An Unusual Case of Acute Aortic Insufficiency from a Torn Non-Coronary Cusp

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ABSTRACT

Background: Aortic Insufficiency (AI) is a valvular heart disease leading to inadequate closure of the valve leaflets. There are many different etiologies of AI.

Case presentation: This case addresses an unknown etiology of acute AI from a torn non-coronary aortic cusp with associated acute heart failure that was ultimately treated with an aortic valve replacement.

Conclusion: Regardless of the etiology, prompt diagnosis, stabilization, and treatment are paramount to preventing progressive heart failure.

Keywords: Aortic insufficiency; Acute aortic insufficiency; Torn non-coronary cusp; Acute heart failure; Etiology

BACKGROUND

Aortic Insufficiency (AI) is a valvular heart disease in which the integrity of the aortic valve is compromised by inadequate closure of the valve leaflets leading to retrograde blood flow from the aorta into the left ventricle. Typical etiologies are infective endocarditis, traumatic and non-traumatic rupture of the ascending aorta, and iatrogenic injury [1]. One source of traumatic AI, Blunt Cardiac Trauma (BCI), is a very rare cause and the aortic valve is the most common involved valve in BCI patients. The tear/avulsion from the annulus frequently occurs at the non-coronary cusp [2-4]. Thus, the presentation of the injury is usually severe and acute in nature [5]. Furthermore, there are often concomitant injuries to other cardiac structures [6]. In contrast, atypical causes of AI include medications such as dopamine or other ergot alkaloids. These agents increase cardiac contraction, increase stress on the aortic valve, and increase valvular fibrosis thereby putting the valve at higher risk for complications [7]. Regardless of the etiology, diagnosis and treatment are paramount.

CASE PRESENTATION

A 39-year-old female presented to the emergency department with progressive shortness of breath and a productive cough. She had a history significant for a motor vehicle accident complicated by a sternal fracture and right wrist fracture where she required intubation for several days for a streptococcal pneumonia [8]. Her past medical history was also notable for methamphetamines and cocaine usage. On this admission, she was tachycardic, lethargic, diaphoretic, and disoriented. Labs were notable for metabolic acidosis with a lactic acid of 11 pH of 7.16, and bicarbonate of 7.6. Her white blood cell count was 14 K, creatinine 1.49, potassium 5.3, and a worsening transaminitis.

A bedside echocardiogram was notable for moderate tricuspid valve regurgitation, moderate mitral regurgitation, and severely dilated left ventricle volume index 99 mL/m² with Left Ventricular Ejection Fraction (LVEF) of 55% (Figures 1-3). The patient was admitted to the intensive care unit, central venous access was placed, and an inotropic agent was started. Cardiothoracic Surgery was consulted and she was taken to the operating room for urgent cardiac surgery [9-10].

Operative technique

After induction of anesthesia, the preoperative Trans-Esophageal Echocardiogram (TEE) demonstrated torrential aortic regurgitation with a nearly absent non-coronary cusp, and an ejection fraction of 25% on dobutamine and epinephrine (Figures 4 and 5). Intraoperatively, the pericardium was incised and suspended by a sternal fracture and right wrist fracture where she required intubation for several days for a streptococcal pneumonia [8]. Her past medical history was also notable for methamphetamines and cocaine usage. On this admission, she was tachycardic, letheargic, diaphoretic, and disoriented. Labs were notable for metabolic acidosis with a lactic acid of 11 pH of 7.16, and bicarbonate of 7.6. Her white blood cell count was 14 K, creatinine 1.49, potassium 5.3, and a worsening transaminitis.

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right atrium and right ventricle were massively dilated indicating signs of right heart failure [11-13]. The patient was then heparinized, cannulated via the aortic arch, and right atrial appendage. After satisfactory ACT, normothermic cardiopulmonary bypass was instituted. The ascending aorta was clamped and the heart was arrested. The ascending aorta was transected, the native aortic valve was tricuspid and the coronary ostia were all normal. The non-coronary cusp was observed to be detached from the annulus in a clean, near complete tear down to the nadir (Figure 6). There was no vegetative tissue or abscess noted. The aorto-mitral curtain was normal, and there was no evidence of infection or abscess. The native aortic valve was then completely resected and submitted to pathology and microbiology and a 23 mm pericardial tissue valve was implanted in a supra-annular fashion using interrupted and pledgeted, horizontal mattress sutures [14-16]. The heart was de-aired, the cross-clamp was removed, and cardiac activity was resumed. A postoperative TEE showed normally functioning aortic valve prosthesis without regurgitation and a depressed left ventricular ejection fraction with cardiomegaly. The patient tolerated the procedure and was taken to the ICU in stable condition [17,18].

