

Infective endocarditis presenting as ruptured Sinus of Valsalva Aneurysm

Salima Bhimani, Sruti Rao, Geetha Challapudi, Simone Jhaveri, Hani Najm, Rukmini Komarlu

Cleveland Clinic Children's Hospital, Cleveland, USA

#### Type of submitter

Fellow in Training

#### Abstract

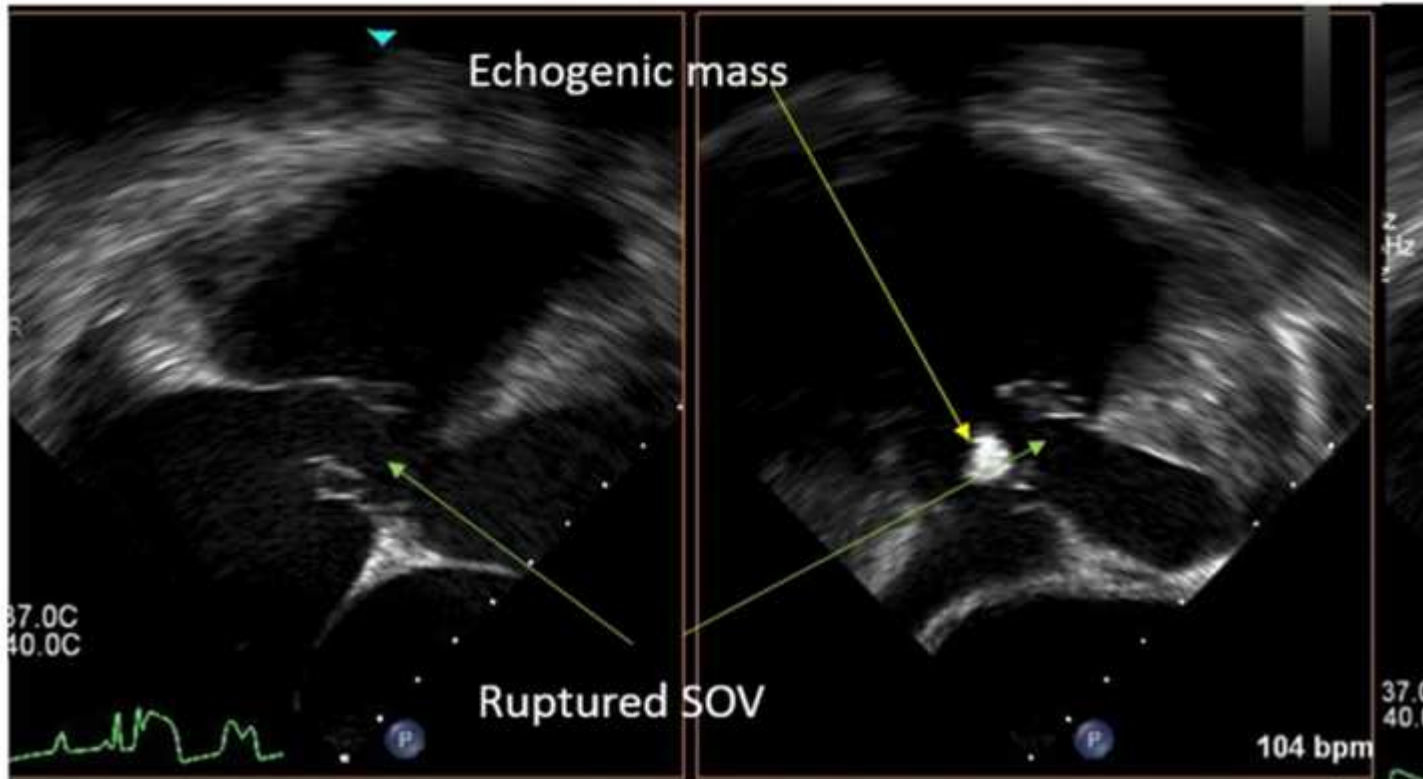
**Background:** Sinus of Valsalva aneurysm is a rare entity, and has multiple causes including post-surgical, trauma, infection, and inflammatory disease with the most common cause being a congenital defect in the aortic media. Most common sites for rupture of sinus of Valsalva aneurysm is into the right atrium and right ventricle. This can result in significant mortality if not recognized. This is the cause of sudden symptomatic aortic regurgitation and should be considered in an ill patient with new onset murmur.

**Clinical presentation:** A 16 year old male with past medical history of developmental delay, Digeorge syndrome, and single kidney presented to an outside hospital with cough, nasal congestion fever and decreased activity of 2 weeks duration. On admission, he had fever, tachycardia, tachypnea, diaphoresis with normal oxygen saturations. Clinical examination revealed an ill appearing 16 year old teenager with new onset systolic heart murmur. His prior echocardiogram 2 years ago was significant for patent foramen ovale and dilated aortic root (measuring 3.44 cm; Z score 2.25). Labs were remarkable for elevated white cell count and CRP along with mildly elevated troponin and acute kidney injury with elevated creatinine. His chest X- ray revealed cardiomegaly and pulmonary edema; ECG revealed sinus tachycardia. The patient was transferred to the PICU due to worsening pulmonary vascular congestion; in the PICU he developed acute onset of melena concerning for GI bleed and his pulse pressure was noted to be wide. A transthoracic echocardiogram was suspicious for coronary fistula to the right ventricle with left coronary artery dilation, left ventricular dilation with normal function and aortic regurgitation. A limited TEE revealed possible ruptured sinus of Valsalva aneurysm. He was then transferred for further cardiothoracic management to our center.

