Case Report

Takayasu Arteritis: A Case Report

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ABSTRACT
Takayasu arteritis is also known as pulseless disease and is an autoimmune disease with unknown etiology which is a rare form of vasculitis involving inflammation in the wall of the aorta and its main branches. Epidemiologic investigation recommended that Takayasu arteritis is large progressively perceived in Europe with detailed occurrence varying from 0.4-1.5 per million. The prevalence of Takayasu arteritis in Japan is 40 per million and at least 0.9 per million in the US. These varieties between the examinations might be gotten from topographical and hereditary contrast between the population.1

Pathogenesis of Takayasu arteritis remains unclear but a hypothesis state that gamma-delta lymphocytes, NK cells, and HSP65 protein play a critical role in vascular cell injury of Takayasu arteritis.2 Neurotic findings depend on aortic tissue test showed that gamma-delta T lymphocytes and NK cells are located in apoptosis of endothelial cells by the creation of perforin and killer cells lectin-like receptor subfamily K(NKG2D). The gamma-delta T lymphocyte and NK cells communicate with the NKG2D receptor and recognize MICA (Major histocompatibility class-1 chain receptor) on vascular smooth muscle and deliver perforin and cause inflammation. Proinflammatory cytokines are additionally delivered from T cells and induce the matrix metalloproteinases (MMPS) which leads to an inflammatory reaction in the vascular wall.3

Takayasu arteritis comprises 2 phases: an initial inflammatory phase which is characterised by fatigue, malaise, fever, night sweats, joint pains, weight loss, fainting, and weight loss. Other symptoms may include arm numbness, claudication in the legs, double vision, stroke, transient ischemic stroke, hemiplegia, and paraplegia. Vascular insufficiency from initial narrowing of the vessel leads to renal artery stenosis causing hypertension, and neurological manifestation due to decreased blood flow to the brain. This stage is followed by fibrosis and may be associated with the remission of symptoms.4

Takayasu arteritis can be assessed based on American college of rheumatology criteria as follows
1. Age at disease onset in year <40 years- Development of symptoms or findings related to Takayasu arteritis at age
2. Claudication of extremities- Defined as the development and worsening of fatigue, and discomfort in muscles of one or more extremities while in use, especially the upper extremity.
3. Decreased branchial artery- Decreased pulsation of one or both branchial arteries.
4. Blood pressure difference > 10mmHg- This difference is mainly seen in systolic blood pressure between the arms
5. Bruit over subclavian arteries or aorta- Bruit are audible on auscultation over one or both subclavian arteries or Abdominal aorta
6. Angiographic abnormality- Angiographic narrowing or occlusion of the entire aorta, its primary branches or large arteritis in the proximal branches or large arteritis in the proximal upper or lower extremities, not due to arteriosclerosis, fibromuscular dysplasia, or similar causes changes usually focal or segmental.5

CASE DESCRIPTION
A 27 years old female patient had a complaint of blurred vision in the left eye for 15 days for which she visited an ophthalmologist. Later she had 2-3 episodes of syncopal attack along with weakness of left lower limb for 3 days, proximal femur bone distal weakness, unable to walk.

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On physical examination, there was a remarkable blood pressure difference between the right arm and left arm. Her left arm BP was 140/90 mmHg whereas her right arm BP was 150/90 mmHg in a supine position with a pulse rate of 80 beats/min.

Cardiovascular examination reveals that:

1. Palpitation - Trachea in the midline, epigastic impulse in left 5th intercostal pressure on the midclavicular line, No palpable murmurs.

2. Auscultation - Pulmonary area - Ejection systolic murmur heard not radiating to left carotid loud, Aortic area Ejection systolic murmur S2 heard.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood picture</td>
<td>Anisocytosis with hypochromic, normocytes and microcytes positive</td>
</tr>
<tr>
<td>WBC</td>
<td>Within the limit</td>
</tr>
<tr>
<td>Platelet</td>
<td>Mild thrombocytopenia</td>
</tr>
<tr>
<td>Differential Count</td>
<td>Neutrophils 87.3%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>11.7%</td>
</tr>
<tr>
<td>Complete urine examination</td>
<td>Numerous calcium oxalate crystals are seen</td>
</tr>
<tr>
<td>PUS Cells</td>
<td>3-4hpf (Normal Range: 1-2/hpf)</td>
</tr>
<tr>
<td>Blood urea</td>
<td>48.97mg/dl</td>
</tr>
<tr>
<td>Serum Creatinine</td>
<td>1.62mg%</td>
</tr>
<tr>
<td>Inflammatory Markers</td>
<td></td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate</td>
<td>60mm/hr (Normal Range: Upto 20mm/hr)</td>
</tr>
<tr>
<td>C- Reactive protein</td>
<td>9.56mg/l (Normal Range: less than 10mg/l)</td>
</tr>
<tr>
<td>Anti-nuclear antibody</td>
<td>Borderline POSITIVE</td>
</tr>
<tr>
<td>ECG</td>
<td>Sinus rhythm, first-degree</td>
</tr>
<tr>
<td></td>
<td>Atrioventricular block (AV block), LVH with repolarisation abnormality</td>
</tr>
</tbody>
</table>

