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October 2021 Critical Care Case of the Month: Unexpected Post-Operative Shock

Sharanyah Srinivasan MBBS
Sooraj Kumar MBBS
Benjamin Jarrett MD
Janet Campion MD

University of Arizona College of Medicine, Department of Internal Medicine and Division of Pulmonary, Allergy, Critical Care and Sleep Medicine, Tucson, AZ USA

History of Present Illness

A 55-year-old man with a past medical history significant for endocarditis secondary to intravenous drug use, osteomyelitis of the right lower extremity was admitted for ankle debridement. Pre-operative assessment revealed no acute illness complaints and no significant findings on physical examination except for the ongoing right lower extremity wound. He did well during the approximate one-hour "incision and drainage of the right lower extremity wound", but became severely hypotensive just after the removal of the tourniquet placed on his right lower extremity. Soon thereafter he experienced pulseless electrical activity (PEA) cardiac arrest and was intubated with return of spontaneous circulation being achieved rapidly after the addition of vasopressors. He remained intubated and on pressors when transferred to the intensive care unit for further management.

PMH, PSH, SH, and FH

- S/P Right lower extremity incision and drainage for suspected osteomyelitis as above
- Distant history of endocarditis related to IVDA
- Not taking any prescription medications
- Current smoker, occasional alcohol use
- Former IVDA
- No pertinent family history

Physical Exam

- Vitals: 100/60, 86, 16, afebrile, 100% on ACVC 420, 15, 5, 100% FiO2
- Sedated well appearing male, intubated on fentanyl and norepinephrine
- Pupils reactive, nonicteric, no oral lesions or elevated JVP
- CTA, normal chest rise, not overbreathing the ventilator
- Heart: Regular, normal rate, no murmur or rubs
- Abdomen: Soft, nondistended, bowel sounds present
- No left lower extremity edema, right calf dressed with wound vac draining serosanguious fluid, feet warm with palpable pedal pulses

- No cranial nerve abnormality, normal muscle bulk and tone

Clinically, the patient is presenting with post-operative shock with PEA cardiac arrest and has now been resuscitated with 2 liters emergent infusion and norepinephrine at 70 mcg/minute.

What ***type of shock*** is most likely with this clinical presentation?

1. Cardiogenic shock
2. Hemorrhagic shock
3. Hypovolemic shock
4. Obstructive shock
5. Septic / distributive shock

Correct!
4. Obstructive shock

Shock is a life-threatening manifestation of circulatory failure leading to cellular and tissue hypoxia (1). It is a life-threatening condition and most commonly manifested as hypotension (systolic blood pressure less than 90 mm Hg or MAP less than 65 mmHg). There are mainly four broad categories of shock: distributive, hypovolemic, cardiogenic, and obstructive. The wide range of etiologies can contribute to each of these categories. Undifferentiated shock means that the diagnosis of shock has been made; however, the underlying etiology has not been uncovered.

In this case of undifferentiated shock, we suspected obstructive shock because of the history of endocarditis and the rapid increase in blood pressure to 2 liters of fluid.

Obstructive shock is usually due to a decrease in the left ventricular cardiac output due to pulmonary or mechanical causes.

1. Pulmonary vascular - due to impaired blood flow from the right heart to the left heart. Examples include hemodynamically significant pulmonary embolism, severe pulmonary hypertension.
2. Mechanical - impaired filling of right heart or due to decreased venous return to the right heart due to extrinsic compression. Examples include tension pneumothorax, pericardial tamponade, restrictive cardiomyopathy, constrictive pericarditis.

What is ***appropriate to do next***?

1. Blood cultures with empiric antibiotics
2. Echocardiogram
3. CT Chest / Angio
4. 1 and 3
5. All of the above

Correct!
5. All of the above

In order to differentiate the type of shock all may be necessary (1). Initially, there was high suspicion for a massive pulmonary embolism and CT/Angio of the chest with contrast was obtained to evaluate this further (Figure 1).

