Case Report

Takayasu Arteritis Presenting As Congestive Cardiac Failure

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Abstract

Takayasu arteritis is a chronic systemic inflammatory disease that usually affects the aorta, its primary branches and occasionally the pulmonary and coronary arteries. The clinical manifestations vary considerably and are typically caused by limb or organ ischemia illness and fever. Occasionally, dyspnea and pedal edema can be the sole primary presentation of Takayasu's arteritis.

Keywords: Takayasu's arteritis, congestive heart failure, pulmonary arterial hypertension

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Introduction

Takayasu's arteritis (TA) is a chronic idiopathic vasculitis that variably involves the aorta and or its main branches and the coronary and pulmonary arteries in 50–80%.¹ Inflammation results in stenosis, occlusion, or aneurysm formation.² Aneurysms may rarely progress to vascular rupture and death.³ The first report of TA was by R Yamamoto in 1830, while the first presentation on TA was in 1905 by Mikito Takayasu at the 12th annual meeting of the Japan Ophthalmology Society, describing a patient with a peculiar optic fundus abnormality, characterized by coronal anastomoses.⁴ K Ohnishi and T Kagoshima also presented similar cases and noted their patients lacked a palpable radial pulse⁴. The first autopsy on a patient with TA was carried out in 1940 by K Ohta.⁵

Case Presentation

A 21 year old male chronic smoker presented to our Emergency department with a history of fever on and off since last six months. He also had breathlessness and chest pain and bilateral pedal edema. Patient was apparently well six months back when he developed fever which was low to moderate in grade, undocumented, continuous. Fever was also associated with breathlessness which was insidious in onset, gradually progressive, aggravated on exertion and relieved on rest. Patient gave history of central, non radiating chest pain,. There was history of bilateral swelling of feet. There was no history of orthopnea or PND. No history suggestive of diabetes mellitus, hypertension, jaundice, bronchial asthma, blood transfusion, surgery or trauma was there.

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On examination systolic blood pressure was 100 mmHg and diastolic 70 mm Hg and he had absent radial pulse on the left side. The rest of his examination was normal. On cardiac examination there was tachycardia. Heart sound both S_1 S_2 were normal and systolic murmur was heard in mitral and tricuspid area. On respiratory examination normal vesicular breath sounds were heard with bilateral crepitations. Abdomen was found to be soft and distended with splenomegaly, hepatomegaly. Shifting dullness was found to be positive, On central nervous system examination, higher mental function, motor and sensory function were found to be intact. Hematological findings and biochemistry were within normal limits. Ultrasonological examination revealed B/L renal parenchymal disease with mild hepatomegaly with ascites with prominent IVC and hepatic veins with right side pleural effusion with pericardial effusion. X ray chest revealed cardiomegaly, obliteration of both costophrenic angles (R>L), alveolar shadowing was seen in both lower zones (R>L) and thickening of blique fissure was on right side. Lower limb doppler study was normal. Upper limb doppler study revealed evidence of thrombus in right internal jugular vein and common carotid artery, altered low velocity in left subclavian artery, thickening of right brachiocephalic artery, right common carotid artery and right subclavian artery. 2D Echo suggested enlargement of right atrium and right ventricle with normal left atrium and left ventricle. Estimated LVEF was 55%. There was severe TR, severe PAH. CECT thorax and abdomen were carried out to investigate the principle differential diagnosis of a dissecting aortic aneurysm. There was stenosis in the left subclavian artery was. A provisional diagnosis of Takayasu arteritis was made. Coronary angiography, MRI angiography along with angiography of the great vessels/ aorta was carried out and these confirmed the diagnosis of Takayasu arteritis. The coronary angiogram showed an occluded Pulmonary artery. The arteriogram showed a long segmental left subclavian artery stenosis and a significant stenosis in right internal jugular vein and left common carotid artery and right common carotid artery

Discussion

Non specific symptoms of inflammatory disease such as fever, night sweats, and malaise and weight loss are common early in disease and often precede more specific features.² Arthralgia and myalgia are common. Some patients develop

true arthritis or, less commonly, lupus-like rashes, erythema nodosum or glomerulonephritis. With regards large vessel disease, the most typical features reflect ischaemia, or aneurysm formation in large vessels such as the aorta and its branches. "Aortic arch syndrome" is the term given to disease affecting the upper extremities, heart, neck and head. Patients often complain of arm claudication, and brachial and radial pulses are absent. Hence TA was previously called "pulse less disease".⁴

Figure 1: The arteriogram showing a long segmental left subclavian artery stenosis and a significant stenosis in right internal jugular vein and left common carotid artery and right common carotid artery

Figure 2: Wall thickening of peripheral pulmonary arteries. e/o small pulmonary embolism Involvement of peripheral pulmonary arteries but main trunk is clear s/o pulmonary artery hypertension.