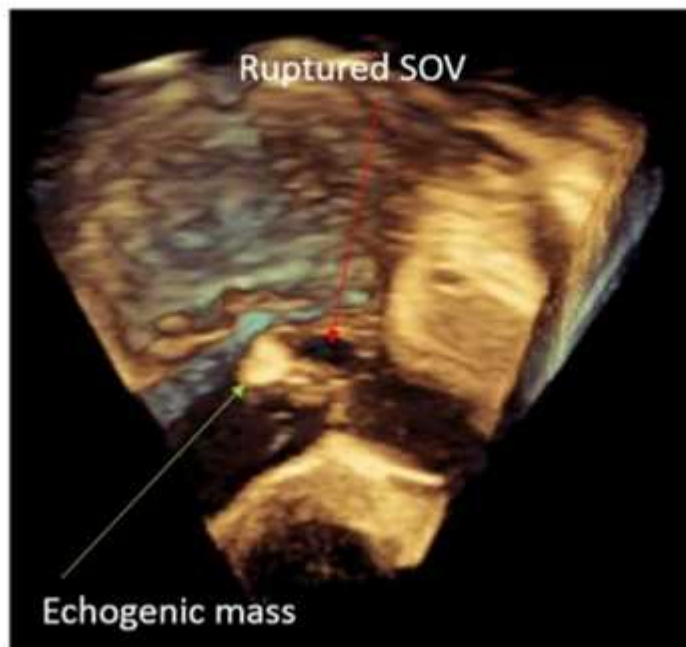
**Clinical Course:** The patient was immediately taken to the OR for surgical repair. Intraoperative TEE was consistent with a ruptured sinus of Valsalva aneurysm of the non-coronary cusp into the right atrium, with resultant large (measuring 1.2 cm) communication into the right atrium with an echogenic mass attached to it suspicious for vegetation. The intraoperative TEE and particularly 3D imaging enabled an accurate delineation of the ruptured aneurysm and its course of drainage. Surgical pathology revealed a calcified thrombus. Repeated blood cultures were negative.

**Discussion:** This particular case was a diagnostic conundrum in many ways. He presented with a clinical picture of sepsis and a widened pulse pressure; in the setting of sepsis, vasodilation is not unusual and a diagnosis of ruptured sinus of Valsalva would be the least likely considered differential. However multiple markers pointed towards a likely cardiac etiology and subsequently non- invasive imaging clinched the diagnosis and aided in the appropriate management strategy.

TEE: Ruptured SOV (sinus of Valsalva) into RA ( echogenic mass attached to the sinus, likely vegetation)



3D TEE image of ruptured SOV



**Categories**

1st year Fellow: Case

**Program Name**

Cleveland Clinic Children's Hospital

Case Report: Cutaneous sarcoidosis, a window into the heart

Laurie Bossory, Rami Kahwash

Ohio State University, Columbus, USA

### **Type of submitter**

Fellow in Training

### **Abstract**

A 61-year-old female with a past medical history of hypertension presented to her primary care physician for evaluation of a rash. She had skin lesions that erupted one year prior on the bridge of her nose and base of her neck. Punch biopsy and resultant dermatopathology revealed well defined non-necrotizing granulomas consistent with a diagnosis of cutaneous sarcoidosis. Initial lab work was significant for normal calcium 10.2 mg/dL and angiotension converting enzyme 5 U/L. Chemistries, liver function tests and hemogram were normal aside from mild leukopenia to 3.28 K/mcL (reference range 4.5-11.0). She was clinically asymptomatic from a cardiac and pulmonary standpoint. On physical exam, lungs were clear to auscultation, she had no elevated jugular venous distension, heart sounds were regular with no murmurs and she did not have any peripheral edema. She was treated with clobetasol cream with improvement of her lesions. Initial chest xray showed stable appearance of bilateral hila without any hilar masses. CT scan of her chest showed bilateral hilar and mediastinal adenopathy with clear lung fields. Her ECG was abnormal with left axis deviation, first degree AV block and incomplete right bundle branch block. Due to the abnormal ECG, she was prescribed a Holter monitor, which showed 105 isolated premature ventricular contractions and 1 premature atrial contraction. Her cardiac MRI revealed normal right and left ventricular function, borderline T2 elevation in the mid to apical anterior and anteroseptal walls concerning for myocardial inflammation or edema, concerning for cardiac involvement of sarcoidosis. The patient declined cardiac biopsy to confirm diagnosis and instead underwent repeat cardiac MRI after a 6-month period that showed stable markers of inflammation and preserved cardiac function. Ultimately, this clinically asymptomatic patient with cutaneous sarcoidosis was discovered to have evidence of likely early cardiac involvement of sarcoidosis of unknown significance on her cardiac MRI. Cutaneous sarcoidosis is the initial manifestation of systemic disease in nearly one third of patients. There is a paucity of data regarding outcomes, treatment and screening of clinically silent cardiac sarcoidosis. Although guidelines do not exist, in such patients, it is prudent to monitor for clinical signs of ventricular arrhythmias (palpitations and syncope) and clinical signs of heart failure that may suggest progression of the underlying cardiac sarcoidosis which warrants consideration of immunosuppressive therapies and prevention of sudden death by an implantable cardiac defibrillator. We propose a follow up with annual echoes to assess cardiac structural changes and screening for conduction system abnormalities by 12 lead EKG and an ambulatory Holter monitor in such patients. In summary, here we present an interesting case of clinically silent cardiac sarcoidosis initially manifesting as a cutaneous lesion and the persistent challenge to physicians on how to appropriately manage these patients.

