

## Steroids as an Acute Treatment for Refractory Ventricular Arrhythmias in Sarcoidosis

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**Type of submitter**

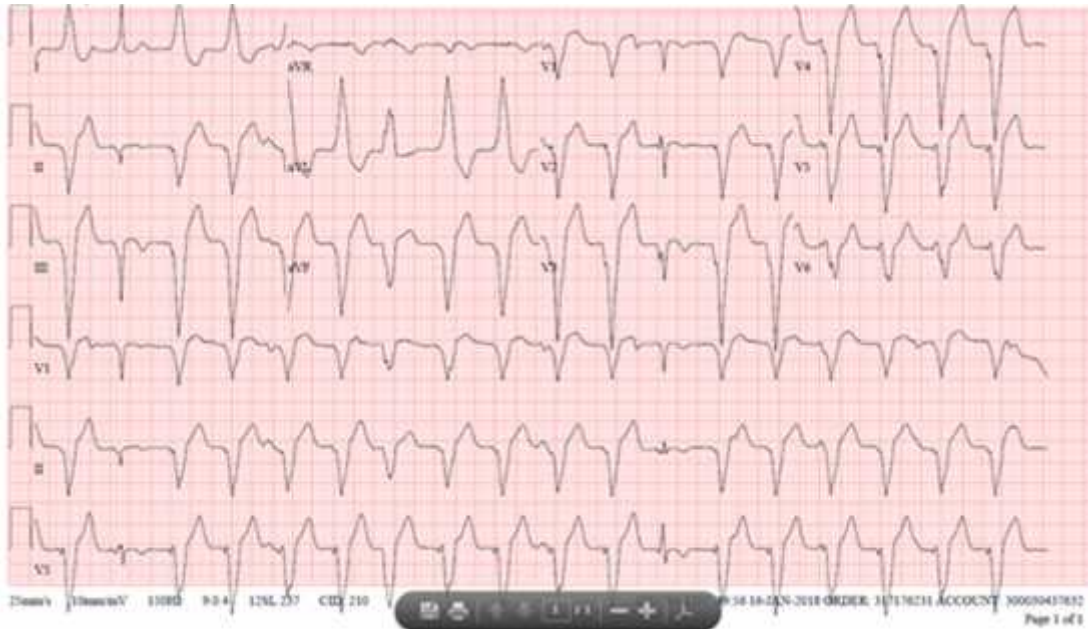
Fellow in Training

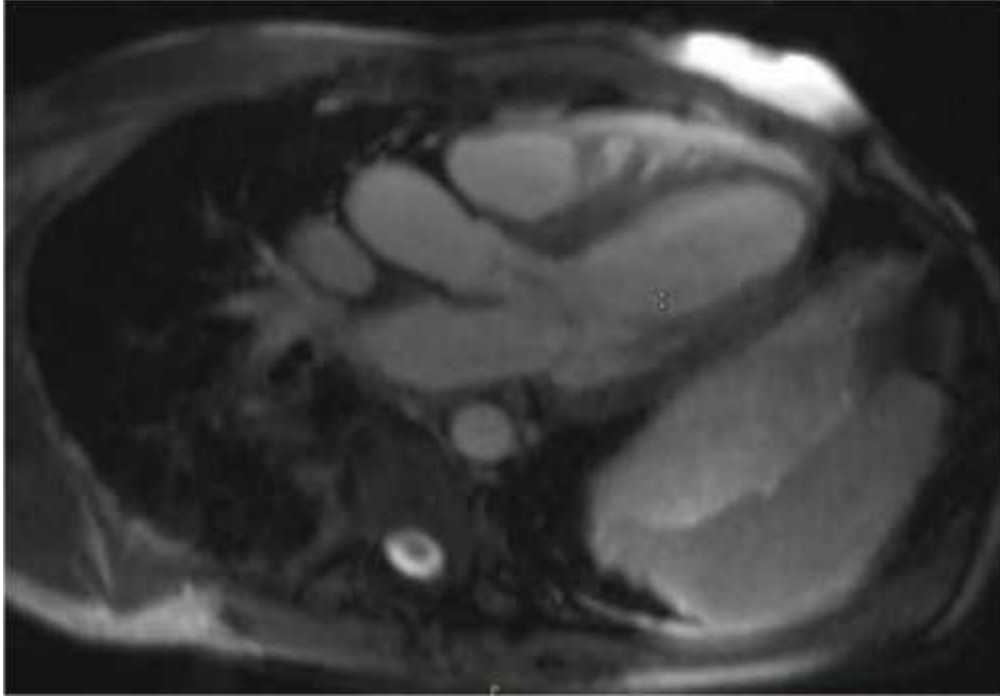
**Abstract**

Sarcoidosis is a granulomatous, multisystem disease of unknown etiology. Frequently, cardiac structure and function can be affected. At times this can have life threatening implications: particularly in regard to sudden cardiac death caused by ventricular arrhythmias. Optimal treatment for sarcoidosis is not well-defined, but typically involves the use of immunosuppressive medications including glucocorticoids, anti-metabolites and biological agents. Standard therapy for heart failure including beta blockers and ACE inhibitors is often also indicated in the setting of ventricular dysfunction.

Discussed is the case of a 63 year old male with known sarcoidosis and cardiac involvement who presented with symptoms of fatigue, palpitations, and dizziness. He was found to have high grade ectopy including frequent premature ventricular contractions (PVCs), bursts of non-sustained ventricular tachycardia (VT) and several runs of hemodynamically stable sustained VT.

Initial treatment with beta blockers and amiodarone had little effect on the ventricular arrhythmias. Subsequent treatment of high dose steroids with 1 gram of methylprednisolone had rapid and near complete resolution of all ventricular ectopy. We discuss the treatment of steroids as an acute therapy for ventricular arrhythmias in the setting of cardiac sarcoidosis and other inflammatory cardiomyopathies.





**Categories**

3rd year Fellow: Case

**Program Name**

Ohio State University

Extremely tall P wave in ESRD patient

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<sup>1</sup>Kettering Medical Center, Kettering, USA. <sup>2</sup>Dayton VAMC, Dayton, USA

### Type of submitter

Fellow in Training

### Abstract

61-year-old male patient with history of hypertension, hyperlipidemia, ESRD on hemodialysis who had presented after abnormal laboratory values, suggestive of hyperkalemia with Serum Potassium of 7.0 mEq/L, Serum Creatinine of 10.7 mg/dL, electrocardiography (ECG) suggestive of atrial tachycardia with variable block and extremely tall P waves as well as peaked T waves (Figure 1). After emergent hemodialysis, results include a Serum Potassium of 3.6 mEq/L and Serum Creatinine of 3.9 mg/dL and ECG suggestive of sinus rhythm (Figure 2). His previous Echocardiograms were suggestive of borderline left atrial enlargement.

P wave morphology has been studied to determine atrial enlargement – (ECG) is highly specific but insensitive for right atrial enlargement. P-wave morphologies are linked with interatrial conduction patterns and may be used to predict clinical outcomes in atrial fibrillation, ischemic heart disease, and congestive heart failure. P pulmonale correlates with hilar thoracic ratio, smoking status, symptom duration of COPD. Transient tall P waves have been described in cases of status asthmaticus, severe hypokalemia and similar electrolyte abnormalities. The amplitude and duration of P waves may be masked by peripheral edema.

