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Research Letter

Acute anterior wall myocardial infraction in asymptomatic severe aortic Coarctation in young adult – A rare case

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ABSTRACT

Coarctation of Aorta is not rare in general population. Aortic Coarctation represents about 5–8% of all congenital cardiac diseases, and it is commonly associated with bicuspid aortic valve. Coarctation of Aorta is typically a disease of childhood and early adulthood, reducing life expectancy in patients, who have not undergone correction. Many case reports of Coarctation of Aorta patients presenting with anterior wall myocardial infraction have been published in various journals, but to the best of our knowledge, no case of Coarctation of Aorta with acute anterior wall myocardial in 30-year-old male is reported till date. We are presenting a rare case of anterior wall myocardial infraction in young male with asymptomatic Coarctation of Aorta.

1. Introduction

Coarctation of Aorta accounts for 5–8% of all congenital heart diseases and occurs more frequently in males.[1] Coarctation of Aorta is typically a disease of childhood and early adulthood, reducing life expectancy in patients, who have not undergone correction. Death in patients, who do not undergo repair is usually because of heart failure, coronary artery disease, aortic rupture/dissection, infective endocarditis/endarteritis or cerebral hemorrhage. Multiple studies have shown that the main cause of death in patients with corrected Coarctation of Aorta is coronary artery disease,[2–4] but no studies were designed to determine whether Coarctation of Aorta is an independent risk factor for coronary artery disease. According to some studies, Coarctation of Aorta is associated with accelerated or premature coronary artery disease despite repair.[5,6] However patients with Coarctation of Aorta present with coronary artery disease in late adulthood. To the best our knowledge, anterior wall myocardial infraction in asymptomatic severe Coarctation of Aorta in this early age is not reported yet. That is why, we are presenting this case here.

2. Case report

A 30-year-old male presented to the emergency department with history of chest pain of 2 days back, diagnosed as acute anterior wall myocardial infraction. He was not thrombolyzed because of late presentation. He was not having chest pain at the time of presentation. He denied any history of alcohol or smoking or intravenous drug use. He had no past history of hypertension and diabetes mellitus. Physical examination showed blood pressure 164/90 mmHg in right arm and 160/90 in left arm, a heart rate of 90 beat per minute. ECG revealed ST elevation with T wave inversion in leads V1, V2, V3, V4 and V5. Cardiac markers were positive. His echocardiogram showed segmental wall motion abnormalities in left anterior descending artery territory, concentric left ventricular hypertrophy, tricuspid aortic valve, ascending aortic diameter (24 mm). The left ventricular ejection fraction was significantly reduced (30–35%). Owing to the severe left ventricular systolic dysfunction, and young male, the patient was being planned for a diagnostic cardiac catheterization to evaluate his coronary artery disease. Coronary angiography was planned through the right femoral artery as usual. But on vascular examination, his both femoral pulses were absent. Therefore, coronary angiography was performed through the right radial artery. The coronary angiography showed 100% complete occlusion of left anterior descending artery. Rest of the arteries were normal. Aortography showed normal aortic root, and a significant stenosis in the thoracic descending aorta. The gradient through this stenosis was measured as 70 mmHg. Next day echocardiography was done again and on supra sternal view (Fig. 1) gradient was measured with maximum gradient of 81 mmHg. His chest X-ray was done which showed typical "figure of 3 sign" and inferior ribs notching. Patient was referred to PET scan for left anterior descending artery territory myocardial viability. Left anterior descending artery myocardial territory was nonviable. So our plan was to do balloon aortoplasty with stenting. We did aortic angioplasty with stenting (Fig. 2). Post-angioplasty gradient was 8 mmHg. Hospital stay was uneventful. Patient was discharged with antiplatelets, statins and ACE inhibitors.

