl; or post-procedure nonbypass surgery-related blood transfusion for patients with a pre-procedure hemoglobin level >=8 g/dl.

Background: Initial observed performance outcomes at IU Health's largest institution, Methodist Hospital, were greater than desired for post-PCI risk adjusted bleeding events. The quality improvement team reviewed individual records that were classified as having a bleeding event in National Cardiovascular Data Registry (NCDR®) CathPCI Registry® Metric #37 and found unexpected local trends in 2011 data. Frail, elderly women were not the majority of the observed post-PCI bleeding events; this population accounted for approximately 12% of the post procedural events. Unexpectedly, males under the age of 70 comprised over 50% of the post PCI bleeding events. Another unforeseen observation was that over 22% of post procedural bleeding events were reported with radial/ulnar access sites.

Objective: In an effort to improve safety, a retrospective review of the statewide healthcare system data to understand why bleeding rates were higher than expected.

Methods: The team analyzed 3,701 records within the health system with PCI procedures in 2012. Among the multiple variables analyzed, major surgery during the same episode of care as the PCI ($t_{(78)}$ = 3.73, p=0.000) was statistically significant. Of the bleeding, 53% occurred in males. Four anticoagulation therapies were independently analyzed and two were found to be statistically significant – Bivalirudin and IIbIIIa. Approximately one half of the bleeding events by definition were attributed to patients who received RBC transfusion. Of that subset, 58.7% (37/63) were anemic at the time of transfusion without evidence of overt bleeding. In the statewide analysis, 67.0% (63/94) of the RBC transfusions were not associated with a hemoglobin drop or other defined bleeding event. Approximately one half of the bleeding events by definition were attributed to patients who received RBC transfusion. Of that subset, 58.7% (37/63) were anemic at the time of transfusion without evidence of overt bleeding. In the statewide analysis, 67.0% (63/94) of the RBC transfusions were not associated with a hemoglobin drop or other defined bleeding event.

The quality improvement team utilized multidisciplinary team education as the best approach to impact post-PCI bleeding rates. This was completed through the following approaches:

1. RBC transfusion awareness and guideline adherence
2. Cultural education of vascular injury and post-PCI observed bleeding
3. Reporting physician specific trends in scorecards
4. Routine organizational reporting in monthly process improvement meetings

Standardized approach to post PCI care through nursing education of vascular injury and post-PCI Blood transfusion

Results: The raw number of post-PCI observed bleeding events continues to decrease at Methodist Hospital. Preliminary performance illustrates a **40%** decrease in events from CY2012 compared to CY2013 (2012= 96, 2013= 58). Continued improvement is observed in published Risk Adjusted Bleeding performance: CY2011= 6.51%, CY 2012= 5.84%, 2013= 4.8%* (*performance includes metric adjudication). During 2013, there was an observed decrease in RBC transfusion at Methodist. The entire PCI population observed a 40% decrease in post-PCI transfusion (2012=75, 2013= 45). There was a 94% decrease in patients transfused when their hemoglobin values were greater than 8 grams (2012=32, 2013=2).Cost Avoidance: 40% Improvement ($41,745) in post-PCI RBC transfusion. An observed 96% Improvement ($59,834.50) in post-PCI RBC transfusion with Hgb values >8.

Conclusion: Since Metric #37 was introduced by the NCDR, significant process improvements and transparencies have changed the way post-PCI care is provided. Metric #37 brought to light varying RBC transfusion practices and increased awareness of post-PCI bleeding events. Standardizing practices, evidence based medication education, increased multidisciplinary awareness are essential for these improvements. Although great gains have been made, there remains opportunity for future projects. The team is currently developing an anticoagulation algorithm and investigating causes for unexplained hemoglobin decreases post-PCI.