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### Categories

3rd year Fellow: Case

### Program Name

Kettering Medical Center

**Mother-and-Child Technique for Diagnostic Angiography in a Dilated Ascending Aorta With an Inner Multipurpose Catheter**

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**Type of submitter**

Fellow in Training

**Abstract**

Case: A 59 year-old man with a history of benign prostatic hypertrophy, nephrolithiasis and hyperlipidemia on statin therapy presented after with episodic non-exertional chest pain. He had a family history of dilated ascending aorta in his father. On CTA of the chest he was found to have a dilated ascending aorta, measuring 5.5 cm in transverse diameter. An echocardiogram was performed which showed the ascending aorta measuring 5.1 cm and severe aortic regurgitation. Cardiothoracic surgery was consulted for the ascending aortic aneurysm. The patient was scheduled for coronary angiography in anticipation of open surgical aortic repair.

The right femoral artery was accessed at the mid femoral head and a 6-French 11 cm sheath was inserted. Given a significantly tortuous external iliac artery (figure) a 6-French 30 cm flexor sheath exchanged in to aid catheter advancement and manipulation.

Multiple attempts to cannulate the left coronary ostium were unsuccessful utilizing both a 6-French Judkins Left-5 (JL-5) and a 6-French Amplatz Left-3 (AL-3) diagnostic catheters. The AL3 was appropriately oriented with lack of length to reach the coronary ostium (figure).

Given the appearance of the root and the inability of the AL-3 to reach the ostium, a 4-French multi-purpose-A2 (MP-A2) diagnostic catheter was advanced through a 6F AL-3 guide catheter utilizing a 'mother and child' technique and was successful in engaging the left main ostium (figure). Selective angiography of the left system was then performed. Angiography of the right coronary was performed using a Judkins Right-5 (JR-5) catheter.

Discussion: The mother and child technique is commonly used in interventional cardiology, especially during interventions on distal lesions, chronic total occlusions, and in unusual anatomy.<sup>1</sup> However, the utilization of this technique is not as common for diagnostic angiography. In the case reports using the mother and child technique, the most common inner ('child') catheter used was the GuideLiner catheter. In contrast, in our case we used a multi-purpose-A2 catheter for greater directional control of the catheter to direct it across the aortic root. In addition, the catheter is stiffer than the Guideliner catheter and as such this maneuver should be carried out with greater caution.

Conclusion: This case uses a novel technique of mother and child catheterization utilizing a 4-French multi-purpose catheter within a 6F AL-3 Guide catheter to successfully engage the Left Main Coronary Artery in a dilated aortic root with an additional challenge posed by a tortuous iliac artery. This technique, using a stiffer, more directional catheter, allows for controlled engagement of the left main in patients with difficult anatomy, especially dilated ascending aortas.

**Categories**

3rd year Fellow: Case

**Program Name**

University of Toledo

Systemic-Pulmonary Collaterals Presenting as Hemoptysis in an Adult with Transposition of the Great Arteries (d-TGA) following the Arterial Switch Operation (ASO)

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Fellow in Training

**Abstract**

**Introduction:** Systemic-pulmonary collaterals are a rare complication noted in the pediatric population following an arterial switch operation. The etiology of these collaterals in association with d-TGA has not been fully elucidated, however they are usually felt to be embryologic ventral splanchnic vessel remnants that persist in patients with underdeveloped pulmonary circuits<sup>1</sup>. These may remain clinically silent or present with heart failure symptoms due to the volume load early in the neonatal period.

**Case:** We report a case of a twenty year old male with d-TGA who previously underwent an arterial switch operation on day 4 of life presenting to the emergency room with large volume bright red recurrent hemoptysis.

On physical examination his blood pressure was 96/53mmHg with normal heart sounds and a physiologically split second heart sound. His hemoglobin had dropped from 15.7g/dl to 13.4g/dl. ECG showed normal sinus rhythm with no signs of ischemia or arrhythmias. Echocardiography revealed a mildly dilated neo-aorta, preserved biventricular function and no supra-valvar or valvar pulmonic stenosis. Chest imaging which included a chest X ray as well as computed tomography angiography showed ground glass opacities in the right lower lobe as well as presence of several collateral vessels. On bronchoscopic evaluation fresh blood with no obvious active bleeding was seen in the right lower lobe.

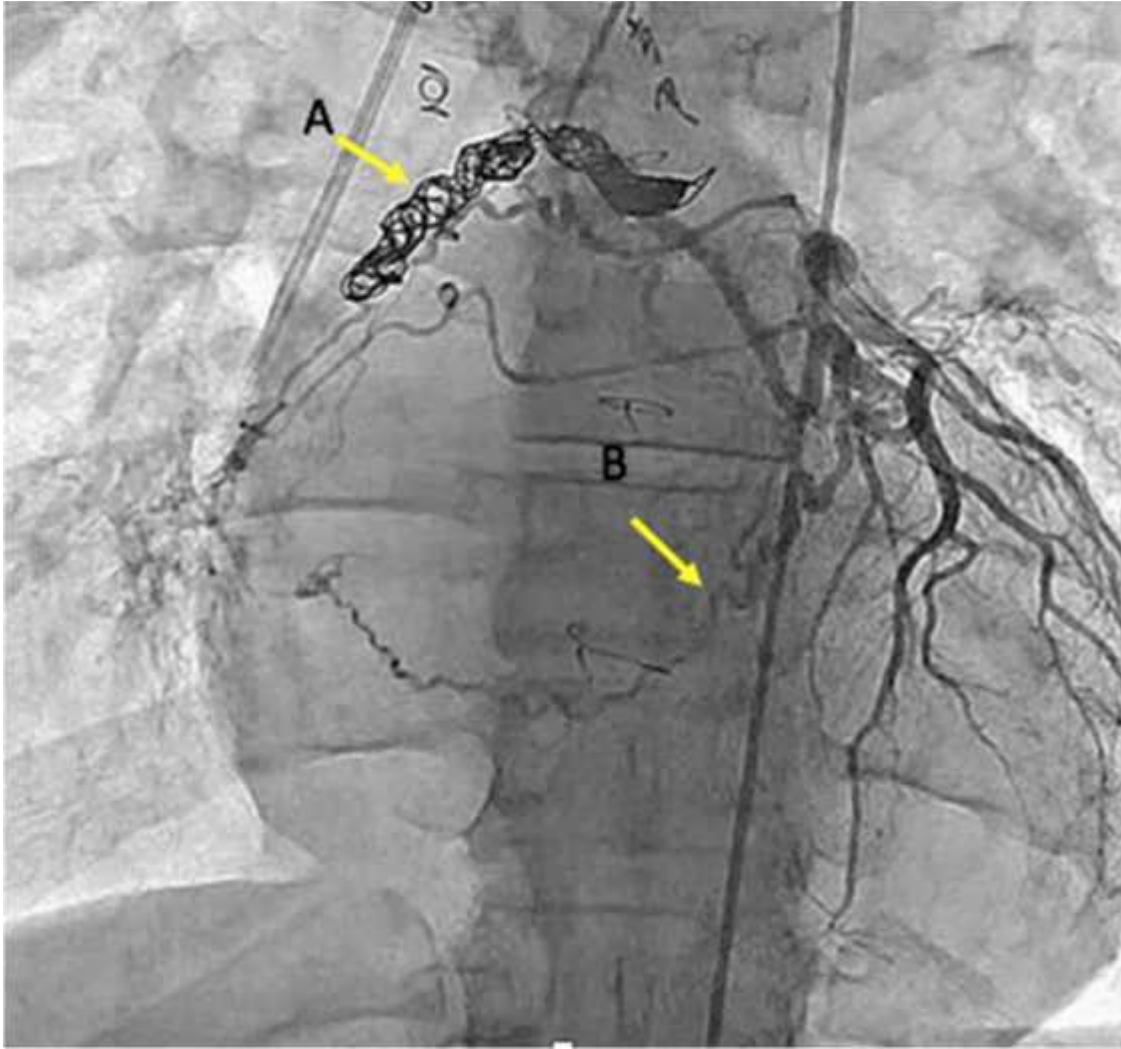
Subsequent retrieval of his prior records revealed coiling of systemic to pulmonary collaterals from the descending aorta and right internal mammary artery, and right vertebral artery at two and three years of age, respectively. These had been diagnosed incidentally upon evaluation for supra-valvar pulmonic stenosis. Based on this history and imaging studies he was sent for cardiac catheterization. Angiography demonstrated several more (not seen previously) systemic-pulmonary collaterals arising from the right internal mammary, right thyro-cervical trunk, descending aorta (Fig 1) as well as from the left circumflex Fig 2(B). These collaterals appeared to be supplying the right lung and was the suspected source of the bleeding. The large systemic-pulmonary collaterals were coiled Fig 2(A). The hemoptysis resolved and the patient developed pleuritic right sided chest pain from infarcted lung tissue which was treated with non-steroidal anti-inflammatory agents. The left circumflex to pulmonary collateral was left alone due to complexity and risks of an intervention. So far the patient has not had any further recurrence.

