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Rare Cardio-Vascular Complications In Takayasu Arteritis : A Case Stady

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I. Introduction

Inflammatory vasculitis involving large vessels such as Takayasu arteritis might mimic congenital structural heart diseases like aortic hypoplasia in rare cases due to post inflammatory strictures in thoracic aorta and aortic arch main branches. This report demonstrates the importance of history taking, physical examination, and using different imaging modalities for the most accurate diagnosis.

Takayasu arteritis (TA) is a granulomatous large and medium arteries vasculitis with unknown etiologywhich mostly involves the aorta and its main branches. Aortic disease is seen in about 85% of the patients with/without aortic branches involvement [1,2] as diffuse or focal thickening of the walls of the vessels resulted in stenosis, occlusion, or thrombosis [3].

We present the case of a young girl with no particular history referred to our center with asymmetrical blood pressure with left subclavian and abdominal souffle which led us to found her suffering after investigation by Takayashu arteritis [1].

II. Case Report

A 25-year-old woman, without cardiovascular risk factors, she presented to our center with headache and angina associated with dyspnea II NHYA, newly diagnosed hypertension. She reported fatigue and intermittent claudication in the previous 2 months when she noticed episodes of rise and asymmetric blood pressure with weakness of her left arm when doing her usual daily work and did not report visual problems, or any other symptoms, her blood pressure was 210/90 mmHg in her right arm and 100/60 mmHg in her left arm, her pulses were absent in her left arm and both her lower limbs, heart rate was 55 bpm, and temperature 37 °C. In her cardiovascular examination, a 3/6 abdominal murmur and a 3/6 left subclavian murmur was heard.

Electrocardiography showed normal sinus rhythm, heart rate: 58 bpm, normal axis with no repolarization troubles or left ventricular hypertrophy (LVH) was seen (Fig.1). In laboratory data, she had mild anemia (hemoglobin: 11.7 mg/dl) and increased inflammatory markers, erythrocyte sedimentation rate (ESR): 90 mm/h, C-reactive protein (CRP): 72 mg/L; troponin us was negative; creatinine : 0.7 mg/dl other laboratory results were normal.

In transthoracic echocardiography, we noticed mild left ventricular (LV) enlargement (Fig.N2) with LV ejection fraction: 40 % (strain: – 15 %), right ventricular(RV) in normal function (Fig.N3)

,mild to moderate aortic regurgitation, proximal ascending aorta with normal measures, small pericardial effusion. Ophthalmic exploration was normal with no sign of vasculitis and cerebral scanner was normal.