Indiana-ACC Poster Competition Abstract

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Fellow in Training Research Abstract

Indiana-ACC Poster Competition Abstract

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Title:

Bleeding Risk Assessment and Bleeding Reduction Strategies for Percutaneous Coronary Intervention

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

Introduction: Bleeding is the most common noncardiac complication in patients undergoing percutaneous coronary intervention (PCI) and is associated with increased morbidity, mortality, and costs.[1] The direct thrombin inhibitor bivalirudin has been shown to decrease PCI-associated bleeding when compared to heparin plus glycoprotein IIb/IIIa inhibitors.[2-4] A clinical algorithm developed from the National Cardiovascular Data Registry has been shown to accurately stratify patients' risk of PCI-associated bleeding.[5] Using bivalirudin to facilitate PCI in patients at higher risk of bleeding should therefore reduce bleeding complications.[6]

Methods: Interventional cardiologists at St Vincent Indianapolis Hospital and the St Vincent Heart Center were encouraged to formally assess their patients' bleeding risk prior to PCI using a previously validated scoring tool.[5] Use of bivalirudin (instead of heparin) for anticoagulation during PCI was recommended in patients with a high bleeding risk score. Data on bleeding risk score, anticoagulant choice, and bleeding complications were collected on all PCI patients from July 1st – October 31st 2013. Bleeding complications during the study period were compared to historical controls from the previous year.

Results: 666 patients underwent PCI at our institutions during the study period. Forty percent of these patients had a bleeding risk score assessed and recorded prior to PCI. In low risk patients, bivalirudin was used for anticoagulation in 9% of cases. Bivalirudin use increased to 26% and 52% in intermediate and high risk patients respectively ($p < 0.0001$). The rate of major bleeding associated with 2533 PCIs at our institution in the year prior to this study was 0.9%. This rate decreased to 0.3% during the study period, showing a trend towards reduced bleeding complications ($p = 0.098$).

Discussion: We were able to demonstrate that formally assessing patients' bleeding risk prior to PCI influences anticoagulation choice with bivalirudin being used more often in patients at higher risk of bleeding complications. Increased use of bivalirudin in high risk patients was associated with a 68% relative risk reduction in bleeding complications during the study period. Our study was limited by a bleeding risk assessment being voluntarily performed in only 40% of PCIs during the study period, an area targeted for future improvement.

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Indiana-ACC Poster Competition Abstract

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Fellow in Training

Case Abstracts

Indiana-ACC Poster Competition Abstract

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Title:

Combined right and left cardiac valve dysfunction requiring quadruple valve replacement in a patient with carcinoid heart disease and PFO.

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

Introduction:
Carcinoid tumors are relatively rare, slow growing neuroendocrine tumors most frequently arising from the midgut structures (small bowel, appendix, proximal colon, etc.). Metastatic lesions most commonly involve the liver. The tumors are capable of endogenous secretion of a number of bioactive substances (serotonin, kallikrein, etc.) which are responsible for the predominant clinical side effects. Resulting “carcinoid syndrome” is most commonly marked by vasomotor symptoms, gastrointestinal hypermotility and bronchospasm. Cardiac involvement is relatively common (from 20-70% of cases). Deposition of plaque like fibrous tissue on the cardiac valves and extravalvular structures can lead to abnormal valve functioning and in rare cases a secondary restrictive cardiomyopathy (cardiac fibrosis). Right sided cardiac valves are most commonly effected though in rare circumstances (<10% of cases) left sided valves can be. This is typically seen in the setting of an atrial right-to-left shunt or primary bronchial tumor.

Case Presentation:
A 67 year old female with previously diagnosed carcinoid and metastatic liver involvement was admitted for evaluation of dyspnea, fatigue and lower extremity edema. Symptoms had been progressive for the past 3 months and also included abdominal bloating, diminished appetite and weight loss. Her carcinoid disease had been symptomatically well controlled with somatostain analogue therapy. As part of an outpatient evaluation by her oncologist the patient had recently undergone restaging of her disease with a CT scan. She was also referred for an echocardigram to evaluate a murmur. Multiple cardiac valve abnormalities were noted. Just prior to her hospitalization she was referred to a cardiologist.

Physical examination was notable for the presence of jugular venous distention, associated hepatojugular reflux and mild pitting lower extremity edema. Auscultation revealed holosystolic murmurs at the left lower sternal border and apex of 3/6 intensity, both of which increased with inspiration.

TEE demonstrated evidence of RV volume overload but preserved LV function. All four cardiac valves demonstrated abnormal thickening of the leaflets with reduced mobility/excursion. Severe valvular insufficiency was noted for the aortic, pulmonic and tricuspid valves. A PFO was detected with the presence of right-to-left shunt. Cardiac catheterization disclosed moderate non-obstructive coronary disease, pulmonary hypertension and an elevated right atrial pressure. Findings were all consistent with carcinoid heart disease.