**Conclusion:** This is an unusual case and the first to our knowledge of an adult patient with d- TGA following the ASO early in life presenting with hemoptysis as a late complication of these collaterals.

**Fig 1 Descending aorta to right lung collateral.**



Fig 2 Left circumflex to right lung collateral.



#### References

<sup>1</sup>Wipf A, Christmann M, Navarini-Meury S, Hitendu Dave, Quandt D, Knirsch W, Kretschmar O. Aortopulmonary collaterals in neonates with d-transposition of the great arteries – Clinical significance early after arterial switch operation. *International Journal of Cardiology*. 2018;258: 237–242.

#### Categories

3rd year Fellow: Case

#### Program Name

Ohio State University

## Coronary Artery Thrombosis in Acute Pancreatitis

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<sup>1</sup>University of Cincinnati, Cincinnati, USA. <sup>2</sup>Advocate Illinois Masonic Medical Center, Chicago, USA. <sup>3</sup>DOW Medical College, Karachi, Pakistan

### Type of submitter

Fellow in Training

### Abstract

#### Introduction

The incidence of acute myocardial infarction (MI) in the setting of acute pancreatitis (AP) is rare. Recognition of such a complex diagnosis may be clinically challenging, as symptoms of both conditions can be indistinguishable. In this case, we encountered both conditions concurrently and hypothesize that the ambient inflammatory and pro-thrombotic milieu of AP resulted in acute coronary thrombosis, despite the absence of significant coronary atherosclerosis. Among multiple coronary imaging modalities currently in use, optical coherence tomography (OCT) provided a unique capability for direct visualization of the coronary thrombus.

#### Case Summary

A 57 year-old male presented to the emergency room with acute epigastric pain and nausea. Serum lipase was found to be markedly elevated at 899 U/L. Initial electrocardiogram (ECG) showed nonspecific ST-segment abnormalities, and serum troponin-I mildly elevated. Approximately one hour later, symptoms intensified and repeat ECG showed 2mm ST-segment elevations in the inferior and lateral leads (Figure 1). The patient was transferred emergently to the cardiac catheterization laboratory with a diagnosis of acute ST-elevation MI.

Heparin and eptifibatide were administered. Coronary angiography revealed an ambiguous haziness within the proximal portion of the right coronary artery (RCA) concerning for a non-occlusive thrombus, though visualization of the lesion was hampered by acute angulation at the involved vessel segment. Notably, left ventriculography demonstrated a small area of dyskinesia at the distal inferior wall. Intravascular ultrasound (IVUS) was performed but showed no evidence for an atherosclerotic lesion or intracoronary thrombus.

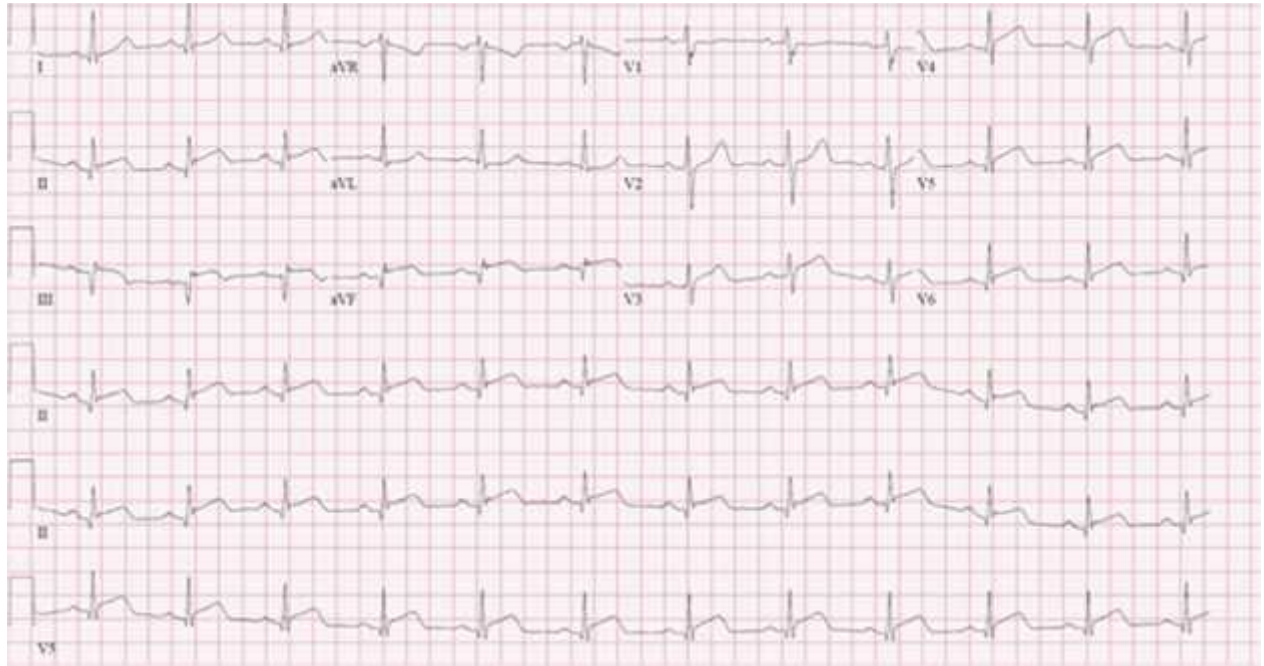
Subsequently, optical coherence tomography (OCT) imaging revealed a large thrombus in the proximal RCA (Figure 3). Attempts at coronary thrombus extraction were unsuccessful, and a 4.0 x 15 mm bare metal stent was placed. The remainder of the hospital course was unremarkable, and three-month follow-up post-discharge showed normal serum lipase and amylase.