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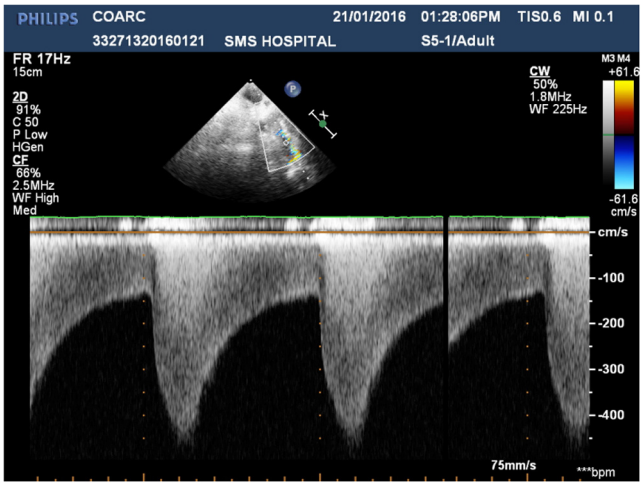

Fig. 1. Supra sternal view showing gradient across descending aorta and diastolic trail.

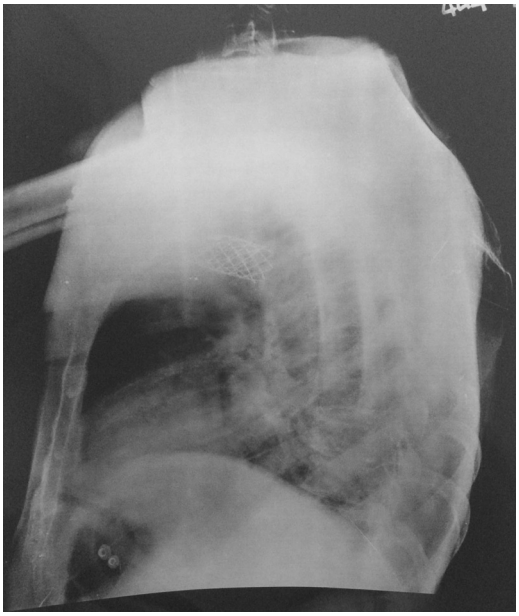

Fig. 2. Chest X-ray lateral view showing stent in descending aorta.

3. Discussion

Coarctation of Aorta is a relatively common congenital defect with apparent clinical manifestations during childhood. A significant Coarctation either requires a gradient of >20 mmHg at angiography with or without proximal systemic hypertension, or the presence of proximal hypertension in addition to angiographic or echocardiographic proof of a Coarctation.[7] Coarctation of Aorta manifests as childhood hypertension, lower extremity fatigue or weakness, diminished lower extremity pulses and/or congestive heart failure. Undiagnosed adults most commonly present with severe hypertension, which may cause symptoms such as heart failure, headaches, epistaxis, or aortic dissection.[8] There are few reports of patients first diagnosed with uncorrected Coarctation of Aorta at very late age.[9] There have been very few reports of Coarctation of Aorta presenting as acute coronary syndrome. Covens et al. described a 72-year-old patient, who presented with a myocardial infarction and had an incidental finding of a Coarctation of the Aorta with a 55 mmHg gradient. This particular patient was found to have 2-vessel disease, which was the primary cause of the MI. Some other reports showed acute coronary syndrome in asymptomatic Coarctation of Aorta in elderly.[10] But in this case report, we present aortic Coarctation with acute anterior wall myocardial infraction in a 30-year-old male. It is rare for this clinical entity to develop ACS in early age of 30 in completely asymptomatic patient of Coarctation of Aorta. The anterior wall myocardial infraction, severe left ventricular systolic dysfunction and young age of our patient, prompted us to perform a heart catheterization which lead to the incidental finding of his significant aortic Coarctation. Hence early evaluation of coronary artery disease in Coarctation of Aorta is required.

4. Conclusion

We present a rare case of acute coronary syndrome with Coarctation of Aorta. Coarctation of the Aorta does not predict for the development of coronary artery disease after adjustment for other risk factors. Rather, aging, associated hypertension, hypercholesterolemia, and diabetes mellitus predispose to coronary artery disease in this patient population. Our case is an exception. We suggest that by careful evaluation and by targeting conventional risk factors, we could possibly decrease the morbidity and mortality of these patients.

Conflicts of interest

The authors have none to declare.

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