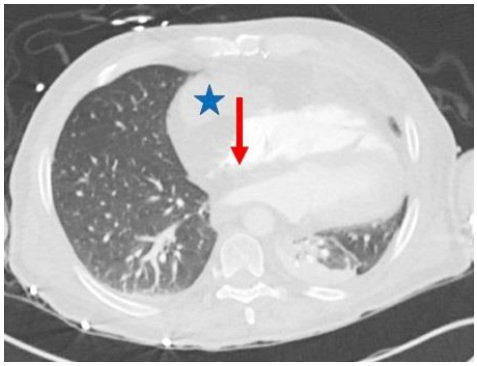

Figure 1. Representative image from the thoracic CT with contrast showing an anterior mediastinal mass (blue star) associated with extensive infiltration around the interatrial septum (red arrow) and under the pericardium.

The results were negative for pulmonary embolus but did reveal an anterior mediastinal mass associated with extensive lipomatous infiltration around the intra-atrial septum and under the pericardium, with evidence of right atrial and ventricular compression (Figure 1).

A point of care (POC) ultrasound of the heart demonstrated significant inspiratory collapse with near total occlusion of the venous inflow into the right atrium, which responded well to increased preload through volume resuscitation and decreased positive end expiratory pressure. he was extubated the same day, which further improved his systemic hemodynamics.

In addition, the POC ultrasound revealed an increase in the size of the interventricular septum (Figure 2).

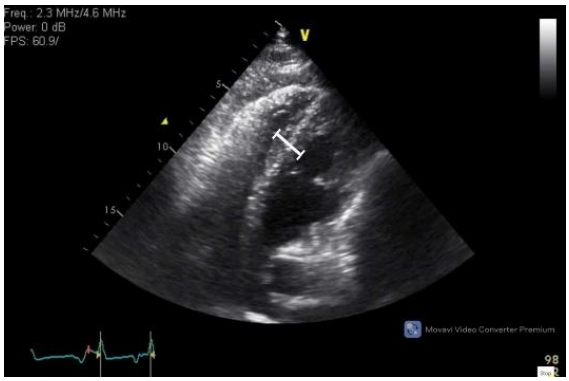

Figure 2. Static image form point of care ultrasound showing a markedly widened vetricular septum of about 1.8 cm (normal < 1.1 cm).

What is the ***most likely diagnosis***?

1. Atrial liposarcoma
2. Congestive heart failure
3. Left atrial myxoma
4. Lipomatous hypertrophy
5. Pulmonary tumor embolus

Correct!
4. Lipomatous hypertrophy

Lipomatous hypertrophy is characterized by fatty infiltration of the interatrial septum. It can be a rare case of obstructive shock due to inflow tract obstruction.

Although the POC ultrasound revealed an increase in the size of the interventricular septum, the cellular cause of the increase in the size was unclear. This could either be due to hypertrophy of the cardiac muscle or an infiltration of fat cells.

Which of the following can show fat infiltration of the interventricular septum?

1. Cardiac biopsy
2. Electrocardiogram
3. Cardiac MRI
4. 1 and 3
5. All of the above

Correct!
4. 1 and 3

MRI diagnosis is straightforward in classic cases of lipomatous hypertrophy and is characterized by a bilobar interatrial septal thickening revealing homogeneous high signal intensity similar to that of subcutaneous fat tissue.

The exclusively fatty nature of such masses can be seen on fat-suppressed imaging. In our patient, MRI showed that the anterior mass previously seen was consistent with the left lobe of liver protruding through a Morgagni hernia defect and showed that lipomatous infiltration was extensive, invading the superior and inferior cavoatrial junctions and both proximal vena cavae (Figure 3).

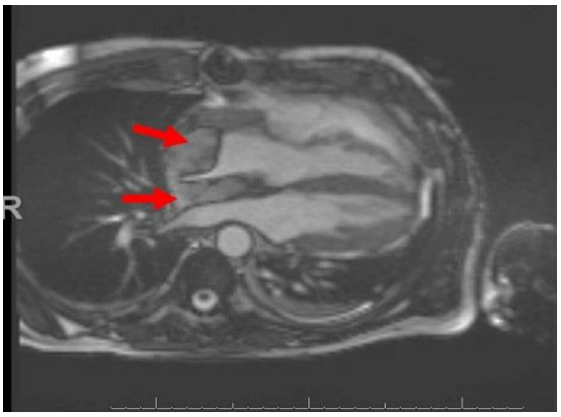

Figure 3. Lipomatous infiltration (arrows) of interatrial septum extending along SVC, IVC with compression of SVC at superior cavo-atrial junction. Three cm mass extension into right atrial wall with sparing of fossa ovalis.