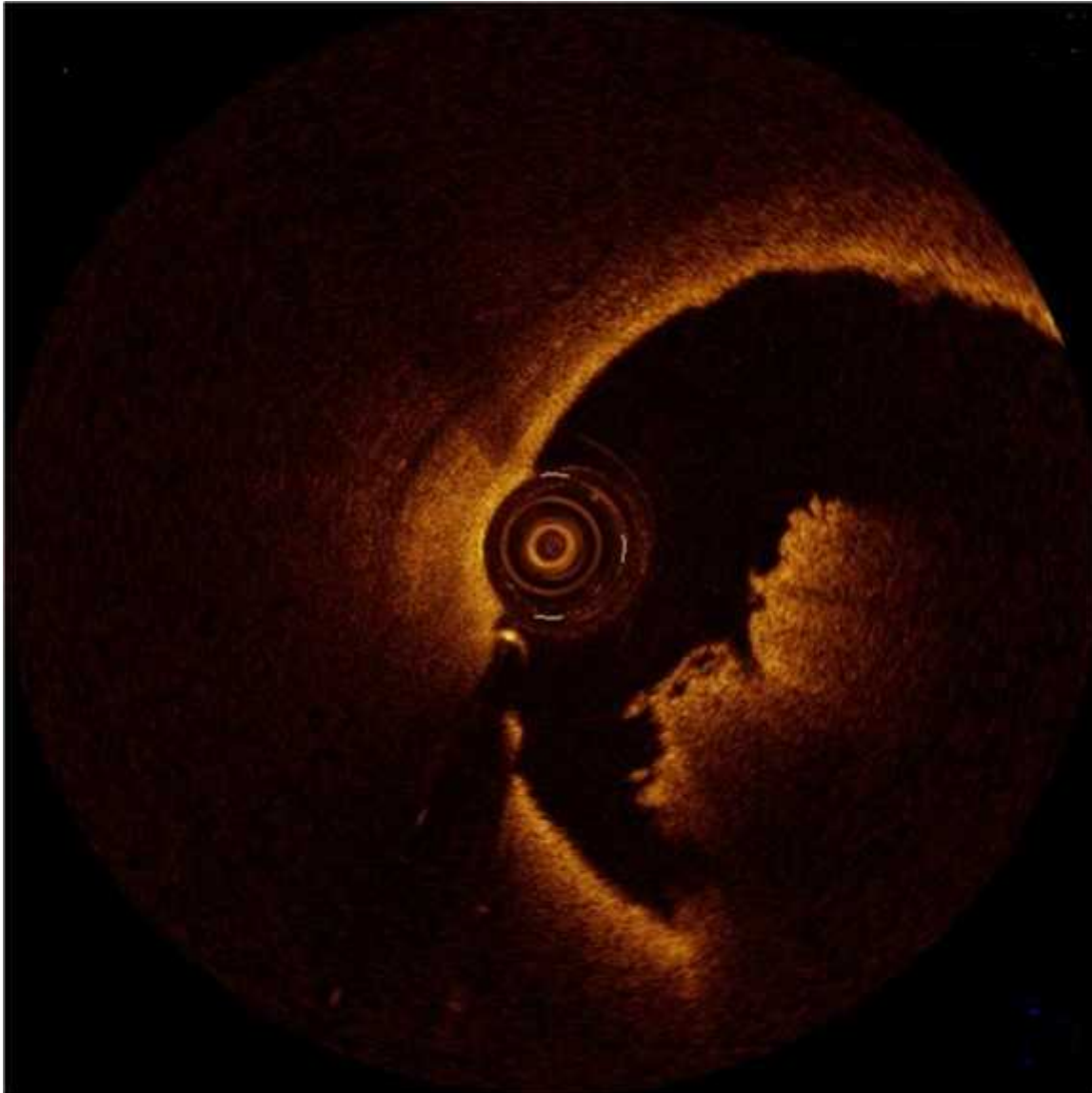
#### Discussion

We hypothesize that the incidence of AP contributed to an inflammatory and pro-thrombotic state that ultimately led to spontaneous arterial thrombosis within a tortuous and angulated segment of the RCA. As previously documented, pro-inflammatory cytokines (i.e. TNF-alpha) activate leukocytes and endothelial cells, promoting expression of tissue factor and initiating the coagulation cascade. Thrombin is formed and produces a clot, but also activates protease-activated receptor-1, which initiates a pro-inflammatory response consisting of cytokines, growth factors, and adhesion molecules. Decreased functionality of antithrombin-III and inhibition of fibrinolysis also likely contribute to hypercoagulation.

OCT allows for greater resolution for evaluation of intravascular pathology compared to IVUS (10-20 microns versus 100-200 microns), coronary computed tomography angiography, and cardiac MRI.

The overlapping clinical presentations and ECG findings of AP and MI can cause confusion and ultimately delay diagnosis. Clinicians must keep a low threshold to perform ECG and cardiac biomarker testing if symptoms are suggestive even in the presence of a clear alternative diagnosis.





**Categories**

3rd year Fellow: Case

**Program Name**

University of Cincinnati

Intracoronary Thrombolytic Therapy in a Patient with ST elevation Myocardial Infarction in the Setting of Carbon Monoxide Poisoning

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**Type of submitter**

Fellow in Training

**Abstract**

**Introduction:**

Carbon Monoxide (CO) is a colorless and odorless chemical asphyxiant that causes tissue hypoxia resulting in manifestations in multiple major organs including the heart.

The mechanism of myocardial injury involves demand ischemia with or without underlying coronary atherosclerosis. Fibrinolytic pathway activation due to endothelial damage from oxygen-free radicals may also contribute.

We report a case of ST elevation myocardial infarction due to a left anterior descending artery thrombus as a rare complication from acute CO poisoning and review all patients reported in the literature in the 21st century.

**Case:**

A 32 year old male with medical problems that include hypertension summoned EMS for complaints of chest pain, headache, and nausea. Chest pain was described as a constant, without radiation and associated with nausea for the previous 5 hours.

It was discovered that the patient was heating his apartment by leaving the door of his gas oven open.

At physical exam his lungs were clear to auscultation. Cardiac examination revealed normal rate and rhythm without murmurs, rubs, or gallops. Bedside transthoracic echocardiogram (TTE) was not significant for pericardial effusion or wall motion abnormality.

Laboratory reported Carboxyhemoglobin: 13.8. Troponin I: 0.126. CKMB: 115. CKMB Index: 4.5. Electrolytes: within normal limits.

An electrocardiogram was done, it showed normal sinus rhythm. Prolonged PR interval. Diffuse ST elevations.

The patient was admitted to telemetry unit and remained without further episodes of chest pain. Overnight, Troponin I increased to 10.7. CK-MB peaked at 168.

Repeat transthoracic echocardiogram was significant for wall motion abnormalities in the apex, apical-inferior, septal, and apical-anterior myocardial walls. Ejection fraction was estimated to be 55%.

At that time an electrocardiogram showed anterior ST elevations.

In telemetry monitor he was found with multiple runs of non-sustained ventricular tachycardia- longest of 5 beats.

The patient was taken for urgent cardiac catheterization that showed proximal left anterior descending (LAD) artery thrombus with 70% stenosis and distal LAD occlusion. The patient was treated with unfractionated heparin and glycoprotein 2b/3a inhibitor (Tirofiban).

During that day the patient remained with chest pain, he went for a second cardiac catheterization that showed residual LAD thrombus. It was treated with a combination of intracoronary thrombolytic (Alteplase) and an aspiration thrombectomy.

Hypercoagulability panel did not reveal underlying pathology that may have contributed to thrombus formation.

Patient was ultimately discharged on oral anticoagulation (Rivaroxaban) for three months.

Three months repeat TTE revealed improvement of wall motion abnormalities. Ejection fraction was 58%.

**Conclusion:**

Intracoronary thrombus is a rare complication of Carbon Monoxide poisoning with only 7 reported cases in the literature. Including our patient, 5/8 (62.5%) reported were men; age range 30-70 years old with a mean 48.5 years. All patients complained of chest pain, exhibited EKG changes, and were diagnosed at cardiac catheterization. All 8 (100%) were found to have a thrombus in the LAD artery.

Our case involves the youngest patient and is the first to be treated with a combination of unfractionated heparin, glycoprotein 2b/3a inhibitor, intracoronary thrombolytic, aspiration thrombectomy, and oral anticoagulation.