### **Categories**

1st year Fellow: Case

### **Program Name**

The Ohio State University Wexner Medical Center, Division of Cardiovascular Medicine

71

Burn, baby, burn – Treatment of ventricular dysfunction in a 2 year old girl with ventricular pre-excitation

Emily Hayes, Anna Kamp, Naomi Kertesz, Deipanjan Nandi

Nationwide Children's Hospital, Columbus, USA

**Type of submitter**

Fellow in Training

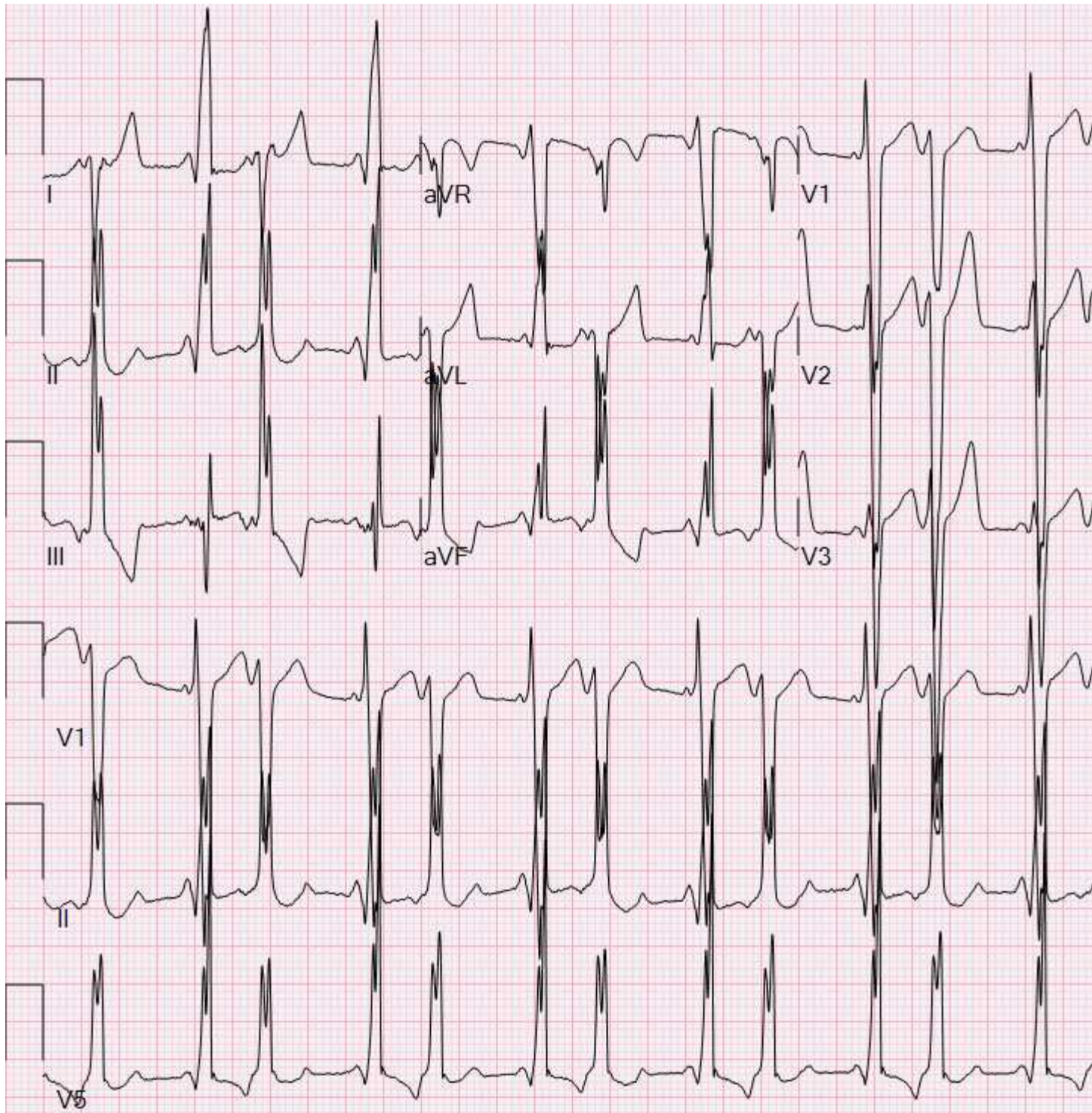
**Abstract**

**Introduction**

Patients with Wolff-Parkinson-White (WPW) syndrome are at risk for life-threatening arrhythmias. Furthermore, recent studies have suggested that dyssynchrony due to an accessory pathway can result in heart failure without associated tachyarrhythmias. Catheter ablation is a Class IIa indication in the recent PACES/HRS guidelines for management of children with WPW pattern and ventricular dysfunction <15 kg who failed medication therapy. In this report, we demonstrate the successful use of radiofrequency catheter ablation (RFCA) as first line therapy for new ventricular dysfunction in a child with WPW who is <15 kg.