Which of the following is/are ***true*** regarding lipomatous hypertrophy?

1. Lipomatous hypertrophy is being diagnosed more frequently with use of transthoracic echocardiography
2. Improved by preload reduction
3. Most cases are asymptomatic
4. 1 and 3
5. All of the above

Correct!
4. 1 and 3

Lipomatous hypertrophy of the interatrial septum was previously considered a rare entity with an incidence rate of 2.2 to 8% as diagnosed with transthoracic and transesophageal echocardiograms, respectively (2). Most individuals with this condition are asymptomatic, and that is presumably the reason for its underdiagnosis. Unlike lipomas, lipomatous hypertrophy is not encapsulated and as a result is able to infiltrate myocardial fibers causing disturbances in conduction system and hemodynamics of the heart. In symptomatic patients, manifestations can include superior vena cava syndrome, cardiac arrhythmias, pericardial effusion, heart failure and sudden cardiac death (2).

Answer 2 is wrong because patients who are hypotensive from inflow obstruction respond to increasing preload and not reduction. Our patient responded well to increased preload through volume resuscitation and decreased positive end expiratory pressure. He was extubated the same day, which further improved his systemic hemodynamics. The patient received cardiac MRI to further delineate the exact nature and location of the mass, and identify locally affected structures.

The patient later confirmed that he had experienced a similar episode of syncope 15 years ago in Denver. He recalls cardiac biopsy was performed and was told it was "fat" consistent with CT Chest findings of extensive lipomatous infiltration. Pericardial takedown was attempted at that time was unsuccessful.

It is important to note that while transthoracic echocardiography can confirm the presence of a cardiac mass-like lesion, further characterization requires cardiac CT angiography or cardiac MRI (4). Characteristic features on CTA imaging include minimal to no contrast enhacement, septal location and sparing of the fossa ovalis (4). Biopsy is usually not required for diagnosis (4).

Our working theory is that the lipomatous infiltration decreased preload by compressing the inflow tracts and right atrium, compounded by increased cardiac demand due to peripheral vasodilation which resulted from the release of toxic metabolites and cytokines after torniquet removal.

Indications for treatment include marginal obstruction of superior vena cava or right atrium (5). intervention of choice is surgical excision of the lesion and septum plasty (5).

Based on our experience, a decrease in preload should be especially be avoided in these patients, especially in the setting of possible inflow tract obstruction due to lipomatous infiltration.

Given the patient's extensive infiltration, history of unsuccessful surgery in the past and dramatic response to an increased preload, surgery was not attempted. he was discharged with a course of antibiotics for osteomyelitis.

References

1. Standl T, Annecke T, Cascorbi I, Heller AR, Sabashnikov A, Teske W. The Nomenclature, Definition and Distinction of Types of Shock. Dtsch Arztebl Int. 2018 Nov 9;115(45):757-768. [CrossRef] [PubMed]
2. Xanthopoulos A, Giamouzis G, Alexopoulos N, Kitai T, Triposkiadis F, Skoularigis J. Lipomatous Hypertrophy of the Interatrial Septum: A Case Report and Review of the Literature. CASE (Phila). 2017 Aug 23;1(5):182-189. [CrossRef] [PubMed]
3. Hejna P, Janík M. Lipomatous hypertrophy of the interatrial septum: a possibly neglected cause of sudden cardiac death. Forensic Sci Med Pathol. 2014 Mar;10(1):119-21. [CrossRef] [PubMed]
4. Ribeiro RNF, Ribeiro BNF, Martins WA, Antunes LO, Marchiori E. Lipomatous hypertrophy of the interatrial septum. Radiol Bras. 2018 Mar-Apr;51(2):130-131. [CrossRef] [PubMed]
5. Bielicki G, Lukaszewski M, Kosiorowska K, Jakubaszko J, Nowicki R, Jasinski M. Lipomatous hypertrophy of the atrial septum - a benign heart anomaly causing unexpected surgical problems: a case report. BMC Cardiovasc Disord. 2018 Jul 28;18(1):152. [CrossRef] [PubMed]