**Categories**

3rd year Fellow: Case

**Program Name**

Summa Health System

## Large Pseudoaneurysm Following Aortic Root Surgery in Marfan Syndrome: A Rare Complication of Cabrol Shunt Occlusion

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### Type of submitter

Fellow in Training

### Abstract

#### Introduction:

A Cabrol shunt is employed as a method to control hemorrhage after complex aortic reconstruction surgery. We present a young male with Marfan syndrome who had a protracted course involving multiple aortic root reoperations for severe bleeding despite placement of a Cabrol shunt.

#### Case Presentation:

A 20 year old male with Marfan syndrome underwent an elective David procedure (valve-sparing root replacement) for progressive aortic root dilation. On postoperative day 10, he developed a symptomatic large hemopericardium prompting emergent pericardiotomy without an identifiable source of bleeding. A pre-discharge CT scan demonstrated development of a pseudoaneurysm in the left ventricular outflow tract (LVOT). Subsequently, reoperation consisting of repair of the pseudoaneurysm, re-implantation of the coronary arteries and patch repair of a tear in the LVOT was performed. Given intractable intraoperative bleeding, a Cabrol shunt extending from the peri-aortic space to the right atrium (RA) was created using a patch. He was discharged home without complications.

Three weeks later, a follow up echocardiogram revealed a large anterior pseudoaneurysm. This space received flow in systole through a small communication with the aorta in the supra-valvar region. Additionally, there were 2 jets of communication between the pseudoaneurysm and the LVOT with systolic flow from the space into the LV and diastolic flow in the opposite direction (Figure 1). The re-implanted coronary arteries had laminar flow. There was no communication between the Cabrol shunt and RA. Given the increasing size of this pseudoaneurysm, a decision was made to re-operate. Intraoperatively, it was confirmed that the pseudoaneurysm was formed by both the dehiscence of the aortic graft with resultant perigraft sanguineous collection as well as occlusion of the Cabrol shunt. He underwent a successful Bentall procedure with an On-X valve as it was deemed that reattempting valve sparing surgery would not be feasible. Three months from surgery he had no recurrence of bleeding with good mechanical valve function.

#### Discussion:

Significant bleeding requiring re-operation following aortic root surgery is common. A Cabrol shunt, first described in 1978, is a perigraft-to-RA communication used for bleeding that cannot be controlled by traditional methods. The goal of this shunt is to drain blood that accumulates in the perigraft space directly into the RA, creating a left to right shunt that decompresses the area around the graft, reduces tension on the anastomotic sites and promotes auto-transfusion of blood. The Cabrol shunt typically closes around the first postoperative week as bleeding subsides. Rarely, a patent shunt can cause right-sided volume overload requiring surgical closure. However, our patient demonstrates a unique complication of Cabrol shunt occlusion wherein the bleeding continued to accumulate in the periaortic space forming a pseudoaneurysm that did not communicate with the RA. Echocardiography was key in diagnosis. Tissue friability and altered coagulation secondary to Marfan syndrome was considered a significant contributing factor for these complications.

#### Conclusion:

With the aid of multimodality imaging, we demonstrate a rare case of Cabrol shunt occlusion with continued bleeding leading to a large pseudoaneurysm that necessitated surgery. The aim is to raise awareness of this potential complication in a subset of patients with Marfan syndrome.

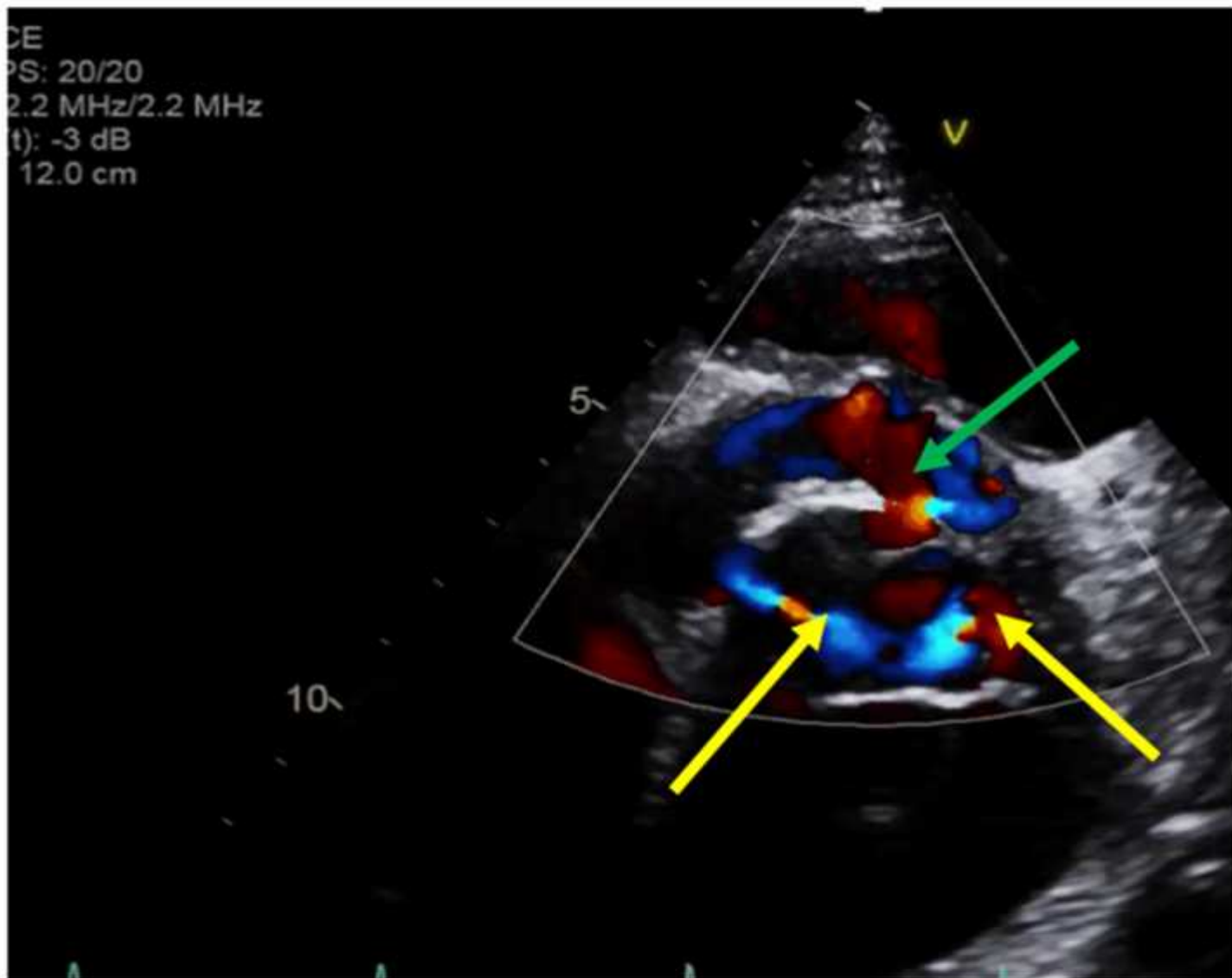


Fig. 1: Parasternal short axis view in color demonstrating a large aneurysm of the aorta. There are 2 jets (yellow arrow) of flow from the LVOT and another communication (green arrow) into the space

Categories

3rd year Fellow: Case

Program Name

Pediatric Cardiology

## Aortopathy in the Fontan Circulation

Sruti Rao, Kenneth Zahka, Hani Najm, Rukmini Komarlu

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**Type of submitter**

Fellow in Training

**Abstract**

**Introduction:** We report a unique case of rapid progression of neo-aortic root dilation, progressive neo-aortic regurgitation and neo-ascending aortic aneurysm in a patient with hypoplastic left heart syndrome palliated to a Fontan procedure necessitating a Bentall procedure. Our patient had clinical signs of Marfan syndrome raising concern for an aortopathy complicating single ventricle palliation.

