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Case Report

Exploring the uncharted: Takayasu arteritis masquerading as severe preeclampsia in a high-risk pregnancy: a rare case report

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ABSTRACT

Takayasu's arteritis is a chronic vasculitis of medium and large vessels. The most involved vessel is the aorta and its major branches. The vessels are characterized by mononuclear infiltration and granulomatous inflammation of vascular media, which leads to arterial wall thickening with stenosis, occlusion, and aneurysmal dilation. Here we present a case of a 32-year-old female patient who presented with headache and mild epigastric pain. CT Aortogram was done which revealed severe stenosis of the left thoracic and upper abdominal aorta measuring 8 cm long along with left subclavian artery stenosis. Delayed diagnosis and lack of specific treatment could explain the extent and the clinical severity of the disease at the time of hospital admission. Therefore, early diagnosis and treatment are warranted. When the disease is dormant, the outcome seems favourable.

Keywords: Takayasu's arteritis, Chronic vasculitis, CT aortogram

INTRODUCTION

Takayasu arteritis (TA) is a large vessel vasculitis of unknown etiology which commonly affects young women. Although pregnancy is a state favourable to this disease, this disease can adversely affect pregnancy.[1] The diagnosis of TA is usually made by the combination of clinical characteristics and radiologic imaging methods.[1] Computerized tomography or magnetic resonance (MR) angiography is routinely used in practice for diagnosis of TA, and updated narrative reviews continue to refine recommendations for imaging surveillance across pregnancy.[2] Complications should be anticipated, and close multidisciplinary maternal and fetal surveillance is mandatory, particularly as obstetric outcomes have been shown to be disproportionately poor among Indian women with this condition.[3] Poor outcomes in pregnancy are often associated with late diagnosis of this condition and severe hypertension.[4] Several drugs are available for treatment of TA, but the most effective treatment remains corticosteroids. Surgical intervention is required in cases with vascular complications.

CASE REPORT

A 32-year-old female, primigravida of 39 weeks gestation, was brought to our hospital casualty with headache and mild epigastric pain. She was a diagnosed case of pregnancy-induced hypertension (PIH) on labetalol 100 mg BD.

On initial assessment, SBP of 160 mmHg and DBP of 100 mmHg were recorded on her right forearm. On examination, a closed cervix without any detectable bleeding was noted. The FHR was 145 beats per minute. She was an unbooked, unregistered case with a single anomaly scan at 20.6 weeks that was normal.

Emergency blood work was carried out, with results showing Hb 12.1 g/dl, platelets 2.1 lakh, Aspartate Aminotransferase (AST) at 1980 U/l and Alanine

Aminotransferase (ALT) at 120 U/l, creatinine 0.8 mg/l, total bilirubin 0.72, with proteinuria. The patient was put on injectable anti-hypertensive treatment and monitored in the labour room for induction of labour. Upon fetal distress, she was taken up for an emergency caesarean section. Intraoperatively, the placenta was found adherent to the uterine wall (Figure 1), due to which an obstetric hysterectomy was performed (Figure 2). She was shifted to the intensive care unit for monitoring in view of raised blood pressure. It was soon found that the patient had a BP of 180/100 mmHg in her right upper limb and 130/100 mmHg in her left limb, a discrepancy that, although a classical feature of large-vessel vasculitis, is easily overlooked in the peripartum period when attention is naturally focused on the standard obstetric determinants of hypertensive disease.[1] Coarctation of the aorta (COA) was one of the differential diagnoses; hence a CT Aortogram was done, which revealed severe stenosis of the left thoracic and upper abdominal aorta measuring 8 cm long, along with left subclavian artery stenosis (Figure 3).