Her USG Abdomen reveals that 1) Retroperitoneum narrowed the calibre of the distal abdominal aorta. USG neck screaming done. Concentric wall thickening of right proximal CCA to consider the possibility of vasculitis. 2) Left kidney not visualised in the left renal fossa and ectopic sites. 2D Echo finding shows Concentric LVH, Grade-2 Diastolic dysfunction (DDF) normal Left ventricular (LV) Systolic function (EF-60%). Fundoscopy reveals that B/L papilledema, Hard exudates positive, grade-4 hypertension with retinopathy changes. MRI scan shows Multiple acute infarcts in the left frontotemporal, parietal lobe, cuneate nucleus, right frontal lobe along the parasagittal plane, genu of the corpus callosum. Narrowing of MI segment of left Middle cerebral artery (MCA) with 40% stenosis of vessel. Narrowing of Aa1, Aa of a left anterior cerebral vessel, hypoplastic left vertebral artery, right fetal Posterior cerebral artery (PCA). CT Angiography reveals that Left renal stenosis, Anterior cerebral artery (ACA) MCA narrowing distal aortitis, subclavian occlusion, B/L carotid occlusion, and right solitary kidney. She was treated with Inj Labetalol 200mg IV, T. Aspirin 75mg OD, T. Atrovas 40mg HS, T.Clopidab 75mg OD, Inj Methylprednisolone 500mg in 100ml normal saline IV OD, T. Nicardia XL 30mg TID, T. Nodosis 500mg TID, T. Artemine 0.5mg OD, Her BP got stabilized by using this medication she was discharged from the hospital with medication i.e., T.Wysolone 30mg OD for 2 weeks, T. Folitrax 7.5 mg OD on Sunday, T. Folinic acid 5mg a/d, T. Aspirin 75mg OD, T. Atrovas 40mg HS, T. Cardivas 3.125mg BD, T.Arkamine 0.1mg TID, T. Nicardia XL 30mg TID, Monitor for syncope.

**DISCUSSION**

Initially, the main aim of therapy, in this case, was to reduce ischemic complications and to treat hypertension. Based on her MRI findings the patient was diagnosed with a Cerebro-vascular accident with left hemiparesis and the treatment was started with T. Aspirin 75mg, T. Clopidab 75mg T. Atrovas 40mg, T. Nicardia XL 30mg, T. Arkamine 0.1mg, Inj Labetalol 20mg IV was prescribed to treat hypertension. As the USG abdomen scan revealed the possibility of vasculitis, so, Inj Methylprednisolone 500mg in 100ml NS were prescribed. It was stopped and replaced with T. Prednisolone 20mg on the same day of the afternoon. As the hypertension was not resolved, Inj Lasix 20mg was prescribed on next day. On the 2nd day, Ejection systolic murmur in the aortic area radiating to carotid, ESM in pulmonary area, abdominal bruits, same treatment was given. On day 3, the ANA profile revealed borderline positive T. IFA was added and planned to start T. Methotrexate or T. Azathioprine. On day-4 same treatment was continued. On day 5 T. Cardivas 3.125mg was added and planned to start T. Methotrexate 7.5mg, on Sundays and T. Folic acid 5mg on alternate days. The same treatment was continued for 14 days. On day 15 CT angiography was performed by this test they confirmed that the patient has Takayasu arteritis, and T. Prednisolone dose increased to 30mg for 2 weeks, T. Folitrax 7.5mg was given on every Sunday, T. Folic acid 5mg on an alternate day was prescribed. The patient was discharged on the 17th day; the same medications were prescribed while discharge.

The treatment of Takayasu arteritis involves corticosteroids as first-line therapy, initiation of therapy with prednisolone 0.7-1mg/kg/day for 1-3 months with gradual tapering once remission is obtained.4 At the initial stage of disease high dose of prednisolone is preferred but a replacement may occur while gradually decreasing the dose, so the physician will prefer immunosuppressants along with corticosteroids.2 Similarly, in this case, immunosuppressants were prescribed along with corticosteroids for better treatment. Hypertension is usually worsened by the use of corticosteroids due to their fluid retaining property and ACE inhibitors, angiotensin receptor blockers are contraindicated in the presence of renal artery stenosis.3 In our case, ACE inhibitors and ARB are not preferred as they are contraindicated.

**CONCLUSION**

As Takayasu arteritis is a progressive vascular disease that may lead to many vascular complications and due to its higher chances of relapse, a long-term follow-up is important. Takayasu arteritis is mainly associated with cardiovascular complications, stroke, renovascular hypertension. Hypertensive urgency and stroke are the most serious complications which should be treated early. Early diagnosis and treatment help us to avoid the rapid progression of the disease.

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**CONFLICT OF INTEREST**

The authors declare that there is no conflict of interest.

**ABBREVIATIONS**

NK Cells: Natural Killer Cells; HSP65: Heat Shock Protein65; MICA: Major Histo Compatibility Class-1 Chain receptor; MMPs: Matrix Metalloproteinases; ESR: Erythrocyte Sedimentation Rate; CRP:
C-Reactive Protein; LVH- DDF: Diastolic dysfunction; LV: Left ventricular; MCA: Middle cerebral artery; PCA: Posterior cerebral artery; ACA: Anterior cerebral artery.

REFERENCES


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