**Case report:** An 11 year old male with hypoplastic left heart syndrome (mitral and aortic atresia), underwent staged palliation to extra cardiac non-fenestrated Fontan. Post Stage 1 Norwood procedure, he developed recurrent coarctation requiring repeated balloon angioplasties. Additionally he had a myxomatous tricuspid valve with tricuspid valve prolapse. Extracardiac findings included bilateral ectopia lentis, tall stature with slender fingers and toes. Genetic testing was negative for FBN-1 (Fibrillin 1) gene despite phenotypic features of Marfan syndrome.

In early adolescence, he presented with acute dyspnea on exertion. Echocardiogram and MRI revealed a relentless increase in aortic root size to 55 mm x59 mm and severe aortic regurgitation resulting in severe right ventricular dilation with decreased systolic function (RVEDVi = 200cc/m<sup>2</sup>; RVEF = 45%). There was severe tricuspid valve regurgitation, secondary to prolapse of a very myxomatous valve (RF 56%). His underlying connective tissue disorder implied that valve repair in a single ventricle would be very risky. The family was counseled about the potential need for transplant if surgical palliation was unsuccessful.

He successfully tolerated resection of the neo-aortic aneurysm, Bentall procedure with a #27 St Jude composite valve graft, re implantation of the DKS to the right side of the Bentall and tricuspid valve repair with a #36 annuloplasty ring using synthetic chordae. His operative course was significant for proximity of the extremely dilated aortic root to the sternum requiring cannulation in the left groin. He was discharged home on losartan and warfarin.

**Discussion:**

Secondary dilation of the neo-aortic root and less frequently the ascending aorta is seen following congenital heart surgery such as the Ross procedure, arterial switch operation and rarely the Fontan. Primary aortic root dilation has been described in lesions such as a bicuspid aortic valve, coarctation of the aorta, conotruncal abnormalities such as tetralogy of Fallot and many connective tissue disorders.

After staged Fontan palliation, the pulmonary valve functions as the neo-aortic valve and is exposed to systemic pressures contributing to secondary neo-aortic dilatation. In our patient, although genetic testing was negative for classic FBN-1 mutation associated with Marfan syndrome, this test is not 100% sensitive and clinical examination strongly suggested otherwise. The accelerated progression of aortic root dilation with aortic and tricuspid valve regurgitation favored co-existent primary aortopathy seen in Marfan's syndrome. This case highlights the aggressive progression in aortic size thought to be from a combination of an underlying connective tissue disorder and the natural history of secondary aortic root dilation in palliated congenital heart disease. Close monitoring of valve function and neo-aortic dimensions are essential particularly in the presence of single ventricle physiology.

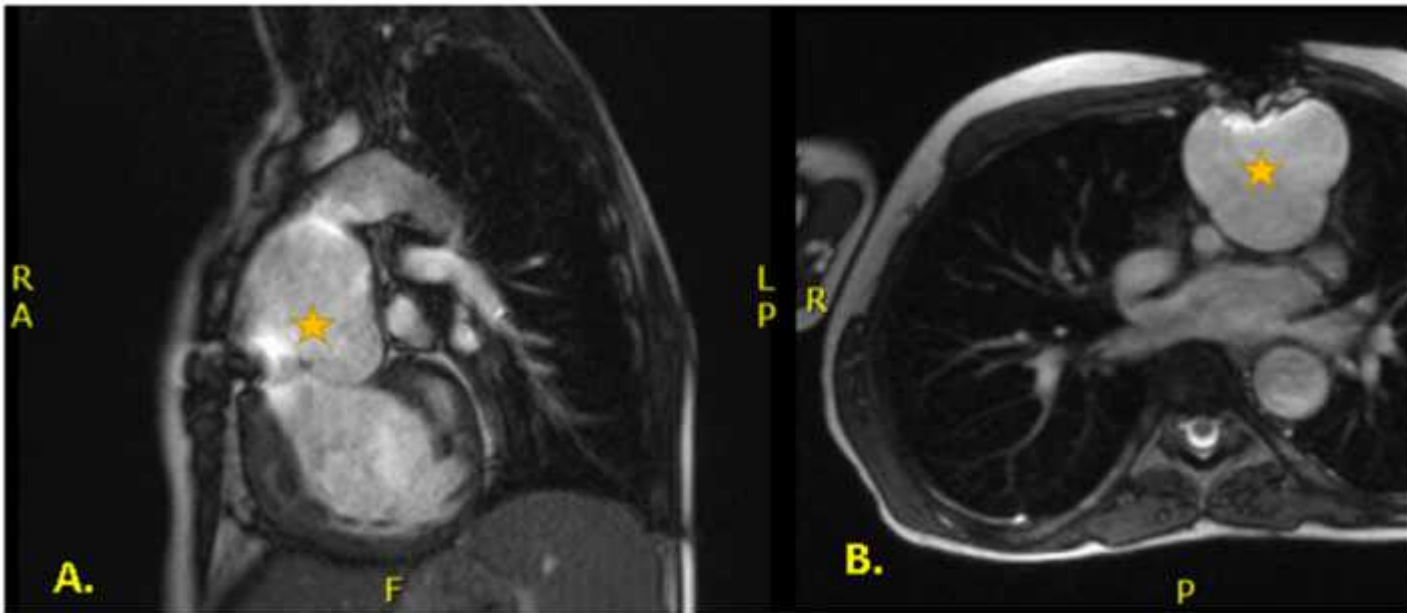


Fig A and B demonstrate dilated neo-aortic root and neo-ascending aorta (★)

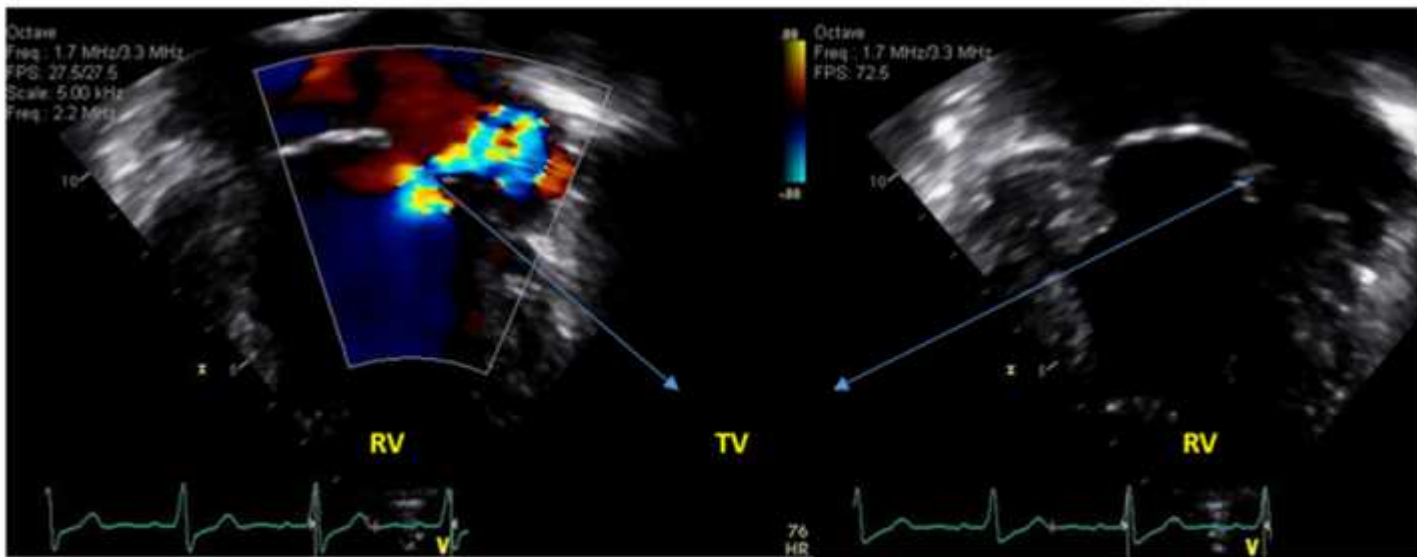


Fig C. Echo demonstrating myxomatous tricuspid valve with severe regurgitation  
 RV –Right ventricle, TV- Tricuspid valve

