Case Report

A rare case of Takayasu Arteritis in young woman with heart failure - A case report

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Abstract

Takayasu arteritis (TA) is a rare, systemic, granulomatous primary vasculitis of medium and large arteries. The name comes from Dr. Mikito Takayasu, who reported the problem in 1905 for the first time. It is also called as Pulseless Disease or Aortic Arch Syndrome and usually seen before 40 years with female and male ratio being 10:1. Takayasu arteritis is a major cause of high blood pressure levels in teenagers and young adults. Around 75 percent of the people having Takayasu get diagnosed usually at an average age of 29 years though they begin to show the symptoms at their teenage years because the early symptoms of Takayasu are nonspecific and common. Heart failure as the first presentation of the TA is rare but has been reported. Angiographic studies help in the diagnosis of Takayasu and patients usually respond to steroid therapy. We report a 16 years old female presented with history of upper limb claudication, dyspnea, orthopnea, non palpable pulse in bilateral upper limbs with non recordable BP, lower limb with high blood pressure recordings and bilateral carotid Bruit present. Imaging studies revealed circumferential wall thickening of arch of aorta, bilateral carotids, left sub clavian, left axillary. 2D echo revealed global hypokinesia with severe left ventricular dysfunction. Takayasu arteritis with heart failure diagnosis was made and administration of steroids, diuretics and ACE inhibitor improved the condition.

Key words

Takayasu Arteritis, Vasculitis, Left Ventricular Dysfunction, Heart Failure.

Introduction

Takayasu arteritis (TA) is characterized by chronic granulomatous inflammation of the blood vessel walls and is included in large vessel vasculitis with an unknown aetiopathogenesis. The arteries getting affected by Takayasu involves the aorta and its branches which supply the brain, arms and kidneys. Though aorta which pumps oxygen-rich blood from the heart to the rest of the body is the main artery involved, it also involves pulmonary and coronary arteries less frequently causing damage to the main organs of the body. There is reduced or absent pulses in the arms and legs. Hence, the name Pulseless Disease, Takayasu Aortitis and Aortic Arch Syndrome were given. TA is also called 'Occlusive Thromboarthropathy', 'Martorell syndrome' [1].

Epidemology

TA is most commonly seen in the Asians and immigrant Asians in Western countries. The prevalence of TA in Scandinavian countries is increased, probably because of increase in the immigration from Asia and Africa [2, 3, 4].

The incidence and prevalence of TA in the northwestern part of Turkey is similar to that of Japan. The disease has a predilection for females with geographic variation ranging from 8:1 in Japan to 1.2:1 in Israel. In a recent Japanese study, females less than 40 years of age constituted a major proportion (83.8%) of the study cohort. The disease is five times more common in females than men, and an interesting feature was that there was a larger proportion of elderly compared to a previous study [5]. TA presents commonly in the second and third decades of life. In women, the disease peaks at 20 years of age [6]. There are geographic variations of the involvement of the aorta and its branch vessels [7].

Genetic predisposition

The pathogenesis of TA has been associated with the human leukocyte antigen (HLA) class I (HLA-A, HLA-B, and HLA-C), HLA class II (HLA-DR, HLA-DQ, and HLA-DP) and non-HLA genes [8, 9]. There is definite HLA B52 allele association in TA beyond ethnicity [10].

The correlation with TNF-α 308/G polymorphism has also been reported in TA. FCGR2A/FCGR3A, IL-12B, IL-6, RPS9/LILRB3 are the non-HLA loci related to TA. A report from Italy demonstrated an association with HLA DRB1 *0405 for early onset vasculitis [9]. DRB1, DR2, DQ 1 are among other alleles related to TA.

Heart failure occurs when the heart muscle doesn't pump blood as well as it should. When this happens, blood flooding up in the lungs, causing dyspnoea. Certain heart conditions gradually leave the heart so weak or stiff to fill and pump blood properly. These conditions include narrowed arteries in the heart and high blood pressure. Congestive cardiac failure is also associated with hypertension, aortic regurgitation, and dilated cardiomyopathy [11].

