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Takayasu's Arteritis: A Case Report

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Abstract

Takayasu's arteritis, also known as pulseless disorder is a rare chronic granulomatous inflammatory disease that primarily affects the aorta and its branches. It occurs in 1% individuals out of million in a year and is not usually marked by an absent pulse in all individuals. We hereby present a case report of 25year old female patient who was diagnosed with Takayasu's arteritis following observation of absent pulse and un-recordable blood pressure.

Keywords: Takayasu's arteritis, large vessel disease, Pulseless disorder, Aortic arch syndrome, Martorell syndrome, case report

INTRODUCTION

Takayasu's arteritis, also known as Pulseless disorder, Aortic arch syndrome, or Martorell syndrome, is a rare chronic granulomatous inflammatory disease that affects large vessels, primarily the aorta and its branches [1], resulting in vascular occlusion of narrowed or blocked arteritis and aneurysm formation. The most significant aspect is granular inflammation of arteries and infiltration of inflammatory cells, which promotes elastic fiber damage and thrombus formation. Although the actual cause of the disease is unknown, several illnesses, such as viral or bacterial infections, can cause sickness [2]. TA is commonly encountered in females aged 20 years and older, with an annual incidence of 1-3 cases/million [3]. It progresses in two stages, "Pre-Pulseless phase or systemic phase" developing first, followed by the "chronic phase or occlusive phase" with the development of vascular insufficiency [1].

CASE REPORT

BACKGROUND INFORMATION

A 25-year-old female patient was admitted to our university hospital's medicine ward for treatment of her complaints of left-sided weakness for a month, inability to move the upper and lower limbs of the left side, headache, and facial asymmetry. The patient had clear past history.

INVESTIGATIONS

On admission, her physical examination showed murmur positive and non-recordable pulse and BP. On blood testing the hemoglobin was 10.1g/dl suggestive of anemia, neutrophils-49%, lymphocytes-44%, ESR-18mm/hr which was increased to 35mm/hr on 7thday, PCV-32.9%, MCV-64.2fl, MCH- 19.8pg/cell, MCHC - 30.7g/dl. APTT was 31.2 (control: 29.4).

The provisional diagnosis was hemiplegia, but after confirmation tests, including an ECG report revealing sinus tachycardia with short PR interval and left ventricular hypertrophy, Echocardiography (Figure 1) , MRI of brain revealing multiple arterial narrowing segments suggestive of arteritis, CT-Scan of the head and neck revealing chronic ischemic infarct at right

cerebral parenchyma, and Angiography revealing chronic ischemic infarct at right cerebral parenchyma with severe luminal narrowing involving CCA, ICA, and wall thickening of approximately 40-50 % and hypoplastic right vascular arteritis suggestive of TA. CT Angiography of the abdominal aorta revealed circumferential wall thickening in the aortic arch, thoracic and abdominal aorta with nominal luminal diameter and significant luminal narrowing of the right renal artery with approx 2mm diameter, the final diagnosis was confirmed as Type V Takayasu's arteritis (Table 1).

The treatment with proton pump inhibitors (IV Pantoprazole 40mg), anti-emetics (IV Ondansetron 2cc), supplements (IV normal saline with multivitamin 1amp), anticoagulant (T.Aspirin 150mg), antihyperlipidemic (T.Rosuvastatin 10mg), and corticosteroids (T.Methyl Prednisolone 16mg) was started. On continuous evaluation, her pulse and BP were initially unrecordable but they were recorded using a pulse oximeter on the seventh day of her stay. She was advised to get backrest and physiotherapy along with CT aortogram, Takayasu's workup, and to refer rheumatologist for the complaint of joint pain. On the 11th day, the patient was referred to a cardiologist with CT Aortogram, and the cardiologist indicated that steroids be started after consulting the rheumatologist. According to the rheumatologist, the patient had rheumatoid arthritis since one month and was instructed to begin steroid (T. Prednisolone 10 mg initially TDS for a week, then taper to BD for another week, and then continue with OD). The patient was discharged on the 12th day with medications including steroids [T. Prednisolone 10 mg, T.omnacortil 20 mg OD], Vitamin [T. Folic acid (5 mg) +pyridoxine hydrochloride (10 mg) + methylcobalmin (750 mcg) every day except Thursday], immunosuppressant [T. Methotrexate 10 mg every Thursday], calcium and vitamin D supplement [T. Calcium+ vitamin D3 (500 mg BD)], and biphosphonate to prevent osteoporosis from steroids (T.risedronate 35 mg every Sunday). The patient was able to move limbs and the pulse was recordable during discharge. One-month follow-up was planned which revealed that the patient's condition was stable, with no more serious underlying issues.

DISCUSSION

TA is characterised by infiltration of inflammatory cells along with intimal fibrosis, diminished or absent pulse in some cases, worsening of limb claudications, weight loss >2kgs, fever, arthritis, arthralgia, severe abdominal pain, seizures, stroke, hypertension (>140/90mmHg), vascular bruits and vascular narrowing [1, 3, 7]. The diagnosis of TA is often difficult because interval for symptom development is considerable, as intimal thickening and artery occlusion occur in the late stages [4]. Angiography, combined with Doppler ultrasound, is the gold standard for diagnosis [1]. It can be diagnosed with 18F-FDG positron emission tomography (PET) and magnetic resonance imaging (MRI),[8, 9], along with other techniques like Transcutaneous B mode ultrasound, which can aid in disease detection and follow-up. [4].