**Case Presentation**

An asymptomatic 11-month-old female referred for evaluation after an irregular heart rate had an electrocardiogram (ECG), which showed ventricular pre-excitation and frequent premature ventricular contractions (PVCs). Echocardiogram at time of presentation demonstrated abnormal motion of the basal septum, but with overall normal left ventricular systolic function. She carried a 24% ventricular ectopy burden by Holter monitor, without supraventricular tachycardia. PVC burden decreased to 4% after atenolol initiation with persistent pre-excitation throughout Holter. Echocardiogram ten months later, however, progressed to moderately depressed global left ventricular systolic function with continued abnormal motion of the basal septum. Due to new ventricular systolic dysfunction despite arrhythmia control, at age 21 months and at 13 kg in weight, she underwent an electrophysiology study and ablation of a right superior manifest accessory pathway with inducible orthodromic atrioventricular reentry tachycardia. Her procedure was without any complications. Echocardiogram one month post-ablation demonstrated improved ventricular systolic function, and at one year follow up, ventricular systolic function had returned to normal with no ventricular pre-excitation on ECG.



### Discussion

Approximately 1% of catheter ablations are performed on children each year with 6% of those procedures performed on children <15 kg. In an earlier era, RFCA in children <15 kg was associated with an increased risk of complications. However, newer data and case series suggest that in comparison to larger pediatric patients, these smaller patients fare no differently with respect to rates of complications, success or recurrence, although these small patients with structural heart disease may

have a longer procedure and fluoroscopy time. The only major complication reported in all studies was one case of femoral vessel occlusion in a child <15 kg.

Our case reaffirms that RFCA can safely and effectively be performed in a child <15 kg as the first line treatment for new ventricular systolic dysfunction with ventricular pre-excitation.

### **Conclusion**

We herein report a case of accessory pathway-mediated LV systolic dysfunction in a 2-year-old, 13 kg patient, with complete resolution of dysfunction following successful and uncomplicated RFCA. Though ablation is currently a Class IIa recommendation for patients <15 kg due to increased risks associated with the procedure, continued technological development and increasing experience of physicians at high volume centers allow successful ablation in smaller patients.

### **Categories**

1st year Fellow: Case

### **Program Name**

Nationwide Children's Hospital

Cardiac arrest post-contrast echocardiography: How safe is safe?

Semaan Kobrossi, Otto Costantini

Summa Health, Akron, USA

### **Type of submitter**

Fellow in Training

### **Abstract**

#### **Introduction**

The safety, availability and relatively low cost of transthoracic echocardiography has made it the modality of choice in assessing heart function especially in patients with heart failure<sup>1</sup>. Due to limitations related to technique and patient factors, the use of contrast has improved ventricular cavity visualization and provided a better assessment of systolic function<sup>2</sup>. Although contrast agents are considered safe, they have been uncommonly associated with serious adverse events<sup>3</sup>.

#### **Case**

Patient is a 70 year-old male with past medical history of persistent atrial fibrillation. He was diagnosed with tachy mediated cardiomyopathy with an EF of ~30%, cardioverted, and started on Amiodarone and Metoprolol. He also has a history of hypertension and dyslipidemia. He presented to the outpatient echocardiography laboratory for a repeat echocardiogram to assess his ejection fraction 4 weeks after being discharged with a newly diagnosed systolic cardiomyopathy. Patient was feeling well; vitals were stable and two-dimensional echocardiogram revealed a left ventricular ejection fraction of 45%, improved from the previous admission. Due to poor image quality, the patient received a 2 ml intravenous injection of Lumason (sulfur hexafluoride lipid-type a microspheres). Five minutes after receiving the injection, he started having headache, flushing, sweating and became pulseless and unresponsive. CPR was initiated and code-blue was called. The observed rhythm was pulseless electrical activity. After 6 minutes of CPR and 1 mg of Epinephrine, patient had return of spontaneous circulation. Post-arrest, patient was alert and oriented to time, place and person. He had no wheezing on lung auscultation and no rash on skin exam. He had a heart rate of 52 bpm, blood pressure of 120/67 mmHg and oxygen saturation of 98% on 6 L/min oxygen by facemask. EKG showed sinus bradycardia with no acute ST-T wave changes. Patient was transferred to the cardiac critical care unit for monitoring and was given 125 mg of IV Solumedrol and 20 mg of oral Famotidine for presumable anaphylactic reaction. Patient was observed overnight and discharged home in stable condition the next day. He was seen in the cardiology clinic a week later, and was doing well, without symptoms of congestive heart failure.

#### **Discussion**

Lumason is one of the newer contrast agents that has been more commonly used over the last year. During clinical trials of Lumason, serious adverse reactions were observed in 2 subjects, one with pre-syncope and the other with anaphylactic shock, both occurring within 30 minutes of contrast administration<sup>3</sup>. Only a few cases of anaphylaxis following the administration of ultrasound contrast agents have been reported, often without a defined etiology. Only one case of anaphylaxis due to Lumason was reported in the literature, in a patient with systemic mastocytosis<sup>4</sup>. The likely mechanism of our patient's cardiac arrest is severe vasodilation in reaction to Lumason with possible anaphylactic reaction, while vasovagal reaction is also a possibility.

#### **Conclusion**

Although contrast use in echocardiography is generally safe, they should be used with caution in patients with unstable cardiopulmonary conditions. The presence of resuscitation personnel and equipment is also recommended in these patients.

**Categories**

1st year Fellow: Case

**Program Name**

Summa Health/Akron City Hospital

Pericardial Tamponade following ST-Elevated Myocardial Infarction complicated by Cardiogenic Shock requiring VA-ECMO

David Pratt, Atif Hassan, Louis Louis, Dennis Wells, Saad Ahmad

University of Cincinnati, Cincinnati, USA

#### **Type of submitter**

Fellow in Training

#### **Abstract**

##### **Introduction/Objective**

Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO) is becoming a more commonly utilized method of managing patients in refractory cardiogenic shock following acute myocardial infarction (AMI). Pericardial effusions are a well described phenomenon following AMI, presumably from myocardial and pericardial inflammation following an ischemic event. Rarely, these effusions can lead to cardiac tamponade. To our knowledge, this is the first described case of a patient developing cardiac tamponade requiring emergent pericardiocentesis following an ST-Elevation myocardial infarction complicated by cardiogenic shock requiring VA-ECMO.