Discussion:
Carcinoid heart disease is the end result of damage caused by biochemically active substances secreted by carcinoid tumors. It most commonly involves the tricuspid and pulmonic valves though in rare instances can effect left sided valvular structures. The condition is progressive and often leads to right ventricular heart failure. The presence of carcinoid heart disease carries a generally poor prognosis with a median survival of 11 months. It is a major source of morbidity and mortality. Medical therapy revolves around symptom control and maintaining euvolemia. Cardiac surgery with valve replacement can successfully relieve symptoms, improve functional class and may afford survival benefit. Perioperative rates of mortality/complications remain somewhat high though overall operative mortality has declined through the years. Candidate selection, timing and choice of valve prosthesis remain controversial.

Conclusion:
The patient was initially managed with conservative medical therapy and responded well to diuretics. Due to the severe nature of her valvular disease consultation by a cardiovascular surgeon was obtained. After discussion the patient elected to proceed with definitive operative management. She underwent successful quadruple valve replacement with PFO closure. Bioprosthetic valves were utilized in each position. The patient’s postoperative course was complicated by difficulty weaning from the ventilator and complete heart block which required insertion of a permanent pacemaker. She was eventually discharged for ongoing care at a long term acute care facility.

Indiana-ACC Poster Competition Abstract

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Title:

Fulminant heart failure and incessant ventricular tachycardia secondary to Giant Cell Myocarditis

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

Introduction: Giant cell myocarditis is a rare cause of fulminant congestive heart failure. It can be associated with significant arrhythmias, and if not treated aggressively, it typically results in death within a few months[1]. There is some correlation with other autoimmune diseases, but no direct cause has been found[1]. It is typically a disease of young adults with average age of approximately 42 years at the time of presentation[1]. This is a clinically distinct and particularly aggressive form of myocarditis that must be differentiated from other syndromes.

Case Presentation: A 59 year old male with history of chronic lymphocytic leukemia (not currently receiving chemotherapy) presented in late January to a facility in southern Indiana with progressive dyspnea on exertion, fatigue, orthopnea and weight gain. At baseline, the patient was active, independent, and had no limitations prior to initial presentation. He denied other chronic medical problems. He was a non-smoker, and he was not taking any prescription or herbal medications. There was no family history of cardiomyopathy or sudden cardiac death. Prior to initial presentation, he reported a 2 week history of upper respiratory symptoms and a flu-like syndrome.

Initial echocardiogram demonstrated a severely depressed LV systolic function with severe global hypokinesis. Ejection fraction was estimated at approximately 20%. A diagnostic cardiac catheterization was performed which demonstrated angiographically normal coronary arteries. He was placed on an appropriate heart failure regimen, diuresed and discharged home. Over the next month, he had multiple recurrent hospitalizations despite escalating care and reported medication compliance. In addition, he developed frequent episodes of non-sustained ventricular tachycardia and atrial flutter. A biventricular ICD was implanted. Despite these therapies, his clinical condition continued to rapidly deteriorate, and he was transferred to a tertiary care center for incessant ventricular tachycardia and cardiogenic shock.

Physical exam demonstrated a diffusely anasarcic male. He was tachypneic and requiring supplemental oxygen. He had significantly elevated JVD with a narrow pulse pressure. His extremities were cool and mottled. His initial laboratory evaluation demonstrated acute renal failure, hepatic congestion and a significantly elevated BNP. Portable chest x-ray showed cardiomegaly with pulmonary vascular congestion and bilateral pleural effusions.