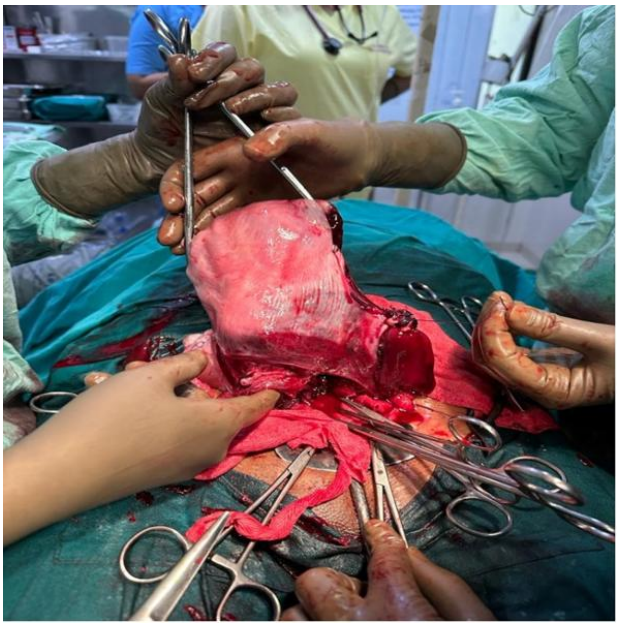

Figure 1: Intraoperative photograph showing the gravid uterus with the placenta densely adherent to the uterine wall, noted at the time of emergency caesarean section.

CT Brain (plain) was done, which showed bilateral basal ganglia calcification, considered physiological. The patient was then started on anti-inflammatory medication and a low-dose beta blocker to regulate her blood pressure.

During the course of the pregnancy, maternal complications should be anticipated, including sustained hypertension, superimposed preeclampsia, congestive heart failure, and progression of renal insufficiency.

Therefore, when such conditions supervene, TA should always be kept in mind in the differential diagnosis. The diagnosis of TA can be confirmed by means of magnetic resonance angiography, as this is safer for the fetus.[2]

Figure 2: Intraoperative photograph of the surgical specimen following obstetric hysterectomy, showing the placenta invading the uterine wall.

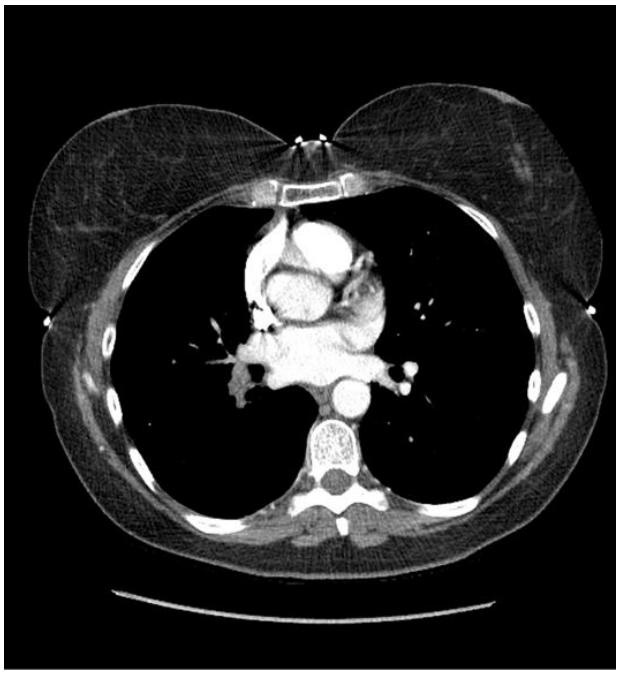

Figure 3: Axial CT aortogram image demonstrating luminal narrowing of the aortic arch and its major branches, consistent with large-vessel vasculitis.

In retrospect, this case highlights how TA can closely mimic, and in this instance appeared superimposed upon, severe preeclampsia, since both conditions may present with headache, epigastric pain, and hypertension

refractory to conventional antihypertensive therapy.[1] The unequal blood pressures recorded between the two upper limbs proved to be the pivotal clinical clue that prompted vascular imaging and ultimately established the correct diagnosis, reinforcing that bilateral upper-limb blood pressure measurement should form part of the routine assessment of severe or atypical hypertensive disorders of pregnancy.[4] We believe a high index of suspicion for large-vessel vasculitis should be maintained in any pregnant woman whose hypertension is disproportionate to the expected disease course or is accompanied by discrepant peripheral pulses, particularly in populations such as ours where TA is not classically anticipated as a cause of hypertension in pregnancy.[3] This case also reinforces the value of a multidisciplinary approach, involving obstetrics, rheumatology, cardiology, and critical care once the diagnosis is suspected, to optimise both maternal vascular status and fetal wellbeing.[2]

DISCUSSION

TA is a chronic, granulomatous, large vessel vasculitis of unknown origin. TA usually affects young women (<40 years) of reproductive age, whereas giant cell arteritis (GCA), also a large vessel vasculitis, usually affects the elderly. TA commonly affects the aorta and its primary branches and rarely involves the pulmonary and coronary arteries. It predominantly affects women more than men, in a ratio of 9:1. TA was first described among Japanese women.