Takayasu's arteritis is a major cause of high blood pressure levels in teenagers and young adults. Around 75 percent of the people having Takayasu get diagnosed usually at an average age of 29 years though they begin to show the symptoms at their teenage years because the early symptoms of Takayasuare nonspecific and common. Heart failure has been described as a co-morbidity in 13.2% of TA patients. In a cohort study of 54 Japanese patients, secondary hypertension was prevalent in 37% of patients, and the cause of death in 7 of 8 patients was heart failure or stroke [12]. Heart failure as the first presentation of the TA is rare but has been reported [13, 14, 15, 16]. TA can cause heart failure through inflammatory myocarditis, but more commonly, it is caused by renovascular secondary hypertension [17].

Clinical features

Takayasu manifests as non-specific systemic inflammatory symptoms like fever, night sweats, weight loss, arthralgia, mild anemia, fatigue, chest pain and headache in the early stage. TA

can also induce ischemic symptoms related to vascular stenotic lesions. Sometimes, it progresses into fatal presentations which include myocardial infarction, heart failure, cerebral thrombosis. TA manifests as two phases

1) A pre pulseless phase characterized by non specific inflammatory features followed by

2) A chronic with development of vascular insufficiency, in few cases with intermittent flares [18].

Diagnostic criteria were as per **Table – 1** and **Table – 2**.

Table - 1: 1990 ACR criteria for the classification of Takayasu arteritis [19].

CRITERION	DEFINITION
Age at disease onset <40 years	Development of symptoms or findings related to Takayasu arteritis at age <40 years
Claudication of extremities	Development and worsening of fatigue and discomfort in muscles of 1 or more extremity while in use, especially the upper extremities
Decreased brachial artery pulse	Decreased pulsation of 1 or both brachial arteries
Blood pressure difference >10 mm Hg	Difference of >10 mm Hg in systolic blood pressure between arms
Bruit over subclavian arteries or aorta	Bruit audible on auscultation over 1 or both subclavian arteries or abdominal aorta
Arteriogram abnormality	Arteriographic narrowing or occlusion of the entire Aorta, its primary branches, or large arteries in the proximal upper or lower extremities, not caused by arteriosclerosis, fibromuscular dysplasia, or similar causes; changes usually focal or segmental

A diagnosis of Takayasu arteritis requires that at least 3 of the 6 criteria are met.

Table - 2: Classification of TA from the Takayasu Conference 1994 [20].

Type	Vessel involvement
Type I	Branches from the aortic arch
Type IIa	Ascending aorta, aortic arch and its branches
Type IIb	Ascending aorta, aortic arch and its branches, thoracic descending aorta
Type III	Thoracic descending aorta, abdominal aorta, and/or renal arteries
Type IV	Abdominal aorta and/or renal arteries
Type V	Combined features of types IIb and IV

Endomyocardial biopsy is a golden standard for detecting myocarditis, but it is invasive with potential procedural complications. Detecting active inflammation is sometimes challenging and can be assessed through a combination of symptoms, and laboratory and imaging findings [21].

Cardiac magnetic resonance imaging (CMR) is a comprehensive imaging technique that offers evaluating exact cardiac size and function, valvular function, identifies myocardial inflammation, and fibrosis and depicts the major arteries' walls and main branches [22].

Positron emission tomography (PET) is another useful method to assess the presence of inflammation with some limitations with regarding the availability and the cost [23, 24].

Case report

A 16 year old female patient presented with history of upper limb claudication of 1 month duration, dyspnea associated with orthopnea of 2 days duration.

History of present illness: Patient was apparently asymptomatic a month earlier, then she noticed sudden onset of weakness in both the upper limbs. She experienced sudden onset of shortness of breath after she woke up of 2 days duration. No History of slurring of speech, No history of headache or vomiting. No history of loss of consciousness or seizures/ head injury, No history of visual disturbances, No history of fever/ arthralgia or rash.

Past history: No history of similar complaints in the past, No history of HTN/ DM/ Thyroid/CVA/ Epilepsy in the past.
Personal history: Mixed diet, Normal appetite, Adequate sleep, Bowel and bladder movements regular, No addictions, No known drug allergies.
Family History: No similar complaints in the family.

General Examination: Patient was conscious, coherent, co-operative and well oriented to time, place and person. pallor+, platynychia+, no signs of icterus/ cyanosis/ jaundice/ clubbing/ pedal edema or lymphadenopathy.