Categories

3rd year Fellow: Case

**Program Name**

Cleveland Clinic Children's Hospital

**Regional Pericarditis: An Unusual Diagnosis**

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**Type of submitter**

Fellow in Training

**Abstract****Introduction**

Post-operative chest pain is a well described clinical phenomenon. Any irritation to the pericardium may lead to a post-cardiac injury syndrome (post-MI, post-pericardiotomy, post-traumatic pericarditis). Classically diagnosed by pleuritic chest pain, fever, pericardial effusion, leukocytosis, pericardial friction rub, diffuse ST-segment elevation, and PR depression. We present a patient without these features with an atypical presentation of regional pericarditis.

**Case Presentation**

A 74 y/o Caucasian male with history of OSA, dyslipidemia presents to ED with left sided chest pain. The pain started forty five minutes after trimming rose bushes. Pleuritic in nature, radiating across the chest and associated with shortness of breath. After he took aspirin and nitroglycerin the pain eased to a dull sensation. Physical exam revealed bilateral 1+ pitting edema. CTA was negative for PE. Labs showed troponin I of 0.189 ng/mL (N<0.04), and EKG showed nonspecific ST-T wave abnormalities. Repeat troponins were 0.432 - 2.140 - 4.520 ng/mL. The patient remained hemodynamically stable and was treated for a NSTEMI. Left heart catheterization was done showing multivessel disease, including a distal lesion in the left main with EF 55%. Two days later a four vessel CABG was performed (LIMA to LAD, SVG to PDA, ramus marginalis and posterior lateral). After extubation the patient began to have pleuritic chest pain the following day. Physical exam was unremarkable, with down trending troponins but EKG was concerning for an acute anteroseptal infarct. Our differentials included unusual localized pericarditis vs LIMA to LAD occlusion vs spasm. Over the following days his symptoms improved and EKG began to

normalize. He was discharged to cardiac rehab a few days later.

**Discussion**

Regional pericarditis has been described following abdominal surgeries, invasive cardiac procedures and post MI. Pericarditis is the most common cause of chest pain following an acute MI. Hypothesized to be an autoimmune response, diagnosis is difficult as findings are nonspecific. Pericardial effusions may be seen in 43% of patients following an MI and 85% after CABG. Localized irritation of the pericardium produces focal ST-segment elevations, making differentiation from an acute STEMI extremely difficult, as was seen in our case. Diffuse PR-segment depressions with PR-segment elevation in aVR are helpful findings more specific to pericarditis and also seen in the regional variant. Other causes of ST-segment elevations including hyperkalemia, early repolarization, and in post-CABG early graft occlusion must also be considered. Friction rub is much less common with an incidence of 13%. This is also transient in nature, and can be easily confused with mediastinal rub (surgical emphysema) frequently seen after cardiac surgery. Treatment consists of NSAIDs, Colchicine or Prednisone for refractory cases, but our patient improved over time without these measures.

**Conclusion**

With a lack of diagnostic and EKG criteria the frequency of regional pericarditis remains unknown. However, prompt recognition can save the patient unnecessary risk associated with invasive testing. Misdiagnosis can potentially lead to a free wall rupture secondary to acute pericarditis after thrombolytic therapy. Further studies are needed to develop comprehensive consensus criteria for diagnosis of regional pericarditis.

**Categories**

3rd year Fellow: Case

**Program Name**

Summa Health System

Pharmacomechanical thrombolysis, adjunctive balloon venoplasty and thoracic outlet decompression for Paget-Schroetter syndrome.

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<sup>1</sup>University of Toledo, Toledo, USA. <sup>2</sup>University of Pennsylvania, Philadelphia, USA

**Type of submitter**

Fellow in Training

**Abstract**

Introduction:

Paget-Schroetter syndrome or ‘venous thoracic outlet syndrome’ is a spontaneous upper extremity thrombosis that is due to a compressive anomaly at the thoracic outlet between the first rib and clavicle, a hypertrophic scalene or a subclavius muscle or tendon. Anatomic abnormalities can be congenital or acquired. It is a rare condition that typically presents in a young patient as sudden severe upper extremity swelling and pain. An aggressive treatment approach including anticoagulation, catheter-directed thrombolysis and thoracic outlet decompression is aimed at relieving the acute symptoms and reducing the risk of post-thrombotic syndrome.

Case:

A 38-year-old delivery man, with no past medical history, presented with right upper extremity swelling for two weeks. He noticed significant worsening of the swelling with color changes of the right hand for 48 hours as well as pain when raising his right upper extremity above his head with tingling and numbness in his fingers. On examination, his right upper extremity was swollen, tense and appeared plethoric. Duplex ultrasonography revealed non-compressible veins with extensive thrombus in the axillary, subclavian, and brachiocephalic veins. A Heparin infusion was started. Given his symptoms and signs, it was elected to proceed with an aggressive invasive approach.

Pharmacomechanical thrombolysis utilizing Angiojet power-pulse spray of t-PA, thrombectomy and adjunctive balloon venoplasty were performed. Completion venography showed widely patent veins with significant residual stenosis of the right subclavian vein consistent with venous thoracic outlet syndrome/Paget-Schroetter syndrome. The patient’s symptomatology improved significantly. Subsequently, he was scheduled for right 1st rib resection and thoracic outlet decompression during his hospitalization.

Discussion:

Primary upper extremity deep vein thrombosis is rare with an estimated annual incidence of 1 to 2 cases per 100,000 population. Diagnosis may be suspected based upon the clinical presentation, but should be confirmed with imaging, typically initially using ultrasound. Treatment of Paget-Schroetter syndrome involves relief of acute symptoms and prevention of recurrence and long term complications. Optimal care, particularly in patients with moderate to severe symptoms, usually includes anticoagulation, catheter-directed thrombolysis and subsequent surgical decompression. Pharmacomechanical thrombolysis may allow treatment in a single session. Prevention of recurrent episodes requires treatment of the underlying condition. In this patient thoracic outlet decompression via resection of his 1st rib was performed.

