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Case Report

Beyond the pulse: unusual presentations of Takayasu's arteritis

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ABSTRACT

Takayasu's arteritis (TA) a.k.a pulseless disease is a chronic inflammatory occlusive thrombo-aortopathy. It is a rare inflammatory disease affecting large arteries, often leading to significant diagnostic challenges, especially when presenting with atypical symptoms. This case report explores a patient, whose primary complaints were chest pain and no neurological or constitutional symptoms, diverging from the common presentations of diminished pulses or limb claudication. Advanced imaging, including CT Aortogram and interventions like cardiac angiography, played a crucial role in detecting vascular inflammation and stenosis, facilitating accurate diagnosis and management. The case underscores the importance of considering Takayasu arteritis in differential diagnoses, even with non-classical presentations. This present case demonstrates rare and unique presentation of TA where patient presents with myocardial infarction and cardiogenic shock and also explains the diagnostic dilemma of TA with such unusual presentation.

Keywords: CT Aortogram, Myocardial infarction, Takayasu's arteritis

INTRODUCTION

Takayasu's arteritis (TA) predominantly affects individuals in Asian countries, with approximately 25% of cases beginning before age 20.[1] It involves chronic inflammation of the vessel wall, leading to stenosis, thrombosis, or aneurysm formation. The late phase of TA features intimal proliferation with atherosclerosis, medial necrosis with scarring, and adventitial fibrosis.[2] Many patients with TA have anti-endothelial cell antibodies that damage vessels by inducing endothelial inflammatory cytokine production, adhesion, and apoptosis.[3] It mainly affects aorta and its branches such as subclavian artery, common carotid artery, renal artery, vertebral artery etc. TA's manifestations are diverse, ranging from constitutional symptoms like fever, malaise, light-headedness, and musculoskeletal pain to upper limb claudication, secondary hypertension, aortic regurgitation, congestive heart failure, retinal ischemia, and stroke.[4] Myocardial infarction at presentation is rare.[5] This case involves a patient presenting with NSTEMI and cardiogenic shock, without prior history or other symptoms of TA.

CASE REPORT

A 22-year-old married housewife developed mild dyspnoea on exertion on March 7, 2024. This persisted for 10 days with little discomfort and was left untreated. Few days after, she experienced acute chest pain and dizziness, leading to her admission to a private hospital in Madhubani, Bihar (On March 17, 2024). Her attendants reported no prior similar illnesses. Initial examination revealed non-palpable peripheral pulses and unrecordable blood pressure. She was dyspneic at presentation. ECG indicated NSTEMI with ST-T changes in leads V1-V6, III, and aVF, and elevated cardiac enzymes (positive Troponin T). She was treated with inotropes, nitrates,

aspirin, clopidogrel, atorvastatin and enoxaparin. Her general condition improved, and chest pain subsided, but peripheral pulses remained non-palpable, and BP was unrecordable. She was referred to Patna Medical College and Hospital, diagnosed with NSTEMI and cardiogenic shock. On March 18, 2024, at PMCH, she was enquired thoroughly and reported no prior illnesses. There was not any history of prior such illness since childhood. Her birth was uneventful. There weren't any chronic symptoms except for dyspnoea for last 10 days. Examination revealed non-palpable peripheral pulses and non-recordable BP in all limbs, mild pallor, without icterus, cyanosis, clubbing or lymphadenopathy. Chest examination revealed bilateral basal fine crepitations, and a low-intensity, soft diastolic murmur in the aortic area. She had decreased urine output. Blood tests showed moderate anaemia (9 g/dl), raised ESR (106 mm/hr), and raised CRP with normal liver and kidney function, lipid profile, thyroid profile, serum homocysteine level, ANA, ANCA, and APLA. ECG still showed ST-T changes, and chest radiography revealed cardiomegaly. Echocardiography indicated global hypokinesia of the left ventricle, left ventricular hypertrophy (18 mm), mild MR, moderate AR, and a reduced ejection fraction (37%). Abdominal ultrasonography and renal vessel Doppler were unremarkable. Next day, on March 19, 2024, her lower limb pulses became palpable, however the upper limb (radial, brachial) pulses were absent, and BP in lower limb was 100/70 mm Hg. Inotropes were tapered and discontinued as her condition improved. On further examining the patient a pronounced carotid bruit, more on the left than the right, was noted. A CT aortogram was performed and showed wall thickening of the thoracic and abdominal aorta with stenosis in the left CCA and left subclavian artery (figure 1) with aortic root dilatation (figure 2). Coronary angiography revealed no abnormalities. She was started on prednisolone 1mg/kg/day. Diuretics, Beta blockers and ARNI were started for heart failure and LVH after which she was discharged.