Surgery, angioplasty or stenting² is only required in a minority of patients and should be postponed until the inflammatory component of the disease has been controlled if possible. TA is medically managed ,initially as giant cell arteritis (GCA), with high-dose prednisolone, 40–60 mg per day, as soon as the diagnosis is suspected. Patients with the HLA A24-B52-DR2 haplotype may require larger doses of corticosteroids for longer periods than patients without this haplotype.⁶ The most common clinical manifestation of takayasu's arteritis is affection of the aorta and its main branches⁷ but the involvement of the pulmonary arteries is also known. Recent studies described pulmonary involvement in 14% to 86% of all cases with takayasu's arteritis.^{8,9,10,11}

Clinical features of chest pain, dyspnea, cough ,haemoptysis, congestive heart failure suggest involvement of pulmonary arteries.¹²

Conclusion

TA can present in wide variety of ways, many with a typical history of other conditions. The use of steroids is paramount to the acute medical treatment but not curative. Surgery, angioplasty or stenting is only required in a minority of patients.

For a definitive diagnosis the use of modern day imaging such as CT, MRI and angiography is vital. From this case report we learn the importance of keeping an open mind with differential diagnoses, despite a "typical" presenting history, and the need for confirmatory investigations and therefore appropriate treatment.

Ethical Approval:

This case report was published after getting approval of the Ethics Committee of MMIMSR, Mullana (Ambala), India

Conflict of Interest

No Conflict of interest has been disclosed by the authors.

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References

1. Gravis MB; Giant cell arteritis and takayasu arteritis ;morphologic ,pathogenic and etiologic factors.
2. Amir O, Kar B, Civitello AB, Palanichamy N, et al.: Unprotrected left main stent placement in a patient with Takayasu's arteritis. ^[1]Tex Heart Inst J 2006,
3. Numano F, kobayashi Y; Takayasu arteritis; clinical characteristics and the role of genetic factors in its pathogenesis.
4. Numano F: Introductory remarks for this special issue of Takayasu arteritis. ^[1]Heart Vessels 1992,
5. Ohta K: Einseltener Fall on bleiderseitigemCarotis-Subclaviaverschluss, EinBeitragzurPathologie der Anastomosis peripapillaris des AugesmitfehlendemRadialpuls. ^[1]Trans SocPathol Jap 1940,
6. Moriwaki R, Numano F: Takayasu arteritis, follow up studies for 20 years
7. Kerr GS, Hallahan CW, Giardano J, Leavitt RY,Fuasi AS, Rottern M, Hoffman GS. TakayasuarteritisAnn intern Med.1994;120;919-929. [pubmed].
8. Sharma S,kamalakarT, rajani M, krishantalwar, shrivastava S. The incidence and pattern of pulmonary artery involvement in takayasu's arteritis. Clin radio.1990;42;177-181
9. Vanoli M, Castenelli, bachianiG, caliG, mietner B, origgiL, Scorza R, non invasive assessment of pulmonary artery involvement in takayasu's arteritis. Clinexprhematol. 1999;42,215-218[pub med]
10. Lupi E, Sanchez G, Horowitz S, Gutierrez E. Pulmonary artery involvement in Takayasu's arteritis. Chest 1975;67;69-74.
11. Yamada I, shibuya H, matsubara O, umeharaI,Makino T, numano F, Suzuki S. pulmonary artery disease in takayasu's arteritis angiographic findings. Am j roentgenol.1992;159,263-269.[pubmed]
12. Karad B, Kilic H, duman D, Ongen Z, Vural VA, YaziciH. Takayasu disease with prominent pulmonary artery involvement; confusion with pulmonary diseases leading to delayed diagnosis. Mod Rheumatol, 2008;18;507-510