Asymptomatic Chronic Traumatic Aortic Valve Perforation with Severe Aortic Regurgitation

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April 28, 2020

Abstract

Here we present a young asymptomatic male incidentally diagnosed to have aortic regurgitation (AR). The patient had a history of a blunt trauma to the thorax two years back but did never have any symptoms. Transthoracic echocardiography showed a moderately dilated left ventricle with normal systolic function and severe AR with normal nondilated aortic root and tri-leaflet aortic valve. To diagnose the etiology of the AR a trans-esophageal echocardiogram (TEE) was done, which revealed a perforation in the non-adjacent leaflet (NAL) and confirmed severe AR with two AR jets being clearly visualised, one through the point of incomplete coaptation and other one through the perforated area in the NAL. The patient was treated with aortic valve replacement and was doing well on follow-up.

Introduction

Aortic leaflet perforation leading to aortic regurgitation is a known complication of blunt chest trauma. There are some case series[1-2] and case reports[3-12]describing such cases. These patients report either immediately or within weeks of the trauma, with dyspnea and chest pain. They need to be operated urgently with either aortic valve replacement or repair as found suitable. Here we describe a patient who was asymptomatic 2 years after blunt chest trauma and was incidentally detected to have severe aortic regurgitation (AR) caused by a tear in the non-adjacent leaflet (NAL).

Case Report

A 26-year-old man was found to have cardiomegaly on chest x-ray on a routine pre-employment health check-up. He was asymptomatic with no history suggestive of any cardiac illness or any pre-existing co-morbidity. He did not have a history of fever, weight loss, drug abuse or rheumatic fever. However, on repeated interrogation he remembered blunt trauma to his face and chest due to a vehicular accident 2 years ago. He did not have a Marfanoid habitus. The BP was 140/40 mm Hg. The pulse rate was 100/min, regular and collapsing in character; pistol shot sounds were also present. A Grade 4/6 high pitched moderately long diastolic murmur was heard in the left third intercostal space.

Echocardiography revealed a moderately dilated left ventricle with normal systolic function. There was severe AR with significant diastolic reversal of flow in descending thoracic aorta. The aortic valve was tri-leaflet, with no significant dilation of the aortic root (Video 1). The Effective Regurgitant Orifice of the AR jet by the Proximal Isovelocity Surface Area method was 0.5 cm^2 with a regurgitant volume of 96 ml/beat. The pressure half time of the AR jet was only 250 ms. The right coronary leaflet appeared elongated and the NAL showed everted edges (Video 2). There was no evidence of rheumatic affection or endocarditis.

We performed a Trans Esophageal Echocardiography (TEE) to delineate the etiology of AR and aortic valve pathology. Normal tri-leaflet valve and aortic root were confirmed (Figure1, Video 3) and there was evidence of tissue gap in the NAL suggestive of perforation (Figure 2). There was no vegetation. Colour Doppler study confirmed severe AR with two AR jets being clearly visualised, one through the point of incomplete coaptation and other one through the perforated area in the NAL (Figure 3; Videos 4). He underwent surgery; intra-operatively the large perforation of the NAL was confirmed (Figure 4). There was no evidence of endocarditis or calcification. The valve was replaced with a 21 mm St Jude Trifecta tissue valve. Post-operative recovery was uneventful, and he is doing well at 18 months follow up.

Discussion

The basic pathophysiology behind post traumatic AR can be either acute rupture of the thoracic aorta at the isthmus where the mobile thoracic aorta joins the firmly fixed aortic arch [13,14], leaflet injury [3-12] or avulsion of the commisures[15]. The mechanism behind such injuries involves increased pressure inside the aortic root with a closed aortic valve and low left ventricular pressure in diastole, leading to high transvalvular gradient. The left coronary sinus being a more posterior structure is less commonly involved in such trauma[6]. Since the left and right coronary sinuses have the origin of coronary arteries, they are pressure release areas; thus, the non-adjacent sinus bears the major brunt and a tear of the NAL is commonest pathology[5].

Patient presenting late with AR after blunt trauma to chest[5,8] have been described in literature, but they are mostly upto few months. Patients presenting late mostly have a tear in one of the leaflets which is small and insignificant to start with and gradually progresses with time. In these patients the collagen in the spongiosa is gradually replaced by mucopolysaccharides leading to myxomatous degeneration and progressive weakness of the leaflet, causing further damage[16]. Our case is unique because a patient remaining asymptomatic for 2 years after trauma and incidentally being diagnosed with severe AR has not been reported.

Management of patients presenting late with severe asymptomatic AR is always a matter of debate and traditionally onset of ventricular dysfunction, acute pulmonary edema, and a systemic diastolic blood pressure of less than 50 mm Hg are indications for immediate surgery.[17] Waiting for symptoms or ventricular dysfunction might lead to some irreversible ventricular damage not correctable by surgery[10]; thus early surgery in asymptomatic patients might be a pragmatic option[6] as done in our patient. Another point regarding surgery requires a special mention. Traditionally valve replacement was the standard approach in such patients, but multiple repair techniques are now being explored in patients with limited damage to a single leaflet[18]. However, the assessment of the valve leaflets preoperatively by imaging or intra-operatively by hand and eye assessment are imperfect as there is a report of microscopic degeneration in an otherwise healthy-looking leaflet, which could progress with time[19].Thus, valve replacement is the standard approach in these cases.

Conclusion

A detailed history taking is always important to solve a medical puzzle. The etiology of the case would have been overlooked had it not been for the history of blunt trauma two years prior to presentation. We should also keep in mind the fact that traumatic AR can present late and these patients can rarely remain asymptomatic. In patients with severe AR, without any evidence of rheumatic heart disease, endocarditis or a bicuspid valve and with an AR jet though a leaflet should raise the suspicion of traumatic AR. TEE is mandatory and diagnostic as it readily confirms the pathology. Surgical management should be the general approach with valve replacement being the standard treatment of choice.

Conflict of interest: None

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Figure and Video Legends

Figure 1: TEE showing tri-leaflet aortic valve with a normal aortic root in mid-esophageal long axis view

Figure 2: TEE showing a tissue gap in the non-adjacent leaflet of the aortic valve suggestive of perforation in mid-esophageal long axis view

Figure 3: TEE showing severe AR. Two AR jets are clearly visualised; one through the point of incomplete coaptation and the second through the perforation in the non-adjacent leaflet.

Figure 4: Pathologic specimen showing the three leaflets with a large perforation in the non-adjacent leaflet

Video 1: Echocardiogram in modified apical 5 chamber view showing tri-leaflet aortic valve without any significant dilation of the aortic root

Video 2: Echocardiogram in parasternal long axis view showing elongated right coronary leaflet and everted edges of the non-adjacent leaflet, with severe AR

Video 3: TEE showing tri-leaflet aortic valve in mid-esophageal long axis view with a tissue gap in the non-adjacent leaflet of the aortic valve suggestive of perforation without any significant dilation of the aortic root.

Video 4: TEE with Colour Doppler study confirming severe AR with two AR jets, one through the point of incomplete coaptation and other one through the perforation in the non-adjacent leaflet.