According to the 2018 EULAR recommendation, the treatment begins with high dose glucocorticoids (40-60 mg/day prednisone) as the first-line drug for induction of remission, and then gradual dose tapering to 15-20 mg/day within 2-3 months, reduced to 5-10 mg/day within 1-2 years. DMARDs such as methotraxate or mycophenolate mofetil are used in addition to GCs. If the disease does not respond to 40-60 mg/day prednisone or a serious recurrence develops, the dose is increased to 40-80 mg/day. Other non-biological disease-modifying agents are given alongside GCs in all TA patients, including tocilizumab (162 mg once a week) or TNF inhibitors, which are considered second-line drugs and then tapering of the initial dose is preferred. If the first medicine is not tolerated, leflunomide or azathioprine is considered, and cyclophosphamide is used as an alternate therapy if treatment fails. [6].

This condition necessitates long-term monitoring and the follow-up is carried out utilising a variety of imaging modalities, including doppler and angiography [1].

CONCLUSION

This case describes a rare Type V TA which is a rare disorder affecting the aorta and its branches and affects only the large vessels. It necessitates close intervention for diagnosis since symptoms are not present in the early stages, and the patient's state in the late stages includes decreased or absent pulse, bruits, claudications, and other conditions that interfere with

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the patient's everyday activities. The condition is often managed with GCs, which produce a positive response in nearly all TA patients. The most significant part of controlling the condition is illness management follow-up since it might impact other organs and cause ischemia.

TABLE 1: New angiographic classification of TA based on the takayasu conference 1994 [4]

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

TABLE 2: According to the 1990 ACR criteria, certain parameters to confirm the diagnosis is involved [1]

Criteria	Definition
Age at disease onset < 40years	Development of certain symptoms or related findings at <40 years
Claudication of extremities	Worsening of fatigue and discomfort in muscles of 1 or more extremity especially in upper extremities
Decreased brachial artery pulse	Decreased pulse in 1 or both artery
BP difference >10mm Hg	Difference >10mm Hg in systolic pressure between arms
Bruits over subclavian artery or aorta	Bruit audible on auscultation over 1 or both subclavian arteries or abdominal aorta
Arteriogram abnormality	Arteriographic narrowing or occlusion of entire aorta, its branches or large arteries in upper or lower extremities, not due to arteriosclerosis or any other known causes
For confirmed diagnosis, atleast 3 of the 6 criteria should be met	

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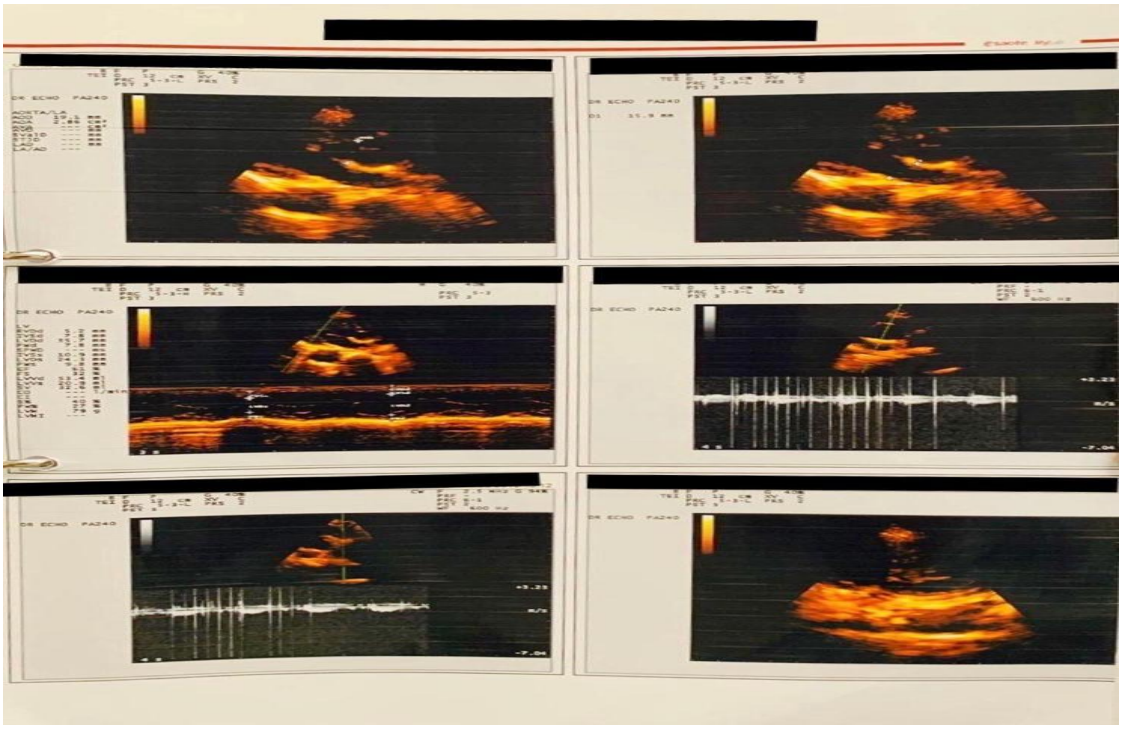
FIGURE 1: ECOCARDIOGRAPHY REPORT



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