On examination
Vital data: Temp: Afebrile, bilateral upper limb pulse - not palpable (common carotid, brachial, radial) with non-recordable BP, lower limb pulse - 74/min regular, with BP – 150/90 mmHg in lower limbs. There was carotid bruit on both sides.

Systemic examination
Cardiovascular system examination showed jugular venous pressure (JVP) was not elevated. Cardiomegaly was present, apex in 6^{th} left inter costal space (ICS), in anterior axillary line, forceful, S1, S2 heard normal, no murmurs or rub,
Lungs - bilateral basal crepitations
CNS - Intellectual functions: - Patient was conscious/ coherent/ co-operative, oriented to time, place and person. Memory – Intact, Speech – normal, Cranial nerves - all cranial nerves normal
Fundus – Normal central retinal artery and vein, no micro aneurysms, No signs of meningeal irritation, Pupils – NSRL, Motor system – normal, Sensory system – all modalities of sensations were normal, Peripheral nerves were not thickened, Skull and spine: Normal.

Investigations
Complete blood picture (CBP) - Hemoglobin was 8.5 gm%, Red Blood Cell (RBC) count was 4.71 million/cumm, Packed Cell Volume (PCV) was 35.4, Total Platelet Count (TPC) was 2.9 Lakh/cumm, Total leukocyte count (TLC) was 7500/ cumm, N60, L35, M3, E3, B0, ESR was 95 mm, Blood Urea was 34 mg%, Serum Creatinine was 0.8 mg, Complete Urine Examination (CUE) – NAD, Random Blood Sugar (RBS) was 103 mg/dl, Serum Electrolytes: Na – 143 mmol/L, K - 3.4 mEq/L, CL – 106 mEq/L, Viral Markers showed HIV – negative, HbsAg - negative, HCV - negative.The polymerase chain reaction test of the nasopharyngeal swab for SARS-- CoV- 2was negative USG Thyroid was normal, T3 - 120 ng/dl (60-180), T4 - 12.6 μg/ dl (7.3-15), TSH – 3.00 μIU/L (0.55-4.78). X-ray Chest PA view showed cardiomegaly, left Ventricular type (**Figure - 1**), ECG - LVH, 2D ECHO- global hypokinesia of left ventricle, with LVEF-28% with severe LV dysfunction, no PE/Clot.

Upper limb arterial doppler: (**Figure - 2**) circumferential wall thickening of visualised arch of aorta, bilateral carotids, left sub clavian, left axillary, proximal left brachial artery with slow mono phasic flow in bilateral upper limb arteries.
CT Aortoangiogram: (**Figure - 3**) circumferential wall thickening of descending aorta up to D8 vertebral level, dilated ascending aorta with arch of aorta of normal diameter. Long segment luminal narrowing with circumferential wall thickening in bilateral common carotid, bilateral subclavian, left axillary, brachial radial arteries is

seen. Celiac, mesenteric, renal arteries are normal. Suggestive of Aorto Arteritis.

Figure – 1: Chest X-ray showing cardiomegaly.

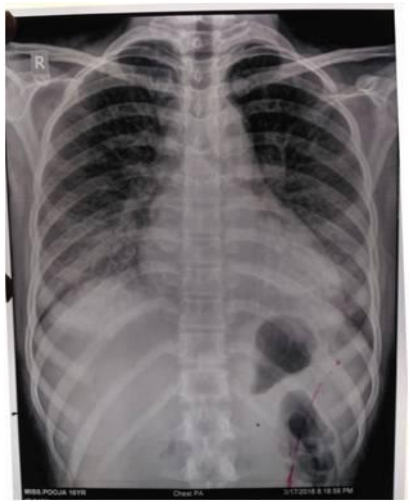


Figure – 2: Upper limb arterial doppler showing proximal left brachial artery with slow mono phasic.

