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Case Report

Internal Medicine Section

Acute Myocardial Infarction with Left Ventricular Failure as an Initial Presentation of Takayasu's Arteritis

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ABSTRACT

Takayasu's Arteritis (TA) is an uncommon chronic inflammatory and stenotic disease of medium and large-sized arteries characterized by a strong predilection for the aortic arch and its major branches. We report a rare manifestation of TA in a 16-year-old female with no previous history of heart disease who presented with heart failure. She was found to have hypertension and discrepancies of pulses in upper and lower limbs. She developed ST elevated anterior wall myocardial infarction (MI) during hospitalization. Her aortography revealed narrowing of descending thoracic, upper abdominal and infrarenal aorta with significant stenosis of both renal arteries. She was diagnosed as a case of TA on the basis of her clinical profile and arteriography. Her coronary angiography did not reveal any stenosis or occlusion or aneurysm in coronary arteries or at coronary ostia which is in contrast to patients of TA with MI reported in the literature who had focal or diffuse stenosis or aneurysm in the coronaries.

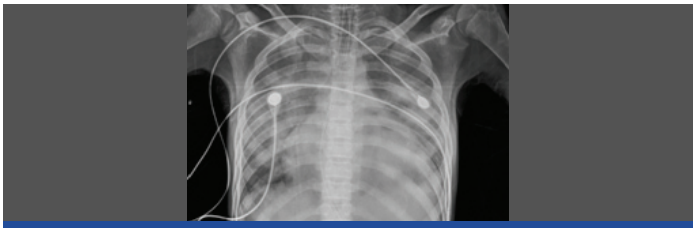
Keywords: Complications, Coronary artery disease, Heart failure, Treatment

CASE REPORT

A 16-year-old Indian female was admitted with complaint of shortness of breath for one month. The breathlessness was progressive which rapidly worsened to breathlessness at rest and orthopnoea (New York Heart Association class 4) four days prior to her admission in hospital. There was no history of fever, cough, weight loss, rashes, arthralgia, raynaud's phenomenon, syncope, blurring of vision or claudication. She had never smoked or consumed alcohol or took psychotropic drugs and had no history of hypertension, diabetes mellitus, heart disease or stroke.

On general examination, she was afebrile, pale and anicteric. She had a pulse of 124 beats per minute, blood pressure (BP) of 170/120 mm Hg in right upper limb and 150/116 mm Hg in left upper limb and respiratory rate of 35 breaths per minute. Her BP was not recordable in lower limbs. Pulsations were absent in bilateral femoral, popliteal and dorsal pedis arteries. Bruit was audible over abdominal aorta. Her jugular venous pressure was not raised. No lymphadenopathy was present. Her fundus examination did not reveal any evidence of hypertensive retinopathy or papilloedema. Respiratory system examination revealed fine bibasilar crepitations extending upto midscapular area. Her cardiovascular examination revealed apex beat in the 6th left intercostal space lateral to mid-clavicular line with no murmur or thrill. Physical examination of other systems is unremarkable. Her arterial oxygen saturation was 82% on supplemental oxygen. In view of her hypoxemic respiratory failure, she was intubated and put on mechanical ventilator. She was started on intravenous diuretic (furosemide) and oral antihypertensives (amlodipine, metoprolol).

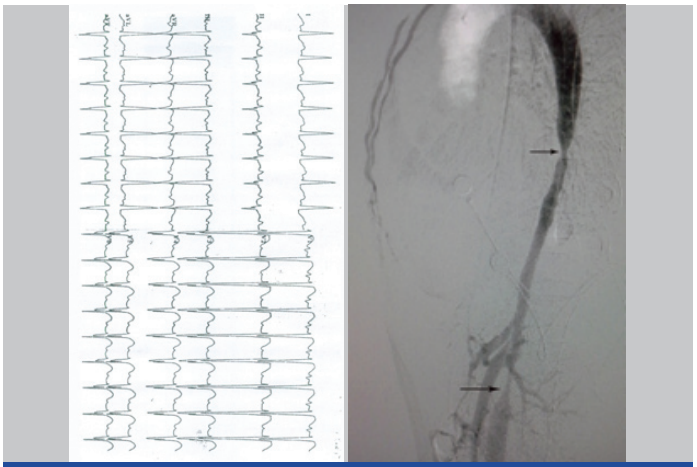
Chest radiograph (posterior-anterior view) revealed enlarged cardiac shadow with diffuse alveolar shadows consistent with pulmonary oedema [Table/Fig-1]. Electrocardiogram (ECG) on day 1 of hospitalization showed sinus tachycardia with left ventricular hypertrophy. Echocardiography showed dilated left ventricle with mild concentric hypertrophy with severe left ventricular systolic dysfunction and ejection fraction (EF) of 15%.



[Table/Fig-1]: Chest radiograph showing cardiomegaly with diffuse pulmonary alveolar shadows.

The erythrocyte sedimentation rate was raised at 44 mm in the first hour and C-reactive protein was 32 mg/L (normal value is less than 10 mg/L). Her baseline biochemical investigations and thyroid profile were within normal limits except for urine albumin which is positive (2+) by dipstick. Her serological tests were negative for antinuclear antibodies, extractable nuclear antigens, anti-double stranded DNA antibodies, rheumatoid factor, antineutrophil cytoplasmic or antiphospholipid antibodies, VDRL, hepatitis B virus, hepatitis C virus and Human Immunodeficiency Virus (HIV). Her ultrasonography of abdomen revealed contracted small left kidney with thin echogenic cortex and loss of corticomedullary differentiation.