##### **Case Presentation**

A 42-year-old male with no significant medical history presented to the emergency department complaining of acute onset, substernal chest pain radiating to the left shoulder. Electrocardiogram was concerning for ST elevation in the anterior leads (Figure A). Cardiac catheterization revealed 100% occlusion of the left main coronary artery (Figure B). The patient was placed on hemodynamic support with Impella CP (Abiomed) and revascularized with two drug-eluting stents in a bifurcation strategy with intervention into the left main coronary artery extending into the proximal left anterior descending artery and proximal left circumflex artery (Figure C). Initial bedside echocardiogram showed severe biventricular dysfunction with no evidence of pericardial effusion. Due to hemodynamic and ventilatory decompensation, the patient was upgraded to percutaneous VA-ECMO support. Overnight, the patient became hemodynamically unstable; a repeat echocardiogram revealed a new, large pericardial effusion with right atrial and right ventricular collapse consistent with cardiac tamponade physiology (Figure D). Emergent pericardiocentesis evacuated 250 cc of serosanguinous fluid with subsequent hemodynamic improvement. Unfortunately, on hospital day 4, his left pupil was fixed; emergent head computed tomography revealed a large left hemispheric intracranial hemorrhage. The family elected to withdraw life sustaining support, and the patient expired shortly thereafter.

##### **Discussion**

Due to right ventricular emptying and altered hemodynamics by VA-ECMO, tamponade may not be easily appreciated. Several case studies have described hemorrhagic pericardial effusions in patients requiring VA-ECMO, many of them from mechanical complications of cannulation. To our knowledge, a serous pericardial effusion causing tamponade physiology in patients requiring VA-ECMO after AMI has not been documented previously. Serous effusions following AMI, commonly known as Dressler's Syndrome, is classically weeks to months following an AMI. One case series in pediatric patients described four patients requiring ECMO who developed serous pericardial effusions requiring pericardial drainage but did not involve myocardial infarction.

##### **Conclusion**

Cardiac tamponade as a manifestation of serous effusion is a rare manifestation post AMI. Due to right ventricular emptying the vulnerability of the right atrial and ventricle for collapse may be increased with low volume effusion. The swan hemodynamics and echocardiogram will be key to appreciate tamponade, which is traditionally a clinical diagnosis. Here, a timely appreciation and intervention altered the clinical course acutely.