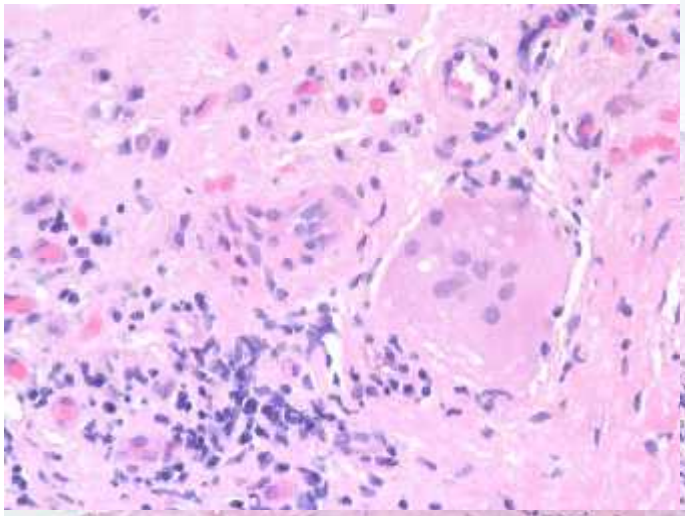
He was placed on inotropic support, high dose diuretics and IV amiodarone and lidocaine. A swan-ganz catheter and intra-aortic balloon pump were inserted. Given his fulminant course, the differential was lymphocytic myocarditis secondary to viral infection versus Giant cell myocarditis. An endomyocardial biopsy were performed. Pathology demonstrated acute and chronic inflammation with occasional giant cells suggesting Giant cell myocarditis. He was treated with high dose IV steroids and IV cyclophosphamide. Unfortunately, his clinical condition continued to deteriorate. He was offered mechanical circulatory support and possible cardiac transplant evaluation; however, he declined. He ultimately passed a few days later.

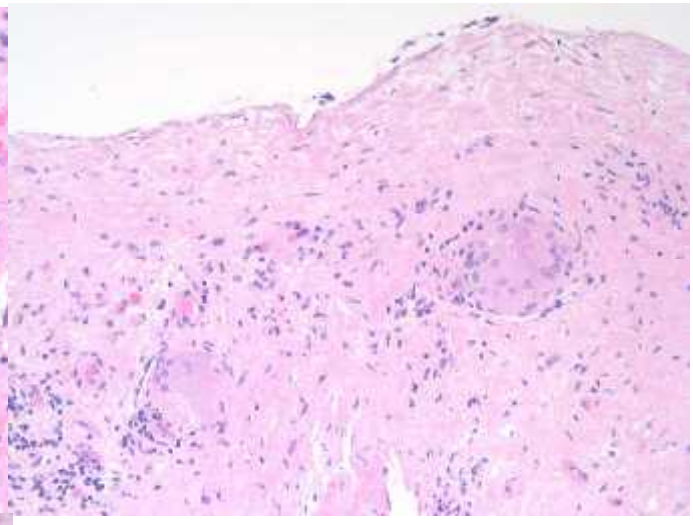
Discussion: Giant cell myocarditis should always be in the differential for a new onset heart failure with a fulminant course. Similar to this case, patients often present with severe, unexplained congestive heart failure/cardiogenic shock. Some may have a viral prodrome[1]. Many will have refractory ventricular tachyarrhythmias or heart block. Early recognition and diagnosis is essential. Median survival to death or transplantation is approximately 5.5 months[1]. Immunosuppressive therapy with a regimen including cyclophosphamide (either with steroids, steroids + azathioprine, or steroids + muromonab-CD3) has been shown to improve 1 year survival[2]. However, in acutely ill patients, mechanical circulatory support via ECMO or a ventricular assist device may be necessary[3]. If the patient improves, the duration of immunosuppressive therapy is unclear[4]. Despite treatment, patients are still prone to ventricular tachyarrhythmias, and withdrawal of immunosuppressive therapy, in some patients, has led to a clinical relapse suggesting the need for indefinite therapy[2,4]. A cardiac transplantation evaluation should be considered on an individualized basis. However, the optimal timing for transplantation is unclear, and there is a risk of recurrence in the transplanted heart[2].