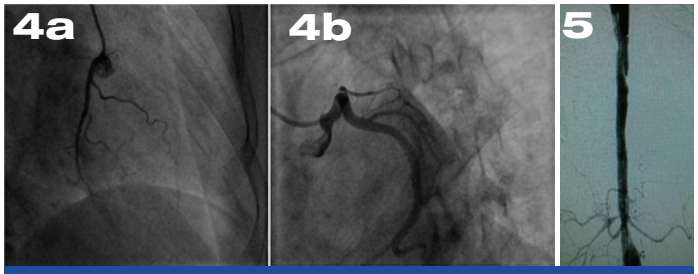
On day 2 of hospitalization, she was successfully weaned off from the mechanical ventilator. On day 3, she developed mild pain in chest associated with breathlessness and restlessness. Her repeat ECG revealed ST segment elevations in V1 to V6 chest leads suggestive of acute anterior wall ST elevation myocardial infarction (STEMI) [Table/Fig-2]. Her creatinine kinase (muscle and brain) activity and troponin T were 37 IU/L (normal value is less than 24 IU/L) and 0.895 ng/mL (normal value is less than 0.02 ng/mL) respectively. She was referred to other centre for angiography and



[Table/Fig-2]: Electrocardiogram showing ST-segment elevation in V1 to V6 chest leads. **[Table/Fig-3]:** Angiography of aorta showing long segment diffuse narrowing of descending thoracic and upper abdominal aorta. Narrowing of infrarenal aorta is also seen (arrows).

intervention. Her angiography of aorta and its branches revealed long segment diffuse irregular narrowing of descending thoracic and upper abdominal aorta with maximal stenosis of upto 80% at mid-thoracic aortic level and an another area of 70% luminal narrowing in infrarenal aorta [Table/Fig-3]. Gradient across the lesion was 70 mm Hg. In addition there was significant stenosis of more than 80% in right renal artery and near complete occlusion of left renal artery. The angiographic features are suggestive of aortoarteritis or Takayasu's arteritis (TA) type III. Her coronary angiography did not reveal any stenosis or occlusion or aneurysm in main coronary arteries or at coronary ostia [Table/Fig-4a&b].

Mid-aorta angioplasty was done with 8×60 mm and 10×60 mm balloon with stenting [Table/Fig-5]. Infrarenal aorta angioplasty was performed using 8×40 mm and 9×40 mm balloon. Post angioplasty there was significant improvement in pressure gradient across the stenotic lesion from 70 mm Hg to 10 mm Hg. Right renal artery angioplasty was performed using 3.5×16 mm balloon. There was significant improvement in clinical symptoms. Patient was comfortable at bed as compared to breathlessness at rest and orthopnea before. The patient was discharged on oral steroids (prednisolone, 1 mg/Kg/day) and antiplatelet therapy. During the 6-month follow up period after discharge, the patient had no chest pain or any other cardiovascular complications and was comfortable in her daily routine activities. Her blood pressure was well controlled without any anti-hypertensive medications.



[Table/Fig-4a,b]: Coronary angiograms showing normal (a) right and (b) left coronary arteries. **[Table/Fig-5]:** Post-angioplasty improvement in the caliber of narrowed aorta.

DISCUSSION

Hypertension, heart failure, cerebrovascular events, retinopathy, glomerulonephritis, renal failure, cardiomyopathy, aortic regurgitation, pulmonary artery hypertension or aneurysm rupture are the usual complications of TA reported in the literature [1,2]. A patient is said to have TA if at least three of the six criteria as proposed by the American College of Rheumatology are present [3]. In our case, four of the six criteria are fulfilled i.e. age at disease onset ≤40 years, difference of at least 10 mmHg in systolic blood pressure between the arms, bruit over the abdominal aorta and arteriographic narrowing of the aorta.

The three distinct morphological types of coronary artery lesions reported in the literature are: type 1, stenosis or occlusion of the coronary ostia and the proximal segments of the coronary arteries; type 2, diffuse or focal coronary arteritis, which may extend diffusely to all epicardial branches or may involve focal segments, so-called skip lesions; and type 3, coronary aneurysm formation [4-6]. Stenosis or occlusion of proximal coronary segments involving the coronary ostia are the most common lesions reported in the literature [4]. In the previous reported cases of MI in TA, obstructive or aneurysmal or mixed lesions in coronary arteries had been described [6-8]. In our case, coronary angiography did not reveal any stenosis or occlusion in main coronary arteries. Transient coronary thrombosis or vascular spasm or inflammatory involvement of myocardial microcirculation might be the cause of MI in our patient. In our patient, myocarditis could be the other possible differential diagnosis for acute chest pain with ST segment elevation in ECG and raised cardiac enzymes. However, cardiac magnetic resonance imaging with gadolinium enhancement did not reveal any features suggestive of myocarditis (negative Lake Louise criteria). TA can present as Dilated Cardiomyopathy (DCM). Non-specific inflammation of myocardium with lymphocyte/mononuclear cell infiltration was found in histopathological studies on autopsy cases of TA with DCM [9].

A combination of glucocorticoid therapy for acute signs and symptoms and an aggressive surgical or angioplastic approach to stenosed vessels markedly improve outcome and decrease morbidity of TA by lessening the risk of heart failure, cerebrovascular events, renal failure, myocardial infarction, or aneurysm rupture [1,10].

CONCLUSION

A patient of TA having no typical systemic or constitutional symptoms may present for the first time with ST elevation MI and may not have classical documented coronary lesions on coronary angiography as reported in the literature.

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