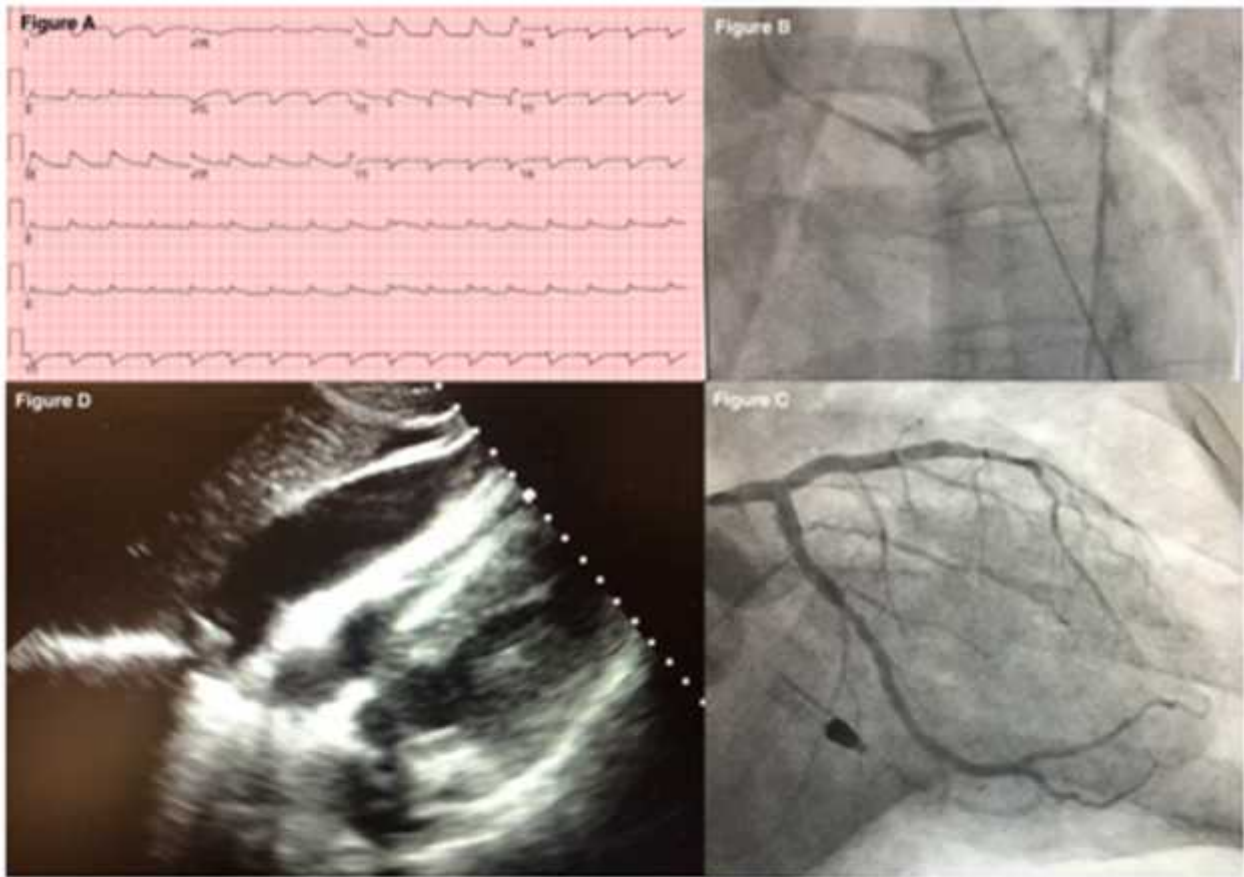


Figure A: EKG on presentation  
Figure B: Cardiac Catheterization with Left Main Coronary Occlusion  
Figure C: Cardiac Catheterization s/p Percutaneous Coronary Intervention  
Figure D: Echocardiogram with RV collapse

**Categories**

1st year Fellow: Case

**Program Name**

University of Cincinnati

The catheter went where? A case of a fractured port catheter in the coronary sinus.

Abdur Rehman<sup>1</sup>, Umbreen Hussain<sup>1</sup>, Stephanie Shelton<sup>2</sup>, Amanda Kitchen<sup>2</sup>, Patricia Frazier<sup>2</sup>, Palaniappan Muthappan<sup>1,2</sup>, Ravi Gurujal<sup>1,2</sup>

<sup>1</sup>Wright State University, Dayton, USA. <sup>2</sup>Miami Valley Hospital, Dayton, USA

#### Type of submitter

Fellow in Training

#### Abstract

##### INTRODUCTION

Central venous access using implantable medical ports is the mainstay of intravenous chemotherapy delivery in cancer patients. Catheter fracture and migration is a rare phenomenon that can cause devastating complications. We present a case of a fractured medical port catheter and its migration into the coronary sinus followed by successful retrieval using percutaneous methods.

##### CASE REPORT

A 65-year-old male patient with metastatic rectal cell carcinoma diagnosed 8 years prior underwent medical port placement for chemotherapy with subsequent need for continued therapy 3 years post placement. He presented for evaluation of port malfunction. Chest X-ray and fluoroscopic studies confirmed 12cm fractured port catheter fragment with the trailing end in the right atrium. Interventional Radiology unsuccessfully attempted foreign body retrieval via transvenous access using a loop snare and an alligator clamp. Follow-up CT chest revealed that the catheter fragment was lodged within the greater cardiac vein and coronary sinus with associated incidental finding of acute left lower lobe pulmonary embolism. After multidisciplinary team discussion including Cardiothoracic Surgery, attempt was made again to retrieve the catheter fragment percutaneously by Interventional Cardiology. Intracardiac echo was performed that showed that the proximal portion of the catheter was embedded against the right atrial appendage. A needle eye snare was used to retrieve the catheter from below which successfully pulled it partially out of the coronary sinus. Afterward, the gooseneck portion of the snare was used to successfully grasp the catheter fragment and withdraw it through a curved sheath. Repeat fluoroscopy and intracardiac echo confirmed complete extraction of the catheter.

##### DISCUSSION

Retained intracardiac foreign bodies and their retrieval have been previously reported, but even rarer is the migration of a catheter into the coronary sinus. More commonly, intracardiac foreign bodies have been discovered in the cardiac chambers or great vessels, including but not limited to catheter fragments, needles, guidewires, inferior vena cava filters, stents, and bullets. Major complications have been described as a result of intracardiac and endovascular foreign bodies, including arrhythmias, sepsis, endocarditis, valvular malfunction, perforation, and pulmonary embolism as occurred in our patient. Historically, potential risk of serious complications from retained catheter fragments was reported to be as high as 71%, with rate of death at 38%. However, more recent literature has suggested removal on a case-by-case basis, weighing the risks and benefits of foreign body extraction. A multidisciplinary approach is optimal, including Cardiology, Cardiothoracic Surgery, and Interventional Radiology as was pursued in this case. In a review of 27 percutaneous retrievals, snare catheter was the preferred method as loops snares did not have adequate grip strength, and basket snares and grasping forceps were preferred for embedded objects. No complications were reported, however cardiac arrhythmias, ventricular perforation, arterial spasm, thrombosis, and vessel injury have previously been noted.

##### CONCLUSION

Intracardiac foreign body migration into the coronary sinus is a rare event that may result in thrombosis and thus require removal. Percutaneous retrieval is a safe method for retrieval of intracardiac foreign bodies, however does not preclude surgical intervention if needed.

**Categories**

1st year Fellow: Case

**Program Name**

Wright state University, Cardiovascular program

## A Case of Acquired Gerbode Defect with Infective Endocarditis

Balaji Shanmugam, Abdur Rehman, Mohammed Abdulrazzaq, Umbreen Hussain, Theresa Ratajczak

Wright state university, DAYTON, USA

### Type of submitter

Fellow in Training

### Abstract

A 54 yr old male with PMH of severe aortic insufficiency s/p bioprosthetic aortic valve replacement, HTN, HLD, COPD, HFpEF, Diabetes was admitted after experiencing acute numbness of left leg. Vitals were T 98.3; BP 176/95 HR 118 RR 18 O2 98% on room air. Patient reported 2 months of intermittent subjective fevers with flu-like symptoms but did not seek medical care.