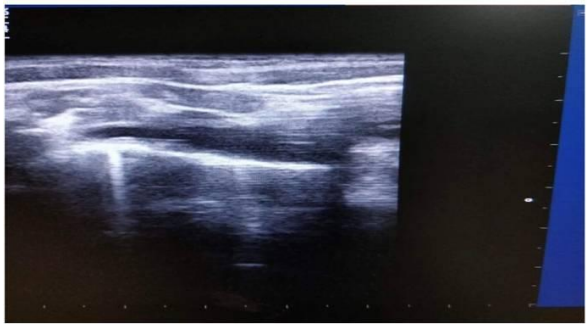
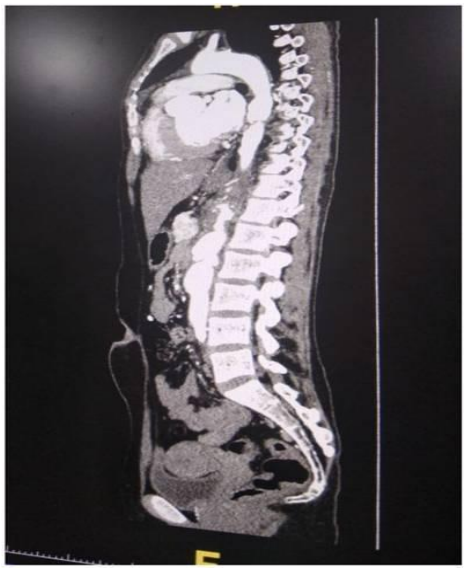


Figure – 3: CT aortoangiogram showing circumferential wall thickening of descending aorta up to D8 level.



Immunology Report – ANA - negative, Anti ds DNA - negative, Anti PR3 - negative, Anti MPO - negative, Cardiolipin Ab IgA - 1.5 u/ml (neg) (N - < 12 u/ml), CardiolipinAb, IgG - 2.0 u/ml (neg) (N - < 20 GPL U/ml), CardiolipinAb – IgM - 10 /ml (negative) (N < 13MPL U/ml), Beta 2 Glycoprotein IgGAb - 0.46 u/ml (neg) (N - <12 u/ml), Beta 2 glycoprotein IgM ab-0.2 u/ml8 (neg) (less than 12 u/ml), Lupus anticoagulant - 29.50 secs (n - 39.20 secs), Factor 5 Leiden mutation – RT PCR not detected, Protein C - 82% (N -70-140), Protein S - 88.3% (N - 63.5-149).

Diagnosis

Based on the significant blood pressure differences between the arms (>10 mm Hg), the elevated inflammatory markers, and arteriographic narrowing and occlusion of multiple branches of aorta, a diagnosis of Takayasu's arteritis was made. She was promptly started on high dose corticosteroids with prednisone at 60 mg/day, along with diuretics and ACE inhibitors. She improved clinically in response to the corticosteroids and later follow up at 2 months, she showed more improvement in her symptoms and her ejection fraction was improved to 40%. The final diagnosis was Takayasu Arteritis, hypertension with Left Ventricular Dysfunction and left ventricular Failure.

Discussion

TA – Myocardial Pathology

In 1988 and 1991, Talwar, et al. performed serial endomyocardial biopsies in TA patients separately .Among these 8 out of 18 and 24 out of 54 patients were found to have myocarditis. For the treatment and outcomes, usage of immunosuppressive therapy early seemed to be responsive. Talwar, et al. also chose combined therapy of prednisolone and cyclophosphamide over 12 weeks, and improvements were evident in myocardial activity, clinical state and hemodynamic states of the subjects [17].

Takeda performed an LV endomyocardial biopsy on a 15-year-old male with TA, and the specimen showing infiltration of T-cell receptor + T lymphocytes plus severe myocyte necrosis and CD16+ natural killer cells. The pathophysiology was proved to be direct immune cytotoxicity towards the myocardium. However, Breinholt, et al. [48] reported a 15 year old girl with TA who developed left ventricular dysfunction and vasculitis affecting small vessels. His endomyocardial biopsy revealed increase in immune complex deposition in the walls of small myocardial vessels.

Takeda used steroid therapy for 2 months, and the patient's symptoms were markedly alleviated, and his cardiac function and morphology greatly improved. In our patient, we initiated the treatment regimen of steroids, diuretics and ACE inhibitors, her symptoms partially resolved after 2 weeks. The result also supported our suspicion of myocarditis as the underlying disease even without performing biopsy [25].