**Figure 1:** It is a TTE 3 chamber view which demonstrate the aortic regurgitant jet, with a large jet area.

**Figure 2:** It is a TTE, Parasternal long axis, showing the vena contracta.
Figure 3: TTE modified apical 2 views which show the severely dilated left ventricle with a volume index of 99 mL/m².

Figure 4: TEE-Mid esophageal, long axis. Which demonstrate the regurgitant across the Left Ventricular Outflow Tract (LVOT).

Figure 5: Which shows a TEE mid esophageal, short axis at the aortic valve which demonstrates the aortic valve regurgitation in the non-coronary cusp.
Post-operative care

The patient was extubated by post-operative day #0 and vasoactive medications were weaned off thereafter. Dobutamine was slowly weaned to off over several days [19]. Intraoperative microbiology and pathology results were negative. She was transferred to the cardiothoracic progressive care unit on postoperative day #3. The heart failure service was consulted for her reduced ejection fraction and she was started on goal-directed medical therapy [20]. A limited TTE was repeated and the patient's ejection fraction improved from 25% to 41%. She was eventually discharged home once her level of activity was satisfactory.

DISCUSSION AND CONCLUSION

Worldwide, the leading cause of aortic insufficiency is rheumatic disease. However, in Western Europe and North America, the leading cause of AI is either congenital or degenerative disease. The incidence increases with age and is more common in men than women. Per the Framingham study, the prevalence of AI in the western world was reported to be 4.9%, with regurgitation of moderate or greater severity occurring at approximately 0.5%. Severe AI is known to have increased morbidity and mortality compared to the general population, and with conservative management, the majority of patients will develop heart failure in ten years.

The causes of acute AI differ from chronic AI in etiology and presentation. In chronic AI, the left ventricle dilates and remodels to maintain forward stroke volume, cardiac output, and Left Ventricle End-Diastolic Pressure (LVEDP). In contrast, remodeling does not occur in acute regurgitation. The normal ventricular size and a marked increase in LVEDP lead to impaired forward stroke volume, decreased systolic pressure, and narrow pulse pressure. The events that ensue are ineffective forward stroke volume, compensatory tachycardia, hypotension, end-organ failure, and other evidence of cardiogenic shock.

In this case report, we are uncertain as to the exact etiology of her acute AI. The possible mechanisms are either BCI or an acute hypertensive event given her prior illicit drug use. BCI occurs from several mechanisms: The direct insult to the chest such as steering wheel trauma leading to compression of the heart against the vertebral column, acceleration-deceleration impact leading to a torsion at the fixation sites such as the pulmonary trunk, hydraulic effect transmitting the increased intra-abdominal pressure to the right atrium via the draining Inferior Vena Cava (IVC) or a blast injury. The mechanism for the BCI in this patient is presumably a motor vehicle accident resulting in sternal fracture and direct chest trauma. This trauma causes a sudden increase in intrathoracic pressure during diastole, when the pressure gradient across the aortic valve is maximal and the valve is closed, resulting in an aortic valve rupture.

In contrast, there have been a few case reports describing aortic rupture from a hypertensive episode. Ayogi, et al. described two cases of severe aortic regurgitation due to non-traumatic rupture of the aortic valve commissures. Both patients underwent aortic valve replacement successfully. Our patient is prone to hypertensive episodes as a side effect of her methamphetamines and cocaine use. However, there has been no reported case reports of either illicit drug directly causing a valve rupture without the presence of an associated aortic dissection.

Regardless of the mechanism, prompt diagnosis, stabilization, and treatment are imperative because of rapid deterioration and heart failure secondary to acute aortic regurgitation. Medical management is directed towards reducing pulmonary venous congestion and optimizing cardiac output in an attempt to stabilize the hemodynamics before surgery. Invasive monitoring such as a pulmonary artery catheter is also useful for monitoring hemodynamics and directing medical therapy. Echocardiography is the noninvasive test of choice. If the patient is stable, TEE achieves better visualization of the valvular anatomy and myocardial function. Cardiac catheterization remains the gold standard for providing information concerning aortic insufficiency including abnormalities associated with the aortic root and coronary vessels in stable patients. Ultimately when there is hemodynamic instability, timely surgical intervention is warranted in an urgent fashion. This case represents an acute aortic insufficiency from unknown etiology. Our patient was identified early and treated expeditiously with an excellent outcome.
AUTHORS’ CONTRIBUTIONS

C.M wrote the Case Presentation, Operative Technique, Discussions and Conclusions, and reviewed the manuscript. T.T wrote the introductions, prepared the figures, reviewed the manuscript and made edits. C.B wrote the abstract, prepared the references, reviewed the manuscript and made edits. D.B reviewed the manuscript and made edits. K.B reviewed the manuscript and made edits. E.D reviewed the manuscript and made edits.

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