Examination revealed pulseless left lower extremity and normal rate and rhythm with no murmurs. CTA abdomen with runoff showed embolic infarcts to spleen, left kidney, and occlusive embolus of distal left common femoral artery into proximal superficial and profunda femoral arteries. He underwent emergent open left transfemoral ilio/femoral/popliteal embolectomy with vascular surgery. Post-operatively the patient had a temperature of 100.3 and Infectious disease were consulted. Given the history and findings of emboli; there was suspicion for infective endocarditis, thus a transthoracic echocardiogram (TTE) and blood cultures were ordered. Cultures were positive for *Granulicatella adiacens* and antibiotics were initiated.

TTE showed normal LV function with EF 55%; doppler showed increased aortic velocities with mean gradient 13mm and valve area ~1.1cm<sup>2</sup>. Due to lack of valve anatomy assessment a transesophageal echocardiogram (TEE) was performed which revealed trivial aortic regurgitation, an echodensity attachment on the aortic valve consistent with vegetation. In addition color doppler showed a possible communication between aortic root and right atrium suggestive of a perforation/shunt. OMFS evaluated the patient and extracted of #3 and #30 due dental caries as suspected source of infection.

Infectious disease recommended surgical evaluation due to the virulence and risk of possible valve abscess causing perforation. Cardiothoracic surgery evaluated the patient; preoperative cardiac catheterization showed patent coronaries and underwent aortic valve replacement. Intraoperatively he was found to have vegetations involving the leaflets of the bioprosthetic valve. In addition there was a fistulous connection between the LV and RA underneath the aortic annulus at the commissure between right and noncoronary cusps. The defect was closed with prolene sutures; the old bioprosthetic valve was explanted and replaced with an Edwards Perimount magna ease tissue valve and adequate seating was confirmed.

Gerbode defect is a direct communication between the left ventricle and right atrium that can rarely be acquired as a complication endocarditis. This defective connection is extremely difficult to identify and requires keen reading of echocardiogram results. Due to systolic pressure gradient between the LV and RA; the jet mimics the regurgitant jet seen in setting of severe pulmonary hypertension and is often misdiagnosed.

When the defect is small in size, it is well tolerated and usually does not produce any symptoms however larger defects can present with holosystolic murmurs. Once discovered, the closure of the defect must be taken with great awareness because damage to nearby conduction system will often require pacemaker implantation.

In our case a careful and complete TTE evaluation led to to the discovery of fistulous connection which allowed the surgeon to accurately seek and suture the defect without any structural damage.

### Categories

1st year Fellow: Case

### Program Name

Wright State University

## Mitral Valve Mass in a Young Male Athlete Presenting as Syncope and Stroke-like Symptoms

Christopher Tanayan, Yesha Patel, Akshay Deotare, Ted Shaub

Summa Akron City Hospital, Akron, USA

### Type of submitter

Fellow in Training

### Abstract

#### Introduction

Young athletes are generally considered a healthy demographic. Unexpected diagnoses call for critical decision-making as they pose significant morbidity and mortality risks. A case is presented of a rare cause of syncope and stroke in a young athlete.

#### Case Presentation

A 20-year-old male college basketball player presented with a 2-month history of transient neurological deficits. He had multiple falls due to lower extremity sensory loss at home, bilateral vision blurriness with lightheadedness during a practice, and a syncopal event while walking. He has no known past medical history or family history of predisposing conditions. He denied alcohol, tobacco or drug use. Vital signs and physical exam were unremarkable. He had no murmurs or neurological deficits. A transthoracic echocardiogram revealed a mobile structure on the anterior leaflet of the mitral valve. Laboratory work up was unremarkable except for a mildly elevated INR. Brain MRI revealed multiple punctate foci of acute ischemia in the left occipital lobe and bilateral cerebellar hemispheres and remote lacunar infarcts in the right cerebral hemisphere. Resection of the mass with pericardial patch mitral valve repair was performed without complications. He was given empiric antibiotics. Pathology of the mass showed an organizing fibrinous vegetation with minimal inflammation. No malignant cells, fungal or bacterial organisms were seen. Additional special stains ruled out fibroelastoma. The patient was discharged and rehabilitated. On follow up after a few months, recurrence of the mass was seen on the posterior leaflet of the mitral valve. The patient was given IV anticoagulation which resolved the mass after a few days of treatment. He was discharged on indefinite oral anticoagulation and scheduled for further work up.

#### Discussion

Cardiac masses, specifically valvular masses, can be broadly categorized into a tumor or a thrombus. Tumors can be either primary or secondary; benign or malignant. Thrombi can be infectious or non-infectious. Generally, signs and symptoms of cardiac masses are determined by size and location and not by histopathology. Determination of the type and cause of the mass dictates management. Regardless of the pathology, a tumor is resected for patients who had embolic events or complications directly related to tumor mobility or size to prevent further morbidity and mortality. Adjuvant or palliative therapy is given if appropriate. Infectious thrombi are treated with source control and antibiotics. In this case, given the recurrence of the mass within a few months and resolution with anticoagulation, non-bacterial thrombotic endocarditis (NBTE) is suspected after a thorough inpatient workup. NBTE is rare and is often found postmortem in patients with malignancy or rheumatologic disorder. Indefinite anticoagulation and continued investigation is planned to identify an underlying prothrombotic condition in this patient.

#### Conclusion

Cardiac masses in a young healthy patient are rare and awareness of the differential helps clinicians surmount diagnostic and management challenges that can be both life-changing and saving in this population.