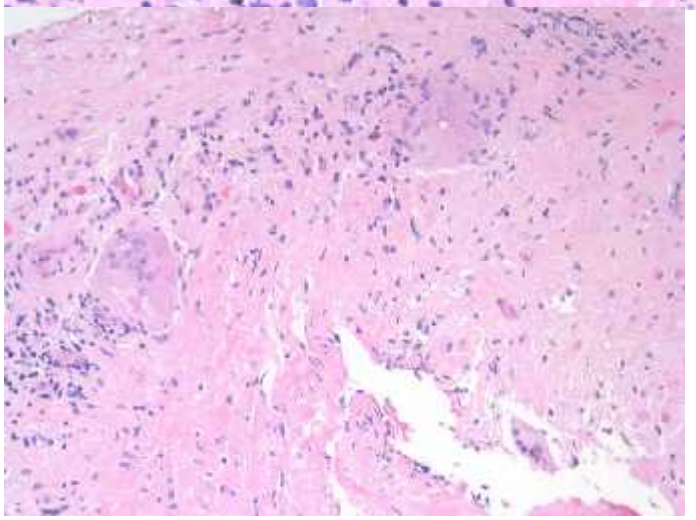
Conclusion: Giant cell myocarditis is a rare, often fatal form of myocarditis. As seen in this case, the clinical course tends to be fulminant, and a high index of suspicion is required to make the diagnosis. If suspected, endomyocardial biopsy can confirm the diagnosis[6]. Treatment with immunosuppressive therapy utilizing a cyclophosphamide regimen has been shown to improve survival, but some may also require early mechanical circulatory support[2,3]. More clinical data is required to determine the optimum immunosuppressive regimen, duration of therapy, and timing of cardiac transplantation if required.

Indiana-ACC Poster Competition Abstract

Pathology Slides







Indiana-ACC Poster Competition Abstract

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Title:

A Rare Myopathic Complication of Statin Use

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

Introduction:
Statin-associated necrotizing autoimmune myopathy is a rare condition with an incidence of approximately two in one million people per year. The etiology of this condition is thought to be due to an auto-antibody directed at 3-hydroxy-3-methyl-glutaryl-CoA reductase. Patients with this condition present with proximal muscle weakness, myalgia, and elevated creatine kinase (CK) levels. As opposed to other statin-induced myopathies, patients continue to be symptomatic even after discontinuation of the offending medication. Diagnosis is based on clinical picture and muscle biopsy, which shows a necrotizing myopathy with little inflammatory component. The condition typically improves with systemic steroids.

Case Presentation:
A 66 year old white male with a history of hypertension and hypercholesterolemia presented to his primary care physician with two weeks of malaise and generalized weakness. One month prior to presentation the patient was noted to have elevated transaminases (AST 144, ALT 266 IU/L), although he was asymptomatic. Atorvastatin, which had been prescribed for years, was stopped by his physician. His only other medication at that time was lisinopril. The patient's initial vital signs and physical exam were normal except for mild hip flexor weakness. At presentation, his serum CK was 16,295 IU/L, serum aldolase was 149 U/L, and urine myoglobin was elevated beyond our laboratory's upper limit. He was treated with aggressive intravenous hydration for presumed rhabdomyolysis despite having no obvious inciting event for this. Over the course of a week the patient's CK rose as high as 25,824 IU/L and he subsequently developed progressive symmetric proximal muscle weakness, especially in his upper extremities. An electromyogram was consistent with muscle necrosis. A muscle biopsy showed a necrotizing myopathy with macrophages infiltrating the necrotic areas of muscle, but overall little inflammatory response. Upon initiation of prednisone the patient's CK level and weakness improved rapidly. He was discharged with a normal neuromuscular exam and will follow up in rheumatology clinic.

Discussion:
This complication of statin use, though rare, needs special awareness by medical providers as the myopathy may persist for weeks to months, and even progress, despite discontinuation of the medication. This is in contrast to typical statin-related myopathies, which improve within days to weeks after the medicine is discontinued. Furthermore, treatment with steroids or other forms of immunosuppression is required to control this condition.

Conclusion:
Knowledge of this diagnosis is important for clinicians as the number of prescriptions for statins is increasing in the United States.

Indiana-ACC Poster Competition Abstract

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Title:

Myocardial Perfusion Images of an Undiagnosed Idiopathic True Left Ventricular Aneurysm