Pathologically, it is characterized by a chronic, progressive and obliterative non-specific panarteritis involving all layers of the vessel wall, which causes thickening of arterial walls and may lead to stenosis, occlusion, dilatation, or aneurysm formation as the disease progresses. Histopathologic diagnosis is generally not possible because TA involves large blood vessels. TA is often referred to as "pulseless disease" because it causes full-layer arterial inflammation leading to vascular stenosis or obstruction in affected vessels.

The diagnosis of TA is made by combining clinical characteristics with radiologic imaging. Clinical presentation varies from asymptomatic (due to collateral formation and insidious onset) to claudication of the limbs, arterial bruits, discrepant blood pressure between arms, hypertension, angina, nonspecific constitutional symptoms, arthralgias, absent or weak peripheral pulses, and neurologic symptoms depending on disease severity.[1] The most common presentation of TA in India is hypertension.[5] Therefore, TA should be considered an important differential diagnosis for hypertension in pregnancy.

Proper radiologic imaging is essential for accurate diagnosis of TA.[1] Conventional angiography used to be the gold standard but is now frequently replaced by CT or MR angiography in routine practice. FDG-PET/CT has emerged as a new diagnostic tool, as it can detect vascular inflammation even before structural changes occur.

Obstetric complications such as gestational hypertension, pre-eclampsia, spontaneous abortion, intrauterine growth restriction (IUGR), preterm birth, placental abruption, intrauterine death, and the need for caesarean section appear to be more frequent in pregnancies of patients with TA.[6] TA is also associated with serious maternal complications such as aortic regurgitation, congestive heart failure, renal insufficiency, antepartum haemorrhage, pulmonary embolism, and myocardial infarction. A recent systematic review and meta-analysis of pregnancies in TA patients confirmed a substantially elevated prevalence of hypertensive complications, preterm birth, and pregnancy loss compared with the general obstetric population, underscoring the need for close antenatal surveillance.[7]

Several factors affect fetomaternal outcomes in TA, including time of diagnosis, control of hypertension, and extent of vascular involvement. Ideally, pre-conceptional counselling should be undertaken in patients diagnosed with TA prior to pregnancy to assess disease activity, optimize blood pressure control, and switch to medications considered safe in pregnancy. Pregnancy should be planned when TA is in sustained remission. Medications such as methotrexate, mycophenolate mofetil, cyclophosphamide, and angiotensin-converting enzyme inhibitors should be avoided during pregnancy due to the risk of teratogenicity.

The preferred mode of delivery following an uncomplicated pregnancy in a patient with TA is the vaginal route, with continuous intrapartum electronic fetal monitoring. Caesarean section is reserved for obstetric indications.

Glucocorticoids, methotrexate, leflunomide, mycophenolate mofetil, tocilizumab, azathioprine, and anti-tumor necrosis factor (TNF)-alpha agents are among the treatment options for TA. Management of TA in pregnancy most frequently involves low-dose prednisone (1 mg/kg) with interruption of azathioprine, if used, until delivery.[8] Recent case-based experience also supports biologic-sparing, corticosteroid-minimizing regimens (including anti-TNF therapy) as a feasible option for maintaining remission through successful pregnancies in selected patients with TA, particularly where standard immunosuppressants are contraindicated.[9] Daily low-dose aspirin has also proved to be a useful prophylactic measure against hypertensive complications.

The most effective treatment remains corticosteroids.8 However, relapses occur in a substantial proportion of patients during corticosteroid tapering. In addition to immunosuppressive treatment, patients with ischemic symptoms may require endovascular intervention or vascular surgery.

CONCLUSION

TA is a chronic disease that progresses with relapses and remissions. Pregnancy may affect the diagnosis, outcome, and management of TA. Adequate control of hypertension during pregnancy, careful planning of the timing and mode of delivery, and close monitoring during the intrapartum period are essential for an optimum outcome. Pregnancies in the setting of TA should be considered high-risk, requiring close collaboration between rheumatologists and obstetricians.

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