TA – Pregnancy

The issue of pregnancy is important because Takayasu arteritis predominantly affects women of reproductive age. Kerr et al reported five pregnancies in their series of 60 patients, all of whom had normal deliveries of a normal live infant. Among these, only one patient had disease exacerbation during pregnancy [26].

A study from Hong Kong in 1983 reported on 13 women who had experienced a total of 30 pregnancies. There were no major obstetric problems and no maternal deaths directly related to pregnancy apart from hypertension. On the basis of maternal vessel involvement (abdominal aorta and renal), severity of maternal hypertension, superimposed pre eclampsia, and timing of adequate blood pressure control the fetal outcome can be assessed [27].

TA – Imaging studies

In available imaging tests, the complementary role of imaging in clinical assessment of disease activity is lacking specificity. The earliest detectable abnormality in Takayasu Arteritis is usually the thickening of the vessel wall due to inflammation. Imaging studies in clinical assessment of disease activity reveal Vessel wall thickening with enhancement which had sensitivity of 88% and specificity of 75%.Historically, Conventional digital subtraction angiography helps to identify stenosis, occlusions and aneurysms which help in diagnosis of Takayasu Arteritis [28].

Conventional digital subtraction angiography has the least sensitivity for visualizing wall thickness. A systematic review showed the presence of a low attenuation ring in computed tomography (CT) angiogram as 100% specificity for disease activity [29].

TA – Delay in diagnosis

The diagnosis of Takayasu's arteritis (TA) is often delayed or even missed because this disease has a non-specific clinical presentation and no specific laboratory tests. A detailed and thorough physical examination is often very helpful in this diagnosis as absent or diminished pulses and BP discrepancy are diagnostic markers of the disease [19]. This case highlights the advanced age and rare presentation of the disease as acute congestive heart failure in a Caucasian woman. The disease is usually seen in the second and third decade of life [30]. In a clinical study of 107 patients, the majority of patients (77%) presented between 11 to 30 years of age [31]. The most common symptoms in this disease in two cohorts of TA patients were diminished or absent pulses leading to limb claudication and BP discrepancies, which was seen in 84%–88% of cases [32, 33]. Some patients with TA develop congestive heart failure, the main causes being increased after load due to renovascular hypertension and aortic regurgitation. Myocardial ischemia induced by myocarditis, accelerated atherosclerosis, or severe pulmonary hypertension can also contribute [17]. The elevated troponin in this patient may be due to an associated myocarditis.

TA – Aortic regurgitation and heart failure

Aortic regurgitation and heart failure have been reported in about 25% of patients [12, 34]. Aortic regurgitation associated with ascending aorta aneurysm is relatively rare in TA [35, 36].

Rivera FB, et al., reported a case of Takayasu's arteritis with giant left ventricular pseudoaneurysm presenting as heart failure [45].

TA -Aneurysms

Aneurysms are more commonly described in Asian populations than in North American populations [18]. Takayasu's arteritis, if left untreated, can have substantial morbidity with life threatening vascular complications. Imaging techniques, such as computerized tomography, magnetic resonance angiography, fludeoxyglucose positron emission tomography-computerized tomography, and contrast-enhanced ultrasonography, can help with this diagnosis [2].

TA – Abdominal Aortic Thrombosis

Heart failure due to abdominal aortic thrombosis is a rare clinical manifestation of TA; so far, only one case of this kind has been reported [42]. Huan Wang present a case of delayed diagnosis in an adult man of TA with recurrent episodes of heart failure and uncontrolled hypertension due to abdominal aortic thrombosis with claudication of two extremities [43].

Management

Vascular interventions with immunosuppressive therapy with corticosteroids and biological agents, especially in refractory cases, are the therapeutic mainstays of this disease [2, 37].

Surgical Indications

Indications include

1) claudication of extremities affecting daily activities,

2) hypertension with critical renal artery stenosis,

3) cerebrovascular ischemia or

4) criticalstenoses of three or more cerebral vessels,

5) cardiac ischemia due to coronary artery involvement, and

6) moderate aortic regurgitation [26].

Review of literature has shown that fewer than 20% of the patients undergo surgery at some point of their disease course [38, 39].