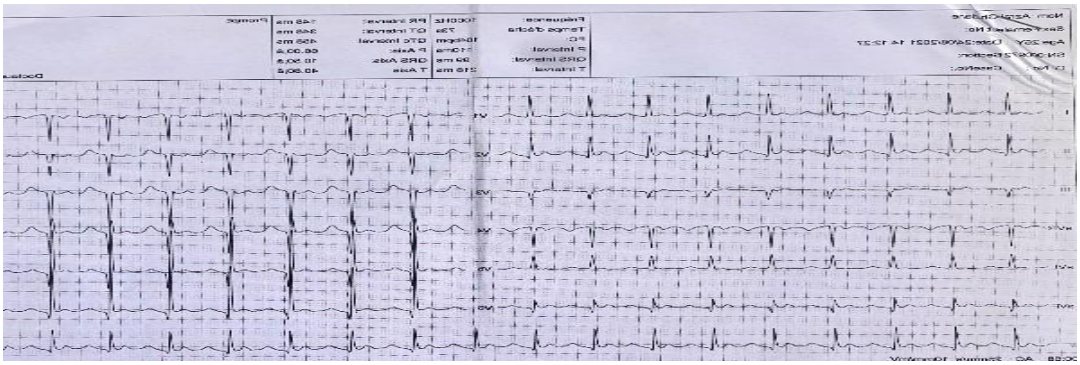
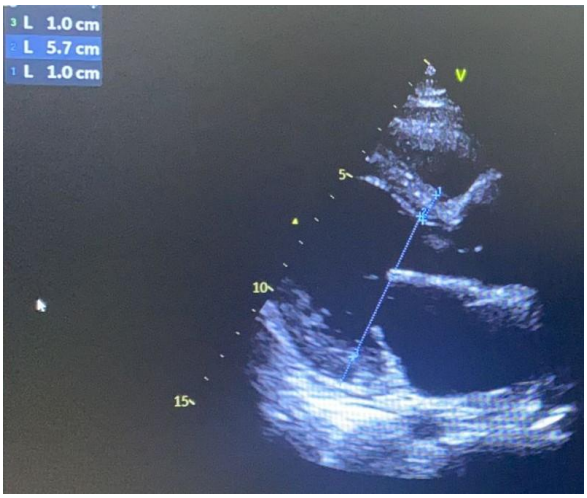
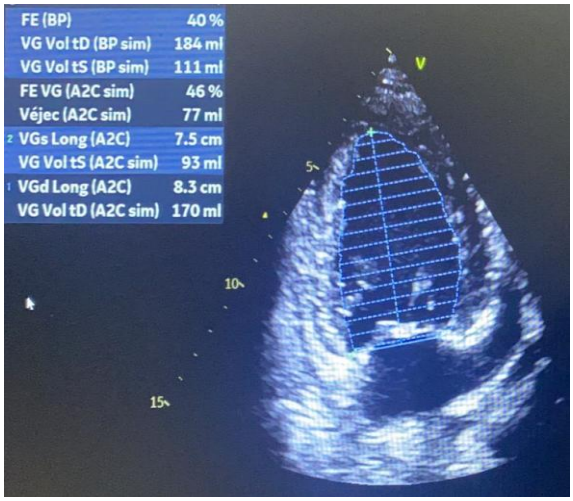


Fig.N1

(Fig.N1): Electrocardiography shows normal sinus rhythm



(Fig.N2)



(Fig.N3)

Fig.N2: PLAX view shows mild left ventricular (LV) enlargement indexed at 31cm/m^2

Fig.N3: TTE shows moderate reduce of ejection fraction estimated by Simpson biplane a 46%.

3D Computed tomography angiography (CTA) thoraco-abdominal aorta presents caliberanomalies associated with abnormalities parietal do as follows:

- At the ascending and horizontal thoracic level: it is of subnormal caliber with wall fine, normally pacified without loss of parallelism of its walls. (Ring: 22x24.4mm, Valsalva sinuses: 35x32.5x32.6mm, Sino tubular junction: 30.7x32.3mm. Ascending aorta: 35.3x37mm, Aortic arch: 34.4x35mm) , (black fleshs at Fig.N4).
- Descending aorta: beyond the isthmus it loses its caliber so decreasing, presenting a circumferential and regular wall thickening measured at 4 mm (Isthmus 25.9x23.1mm, Diaphragmatic aorta: 10.5x10.5mm) (Red fleshs at Fig.N4).
- At the abdominal level: it gradually loses its caliber until it is completely invisible under renal (at L3 height) (yallow fleshs at Fig.N5 - Fig.6 - Fig.7)
- Over a distance of 69mm it presents a first strict renal stenosis (up to D12) estimated at 77% in surface area and 52.5% in diameter according to NASCET criteria (orange flesh at Fig.N5). Repermeabilization of the iliac and digestive arteries refer to the development of a large net of collaterals (green flesh at Fig.N5) . No intimal flap or sign of aortic dissection.

In peripheral vascular system we it demonstrated a very extensive stenosis of the left subclavian artery, permeable , evaluated at 88% in surface and 65.6% in diameter according to the criteria NASCET .
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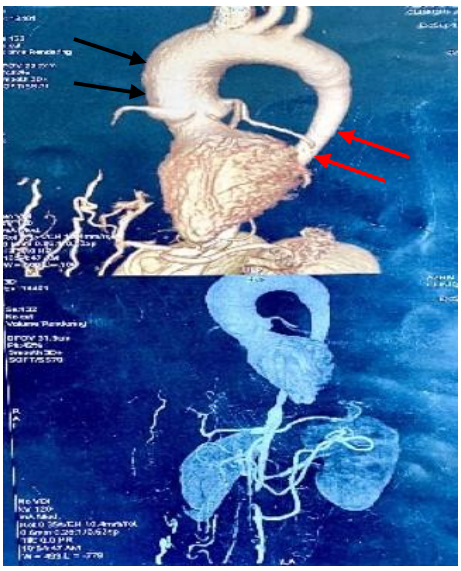