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

A 51 year old male with a 12 year history of nonischemic dilated cardiomyopathy presented with worsening ventricular arrhythmia. His medical history included a prior ejection fraction of 10-15%, a biventricular implantable cardioverter defibrillator (ICD) with epicardial leads placed in 2004 and subsequent subcutaneous leads due to unacceptable defibrillation thresholds in 2006. After many years of controlled arrhythmias he received a lifesaving discharge for sustained ventricular fibrillation in 2013. Investigation for cause of his worsening arrhythmia included a transthoracic echocardiogram (ECHO) which concluded he had a severely increased LV cavity size, global LV hypokinesis and an ejection fraction of 25-30%. Catheter wires from the biventricular pacemaker were confirmed to be in the right atrium and ventricle. He underwent a myocardial perfusion SPECT scan with Regadenoson. These images were challenging to interpret due to the unusual appearance of the defect and were interpreted as possible anterior and anterolateral scar which was not clinically correlated with the ECHO interpretation. These new findings on nuclear imaging led to further investigation with left and right heart catheterization which revealed what was believed to be a very large pseudo-aneurysm. This was further imaged by computed tomography (CT) as magnetic resonance imaging (MRI) was not possible due to his ICD. The patient was emergently hospitalized for surgical management of a potentially life threatening pseudo-aneurysm and underwent surgery where the abnormality was identified as a true LV aneurysm with an intact myocardial wall. It was resected and the epicardial leads were replaced. The cause of the true aneurysm remains unclear but surgically it was not related to the epicardial lead location. The patient recovered well from surgery without complication. Images include echo, LV gram, nuclear stress test and cardiac CT scan.

Indiana-ACC Poster Competition Abstract

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Title:

Atrial Pacemaker Lead Causing Tricuspid Valve Obstruction

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

Introduction

Cor pulmonale is one of the recognized consequences resulting from cardiac valvulopathy in patients with right-sided bioprosthetic valves. It may be caused by insufficiency or stenosis of either the tricuspid or pulmonic valve. Tricuspid valve prosthetic stenosis is a rare cause of cor pulmonale. However, obstruction of the tricuspid valve inflow from an atrial pacemaker lead is considerably less common.

Case Presentation

A 58-year-old engineer and computer programmer presented with increasing symptoms of upper respiratory tract congestion, shortness of breath, and dizziness. He had a long history of known aortic stenosis for at least 18 years. He had undergone Hodgkin's disease treatment with radiation therapy to the neck and mediastinum in 1977. He presented to an outside hospital with rapid atrial flutter and fibrillation. He has history of radiation induced heart block and has had a permanently implanted dual chamber pacemaker. His echocardiogram showed a peak gradient across his aortic valve at 75 mmHg, with a valve area of less than 1 square cm. He had preserved left ventricular function and severe tricuspid regurgitation. He underwent valve replacement with a 25mm On-X mechanical aortic valve and a 31 mm Medtronic Mosaic porcine tricuspid valve. The right ventricular pacemaker lead was left in place and the tricuspid valve was placed so that the lead was sewn between the native valve annulus and the prosthetic valve. The atrial pacemaker lead was obstructing the surgical field, so it was removed from its original position and attached to the right atrial appendage. His postoperative course was complicated by renal and hepatic failure with the eventual development of anasarca. An echocardiogram was performed 2 weeks later which revealed an elevated gradient across his tricuspid prosthesis. A 3-D transesophageal echocardiogram was performed to better evaluate the tricuspid valve, which revealed the atrial pacemaker wire was overlying the inflow to the bioprosthetic valve. The patient had his pacemaker pocket excised, his atrial lead withdrawn, and a loop was sewn in place in his pacemaker pocket. A TEE was performed after the procedure which revealed a decline in the maximum gradient from 25 mmHg to 10 mmHg. Unfortunately, the patient continued to develop multisystem organ failure and died in the hospital after a 3 month hospitalization.

Discussion

Early complications of tricuspid valve replacement that have been previously well described in the literature include thrombosis, patient-prosthesis mismatch, paravalvular regurgitation, and hemolytic anemia. Endocardial pacemaker or defibrillator leads crossing a native tricuspid valve complicate the surgery and can cause or exacerbate TR by mechanisms including impingement on a tricuspid valve leaflet, leaflet tethering to the lead, leaflet perforation, and entanglement of the lead with subvalvular chordae [1]. Lead removal after development of moderate to severe TR and tricuspid annulus dilation may not reduce TR [2]. Transvalvular pacemaker leads also increase the risk of recurrent regurgitation after tricuspid valve repair [3]. Strategies to avoid endocardial lead interference with native, repaired, or prosthetic valves include securing the leads in a commissure, securing them in a position outside the valve annulus, or replacement with epicardial leads [4, 5].

Conclusion

Pacemaker leads can cause significant functional obstructions of the tricuspid valve.

Indiana-ACC Poster Competition Abstract

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