**Categories**

3rd year Fellow: Case

**Program Name**

University of Toledo

Pneumo-pericardium

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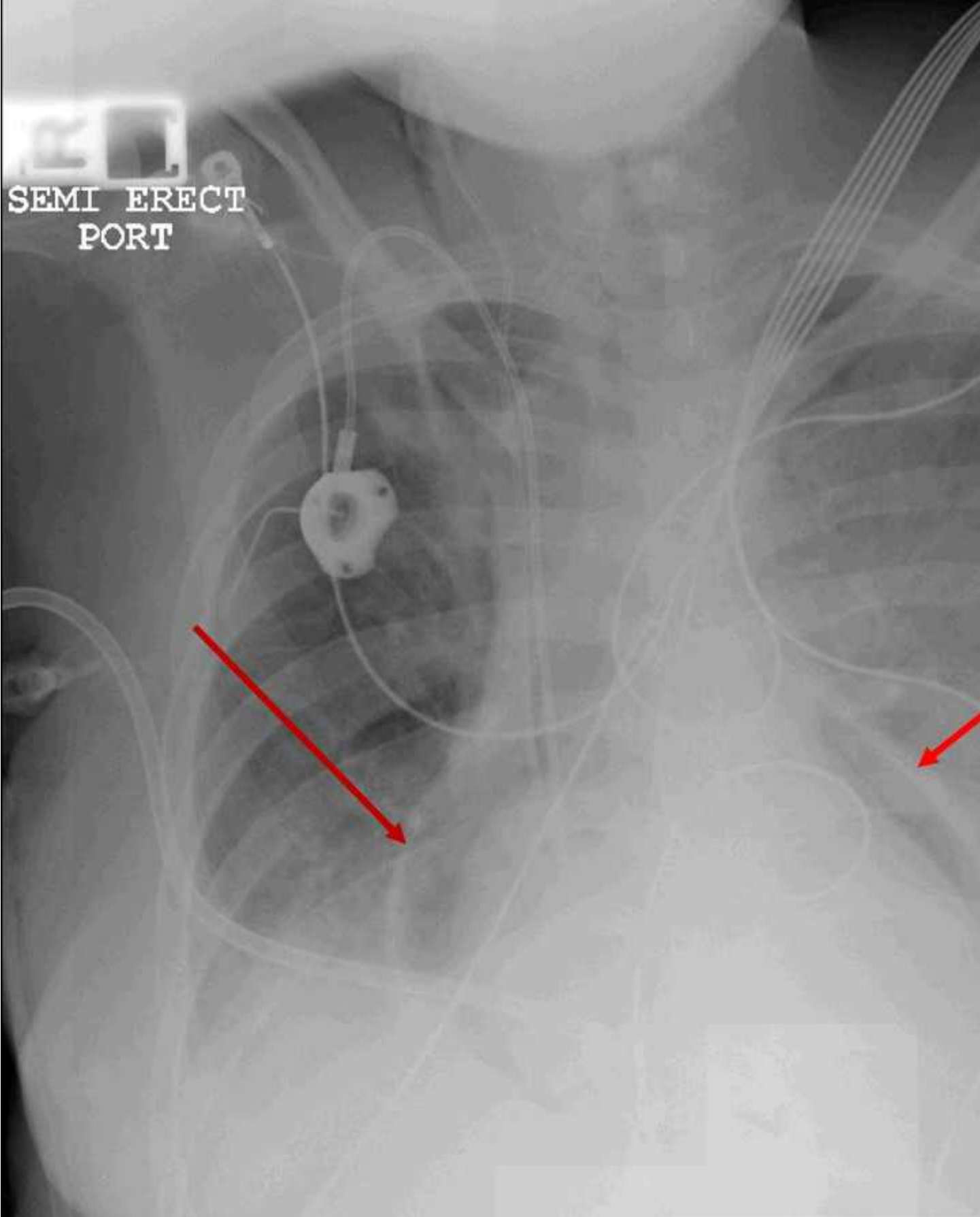
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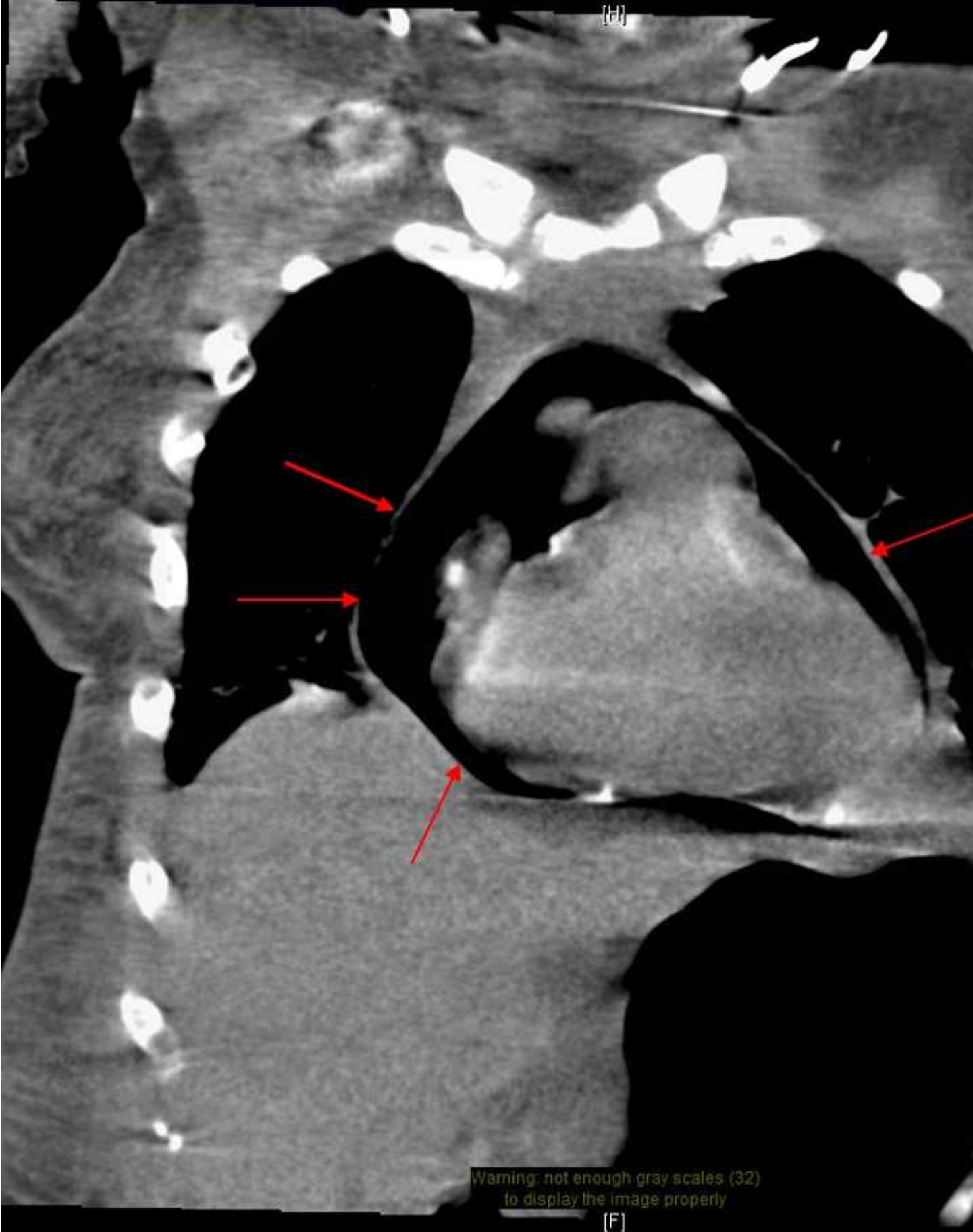
Fellow in Training

**Abstract**

A 32 year old female with known recurrent stage IIIC ovarian cancer presented to the hospital with worsening shortness of breath over a two week period. She was found to have bilateral pleural effusions and a pericardial effusion. Her hospital course was complicated by worsening hypoxic respiratory failure requiring intubation. She was later extubated, however, on a routine chest radiograph, was found to have a pneumopericardium (Figure 1). This was followed up by a computed tomographic scan of her chest confirming the diagnosis (Figure 2 and Figure 3). She was taken to the operating room where an esophageal perforation was identified and an esophageal stent was placed as well as bilateral chest tubes, and a pericardial window procedure was performed. The esophageal perforation thought to be due to orogastric tube placement after intubation. The patient tolerated the procedure well, and is currently still hospitalized in improved condition.

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**Categories**

3rd year Fellow: Case

**Program Name**

Metrohealth Medical Center - Case Western Reserve Hospital