Fig.N4

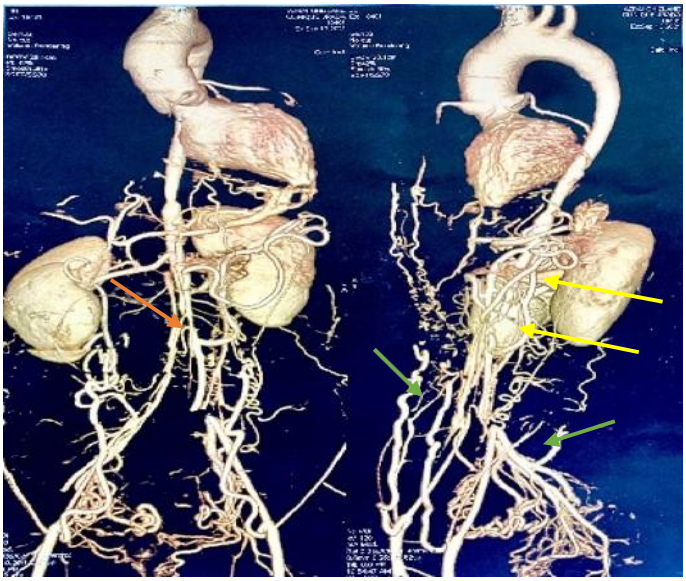

Fig.N5

Fig.N4: 3D-CTA thoracic-abdominal shows ascending and horizontal aorta with subnormal caliber (Black flesh), in contrast descending aorta beyond the isthmus decrease its caliber progressively (Red flesh).
Fig.N5: 3D-CTA thoracic-abdominal shows abdominal aorta gradually loses its caliber until it is completely invisible under renal (yallow fleshs) made its first strict renal stenosis up to D12 estimated at 77% in surface area and 52.5% in diameter according to NASCET criteria (orange flesh) with development of a large net of collaterals (green fleshs).

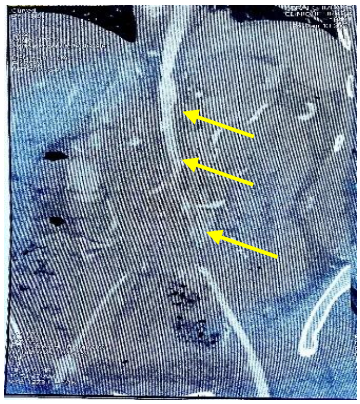

Fig.6

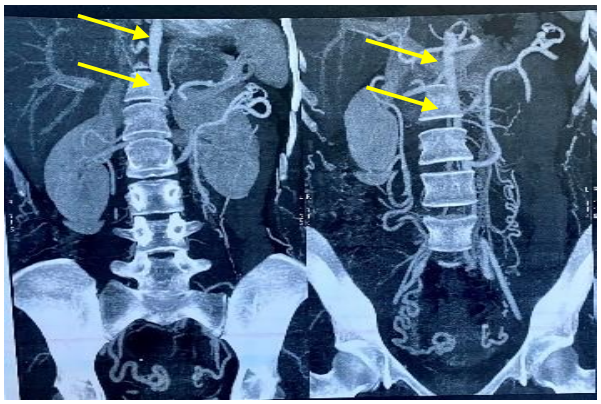

Fig.7

Fig.6-Fig.7: Abdominal aorta Computed tomography angiography shows how aorta caliber's completely invisible under renal level at L3 height fulfilled the aspect of aortic hypoplasia (yallow fleshs).