TA – Surgical outcomes

Surgical outcomes are better when done during periods of inactivity. Procedures during acute inflammatory stages have a seven times greater chance of failure [40]. The mean survival rate in a longitudinal study of 106 patients for 20 years was 73.5%, the main cause of death being congestive heart failure [41]. Takayasu's arteritis is a progressive disease of systemic vasculopathy and if untreated is potentially fatal. It requires an interdisciplinary approach to management and long term follow up for satisfactory patient outcomes.

TA – AortoArteritis, Vasculitis, and Cardiac Involvement

As a large-vessel vasculitis with unknown etiology, TA predominantly affects young Asian women but can be seen in different parts of the world [14]. The entire aorta and major aortic branches can be involved; however, the distribution of involvement varies in each part of the world [23]. TA has a highly variable presentation, including constitutional symptoms, valvular insufficiency, LV dysfunction and thrombus formation in ventricles and pulmonary artery, are reported among the first presentations [13, 23, 44]. Coronary artery disease, pulmonary hypertension, or hypertrophied left ventricle are the other possible presentations [44]. Atypical forms of initial presentation may lead to a delay in diagnosis and alter the outcome. Aortic regurgitation and renovascular-induced hypertension have been introduced as the known causes of symptomatic myocardial damage [13]. The prevalence of aortic regurgitation in TA is reported to be as high as 25%, with a significant prognostic impact [14]. In pathological studies on post-surgical specimens, thickening of intima, media, and adventitia of aortic wall has been documented. AR is usually due to annular dilatation [14].

Out of 195 cases of Takayasu's arteritis who presented in our institute between January 1988 and December 1997, 12 (5.58%) had dilated cardiomyopathy [46]. Cardiac involvement in 75 cases (mean age 21.1 +/- 6 years) with non-specific aorto-arteritis was studied. Detailed clinical examination, echocardiography and cardiac catheterization, including angiography, were done in all the cases, as was coronary angiography. Features of cardiac failure like sinus tachycardia, cardiomegaly, left ventricular third heart sound gallop and pulmonary congestion were detected in 27 cases with reduction of left ventricular ejection fraction (25-48%). Systemic hypertension was seen in 60 cases [47].

Cardiac manifestations include hypertension and involvement of the cardiac valves, myocardium and coronary arteries. Surgery on these patients is always a challenge given the tissue quality and the disease activity. They are prone to long-term complications such as restenosis and graft occlusion, hence requiring lifelong surveillance. The prevalence of coronary artery disease (CAD) in TA ranges from 9 to 11%. Coronary artery bypass grafting is preferred to percutaneous coronary intervention, as the latter has a high rate of restenosis and major adverse cardiovascular events. As left subclavian artery is commonly involved, saphenous vein graft is advised as a conduit rather than internal mammary artery. Other surgical procedures described for CAD are surgical angioplasty of the left main coronary artery and transaortic coronary ostial endarterectomy. Aortic regurgitation in TA has an incidence of approximately 20%. These patients tend to have prosthetic valve detachment, paravalvular leak or pseudoaneurysm at the anastomotic site. Further repair of these valves have a high rate of failure. Considering these facts, it is advisable to do an aortic root replacement for TA patients than to consider an aortic valve replacement or David's procedure [7].

Conclusion

Takayasu arteritis (TA) is characterized by chronic granulomatous inflammation of the blood vessel walls and is included in large vessel vasculitis with an unknown aetiopathogenesis. TA is most commonly seen in the Asians. The pathogenesis of TA has been associated with the human leukocyte antigen (HLA) and non-HLA genes. TA may present as congestive cardiac failure which is also associated with hypertension, aortic regurgitation, and dilated cardiomyopathy.

Here, we report a case of Takayasu arteritis in a Young woman who had symptoms symptoms of heart failure as initial presentation. Multi-modality imaging approach plays a crucial role in making the diagnosis by providing a full assessment of this multi faceted disease and in determining the treatment choices and follow-up care. Vascular interventions with immunosuppressive therapy with corticosteroids and biological agents, especially in refractory cases, are the therapeutic mainstays of this disease. It requires inter disciplinary approach to management and long term follow up for satisfactory patient outcomes.

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