Follow Up

On one moth follow up, there was no relapse of symptoms and initial remission was achieved. Patient showed marked improvement in symptoms (dyspnoea on exertion and chest pain). Biochemical investigations were normal and 2D ECHO revealed no deterioration in cardiac function. Prednisolone and other medications were continued till the next follow up.

DISCUSSION

A spectrum of disorders can cause myocardial infarction (MI) in young individuals, including antiphospholipid antibody syndrome (APLA), familial dyslipidaemia, hypercoagulable states, and Kawasaki disease. Takayasu arteritis is a rare cause. Chronic inflammation of the aorta, especially the aortic arch, leads to aortic root dilatation and aortic regurgitation (AR). Long-standing AR causes left ventricular (LV) remodelling, resulting in LV dilatation and increased LV mass (hypertrophy).[6]

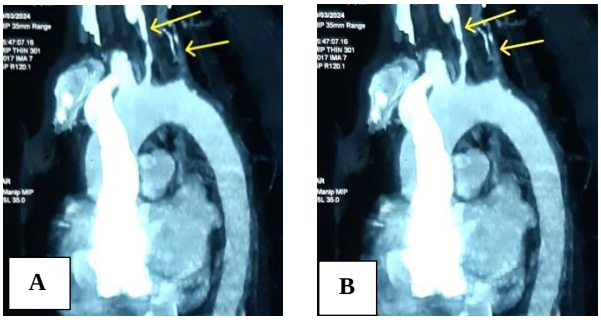

Figure 1 (A and B): CT aortogram findings.

Table 1: Examination findings.

Findings	Observations
General Examination	
Pulse	Non recordable
Blood pressure	Non recordable
Temperature	99°F
Pallor	Present
Icterus, Cyanosis, Clubbing, Lymphadenopathy, Edema	Absent
Biochemical Investigation	
CBC	10300 Cells/dl
Hb	9g/dl
LFT and KFT	Bilirubin-1.2mg/dl SGPT/SGOT-40/44 Creatinine-1.3mg/dl
ESR	109 mm/hour
Iron profile	Suggestive of anaemia of chronic disease
Reticulocyte count	0.6%
PBS	Normal, no any immature cell present
HIV/HBsAg/HCV	Non-reactive
Other Investigations	
ANA by IFA	Non-reactive
c-ANCA and p-ANCA	Non-reactive
APLA profile	Negative
USG whole abdomen and Renal artery Doppler study	Unremarkable
Arterial biopsy	Could not be performed

Prolonged LV hypertrophy and dilatation, combined with ongoing inflammation, can precipitate myocardial ischemia due to an oxygen supply-demand mismatch, potentially leading to MI. It may also result in LV aneurysm or septal/free wall rupture. In this patient, prolonged LV hypertrophy likely caused the MI. Lesion in Coronary artery causing stenosis or aneurysm due to inflammation can also lead to acute MI but on coronary

angiography no such lesion was found.[7,8] Although hypertension is a common cause of LV hypertrophy, there was no evidence of hypertension, renal vessel abnormalities or previous documented history of hypertension in this patient. Initially, cardiogenic shock masked the pulse-less condition in the upper limbs, typically an early sign of Takayasu arteritis. In this case, chronic asymptomatic AR and LV hypertrophy compromised cardiac function, leading to MI and necessitating emergency treatment. The patient's asymptomatic status until presentation is unusual and highlights the variability of disease presentation. Though Conventional angiography has been traditionally considered the gold standard for the diagnosis of TA, However, multidetector CT angiography (CTA) is emerging as a reliable tool in non-invasively depicting both luminal and mural lesions in the aorta and its main branches, which may facilitate the detection of vasculitis during the early phase of TA.9 According to the recently proposed angiographic classification of TA, this patient is having TA Type V which is also the most common type.[10]

CONCLUSION

Takayasu's arteritis (TA) is a rare but significant cause of myocardial infarction in young individuals, characterized by chronic inflammation of the aorta leading to serious cardiovascular complications. Early diagnosis can be challenging due to varied and sometimes asymptomatic presentations. This case underscores the importance of considering TA in young patients with unexplained cardiac symptoms, especially when peripheral pulses are not palpable. This patient's prolonged LV hypertrophy likely precipitated her MI. The initial cardiogenic shock masked typical TA symptoms, highlighting the disease's variable presentation. Multidisciplinary management and prompt treatment with anti-inflammatory and heart failure medications are crucial for improving outcomes. Awareness of TA's diverse manifestations can lead to earlier diagnosis and better patient care.

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