Based on the above finding she was diagnosed as having takayasu arteritis and related myocarditis. After an opinion of internal medicine, we prescribed high-dose prednisolone 1 mg/kg/day and 5 mg of ACE inhibitor, prevent atherosclerosis aspirin, and statin to and with regular clinical, ultrasound and biological control.

Tow month later she reported improvement in her fatigue and arm claudication but blood pressure still asymmetric and we avoid forcing the anti-hypetenseur to avoid the ischemia of her left hand ; and full recovery in cardiac function with EF 60% . in laboratory data; the ESR declined to 157 mm/hour, and CRP was 2.4 mg/L, that we considered as a favorable response to treatment .We planned a tapering program and long-term low-dose continuation after that , patient was presented to the heat team and vascular surgeons to revascularization her left subclavian artery and aortic reconstruction .

III. Discussion

The diagnosis of Takayasu's disease is based on the conver- gence of clinical, biological, and radiological elements grouped according to Sharma criteria [3] .Classically, there are 2 evolutionary phases of Takayasu's disease. The initial stage is called "preocclusive," characterized by general and nonspe- cific signs, such as arthralgia, erythema nodosum, pyoderma gangrenosum, carotidynia, and sometimes ocular lesions as episcleritis or anterior uveitis [4]. The "occlusive" or "vascular" phase is characterized by symptoms related to arterial stenosis or aneurysm; therefore, ischemic complications are in the foreground [4].

We can find the increasing of some nonspecific biological markers like C-reactive protein (CRP), alpha-2 globulins, the erythrocyte sedimentation rate (ESR), and fibrinogen. Pentraxin-3 is currently considered an activity marker for Takayasu disease [5] . Radiological exploration is founded on Doppler ultrasound, CT angiography, and Magnetic resonance angiography (MRA) that are far more sensitive than angiography on detecting lesions at the initial stage [6] . The essential imaging finding is the mural thickening of an arterial segment, typically smooth, circumferential, and exceeding 3 mm, forming the

« macaroni »sign. This aspect is found at 97% in the preocclusive phase, with a high prevalence in the carotid and subclavian arteries [4,6]. The disease's activity can be assessed on postcontrast delayed phase images (10 minutes after injection of contrast agent), which shows a delayed enhancement of the active thickening as a "double-ring sign." The transition zones between the pathological and healthy arterial segments are abrupt, giving the appearance of a suspended lesion .

Takayasu disease's lesions can be complicated by arterial stenosis, aneurysms, baroreceptors abnormalities, and a drop in arterial compliance, with repercussions on the blood flow and arterial hypertension [4 ,7] . Ischemia is a frequent complication that occurs following hypoperfusion.

Our patient presents a left subclavian artery stenosis responsible of asymmetric blood pressure as well as the abdominal aortic hypoplasia responsible of her legs claudication intermittent (Fig. 2.3.4

). Dissection Stanford type B is more frequent than type A in association with Takayasu disease , the subclavian artery stenosis has frequent association with Takayasu disease may To our knowledge, our case is the first symptomatic aortic hypoplasia reported as a complication of Takayasu disease .

The presence of a circumferential and regular parietal thickening of the aorta is typically related to an inflammatory origin that is compatible with Takayasu disease, and less likely consistentwith an atheromatous background, given the absence of calcifications and the young age of the patient

.the literature review reports only a few cases of this kind of aortic complication complicating Takayasu disease .

The surgical treatment indications in the vascular complications of takayasu disease are not clearly defined, dominated by ischemic manifestations. Nevertheless, it is recommended to perform the surgery beyond the inflammatory phase of the disease.

IV. Conclusion

The aim of this work was to study the cardiac complication of Takayasu's disease, clinical patterns, therapeutic particularities and the outcome of patients with hypertension, and showed the rare case of aortic hypoplasia in Takayasu's disease. Hypertension incidence in Takayasu's disease is hard to appreciate because blood pressure is often up or under-estimated when supra-aortic trunks and the 2 sub-clavian arteries were involved altering hence wave pulse propagation and blood pressure clinical assessment. Finally, what is the place of surgical and endovascular interventions for treatment of HTA in Takayasu arteritis?

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