### Categories

1st year Fellow: Case

**Program Name**

Summa Akron City Hospital

76

Postpartum Unmasking of Long QT syndrome

Navdeep Tandon, Jessica Kline, Otto Costantini

Akron City Hospital, Akron, USA

**Type of submitter**

Fellow in Training

**Abstract**

Introduction:

Congenital long QT syndrome (LQTS) is a rare entity that is inherited as an autosomal dominant or less commonly as an autosomal recessive pattern. These genetic patterns encode cardiac ion channels and can remain clinically silent until a patient is exposed to a particular predisposing factor. It is important clinically because it can lead to life-threatening polymorphic ventricular tachycardia and can cause syncope or sudden cardiac death. There are 15 different genetic mutations that are known. LQT1, LQT2, and LQT3 account for 90% of cases of congenital LQTS.

Case:

A 24 year old woman who was 5 months postpartum with no known past medical history presents to the ER via EMS after being found unresponsive and pulseless by family. She had a syncopal event with a brief loss of consciousness the prior night and was evaluated at an outside ER. After a "normal" EKG, BMP, UA and head CT she was discharged home. It was later discovered her EKG at that time showed a QTc of 550 msec. She had a history of several syncopal episodes in the past without workup. Her brother also had a history of syncopal episodes as a child, however, no etiology was discovered. She denies any medication or drug use or any family history of sudden cardiac death. While in our hospital she had 2 cardiac arrests secondary to polymorphic ventricular tachycardia. After the first arrest she was treated with amiodarone in the ED, which further prolonged her QTc interval to 615 msec. The amiodarone was stopped and lidocaine and metoprolol was started. Prior to discharge a subcutaneous ICD was placed. Genetic testing is pending at this time.

Discussion/Conclusion:

LQT2 is caused by a genetic mutation to the gene KCHN2 that encodes for the repolarizing potassium current IKR which leads to an action potential prolongation by impairing repolarization of myocardium. The most common manifestation of LQTS of any type are syncope or sudden cardiac death. We utilize genetic screening to attempt to identify the type of LQTS and to screen first degree relatives. Genetic screening is successful in identifying about 70-80% of patients. The diagnosis of LQTS can be established in patients with heart-rate corrected QT (QTc) intervals greater than 480 msec in repeated EKGs for patients with syncope after excluding any secondary causes. The risk of sudden cardiac death is dependent on QT interval duration, genotype and gender. LQT2 and 3 tend to have longer QT duration. If QTc is >500 msec there is a 5-8x increased risk of a cardiac event by age 40. Females with LQT2 with QTc interval >500 msec are in the highest risk category along with males with LQT3. These 2 types have suboptimal responses to beta-blockers. These 2 subgroups are also most likely to be considered for primary prevention ICDs. Our patient demonstrated a rare syndrome with a history of prior undiagnosed syncopal episode and predisposing trigger for LQT2 given that she was postpartum. She was treated with metoprolol and had a subcutaneous ICD placed for secondary prevention of sudden death.

**Categories**

1st year Fellow: Case

**Program Name**

Akron City Summa Cardiology

73

AV Nodal Ablation to Treat Monomorphic Ventricular Tachycardia

Muhammad Ali Zulqarnain, Vishal Dahya, Otto Costanini

Summa Health, Akron, USA

**Type of submitter**

Fellow in Training

**Abstract**

Introduction:

Patient with ischemic cardiomyopathy are at risk for monomorphic ventricular tachycardia (MMVT) and sudden cardiac death. Atrial Fibrillation has been associated with a higher risk of ventricular tachy-arrhythmic events. We present a case of recurrent MMVT precipitated by rapid AFib treated with AV nodal ablation.

Case presentation:

66 yo man with an ischemic cardiomyopathy due to a large anterior wall MI in 2008 who presented in February 2017 to the hospital with dizzy spells and was noted to have frequent long runs of non-sustained VT lasting 10-20 beats at a rate of ~220 bpm. His LVEF was measured at 30% by echo. He had been in permanent atrial fibrillation for several years. His QRS was narrow and a single chamber ICD was implanted then. Over the next several months, he was noted to have frequent sustained MMVT often interrupted by anti-tachycardia pacing, but sometimes requiring ICD shocks. It was noted that these episodes were often initiated by exertion when his atrial fibrillation rate got above 120 bpm. His ventricular response in atrial fibrillation was well controlled at rest, but often above 120 bpm with minimal exertion. His AV nodal blockers were maximized as tolerated by his blood pressure. He was in class II HF on optimal medical therapy. In July 2017 he was started on amiodarone, but the episodes, though less frequent, continued. In Dec2017, he had an AV-nodal junction ablation and his device was upgraded to a CRT-D. Since then he has had no episodes of MMVT or even non-sustained VT.

Discussion:

Studies have shown that atrial fibrillation is independently associated with an increased risk of ventricular tachyarrhythmias. Although other co-morbidities such as the use of antiarrhythmics or QT-prolonging drugs, or acute myocardial infarction may play a role in increasing this risk, they do not fully account for it. The exact mechanism by which atrial fibrillation increases the risk is unknown, but it may be related to frequent variation in R-R intervals with associated pauses, or with a fast ventricular response causing ischemia.

Conclusion:

Our case proves that atrial fibrillation itself can be the cause of the frequent episodes of ventricular tachycardia. In cases where the episodes of ventricular tachyarrhythmias are frequent and can't be controlled by an anti-arrhythmic drug, an AV nodal ablation with a CRT upgrade is a novel way to treat these patients.

**Categories**

1st year Fellow: Case

**Program Name**

Summa Health Cardiovascular Medicine Fellowship