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Case Report- A Rare Presentation of Takayasu Arteritis

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I. INTRODUCTION

Takayasu's arteritis (TA) is a large vessel vasculitis of unknown etiologycharacterised by a chronic granulomatous panarteritis of the aorta and its major branches.The chronic and progressive vessel wall inflammation leads to concentric wall thickening and stenosis producing a variety of ischemic symptoms or aneurysms, with a high incidence of morbidity and a significant risk of early death.[1]The etiopathogenesis of this disease is still poorly understood, but an autoimmune basis is widely suggested. In addition, genetic and environmental factors also probably play an important role.[1] Among the environmental factors, evidence implicating Mycobacterium tuberculosis (MT) has been provided for more than five decades (2,3,4) Infectious diseases, particularly Mycobacterium tuberculosis, can be a trigger for the development of TA2 [3] as a hypersensitivity reaction.[4] In contrast, inflammation of the aorta may be secondary to the direct invasion of the artery by the M. tuberculosis.

II. CASE REPORT

HISTORY

A rare case of 8year old female who presented with breathlessness (NYCA GRADE 3) and vomiting since 20 days with a positive family history of TB contact

On examination

GC- CRITICAL

HR-132/MIN

RR-44/MIN

PV -FEEBLE IN B/L UPPER LIMB
ABSENT IN B/L LOWER LIMB

BP- IN RT UL-80/50mmHG and LT UL-82/56mmHG

RT AND LT LL-NOT DETECTABLE.

SYSTEMIC EXAMINATION

CVS-RAISED JVP,PECTUS CARINATUM, APEX BEAT -2CM LAT TO MCL,OUTWARDOWNWARD IN 6TH ICS.TACHYCARDIA,S1 S2 HEARD

PER ABDOMEN -LIVER-6.5CM PALPABLE,SOFT ,MILD TENDERNESS PRESENT.

RESPIRATORY SYSTEM-SCR,ICR PRESENT.NASAL FLARING WAS PRESENT, B/L CREPITATIONS PRESENT.

INVESTIGATIONS

On admission- HB-10mg/dl,TC-11900,ESR-15mm/1hr,CRP-4.28Na-136,K-4.7,CL-104, UREA- 43, CREATININE-1.3.Ca-7.5SGOT-207,SGPT-106,Sickling-negative,CPKMB-54,TROPONINE- NEGATIVE, ASO TITRE-NEGATIVE.MT- POSITIVE(15 MM INDURATION).

USG ABDO/PELVIS- S/O PERICARDIAL EFFUSION, CONGESTIVE CHANGES IN LIVER, FREE FLUID IN MORRISON'S POUCH AND PELVIS.2D ECHO - Severe LV dysfunction with mild AR, moderate MR with normal aortic arch

Thus on the basis of clinical presentation and basic investigation , child was dignosed to have ccf with mr with lv dysfunctin and pericardial effusion and was thus managed accoringly. but on doa-4 , when the signs of ccf resolved , lower limbs pulses and blood pressure werenot still detectable and thus we planned to get a ctaortogram done.

CT AORTOGRAM was Suggestive of takayasuarteritis , affecting the thoracic aorta ,probably of tuberculous origin.

III. RESULT

Hence, the child was diagnosed to have takayasu'sarteritis with tubercular origin and was started with AKT AND PREDNISOLONE.On

follow up , child was found to have shown significant improvement.

IV. DISCUSSION

The relationship between TB and TA is well known. Some patients with TA have had previous contact with M. tuberculosis, shown either by the positivity of the skin tuberculin test[5 6] or by the detection of specific gene sequences of the bacillus in aortic tissue.[7] The present case is intriguing because her manifestations did not allow for the distinction of whether or not it was a true TA in a patient previously exposed to M. tuberculosis or a tuberculous arteritis itself.The natural history of this arteritis is highly variable. The response of TA to anti-tubercular drugs remains controversial among authors; Pantell and Goodman [4] described the case of a complete symptomatic remission as well as the return of pulses simultaneous with anti-Tb therapy. However, most reports on co-occurrence of active Tb and TA combined antitubercular drugs and corticosteroids. In our report, antitubercular drugs, had no effect on TA vasculitis and did not prevent new relapses. Hahn et al.[9] reported that 90% of their patients had strongly positive Mantoux tests, usually without active Tb, suggesting an autoimmune trigger.The literature currently hypothesizes an autoimmune basis[10] and not a direct role of MT. Indeed, Arnaud et al.[11] failed to detect MT in arterial lesions of either active or inactive TA but did not exclude the possibility of a cross-reaction between mycobacterial and arterial antigens. Aggarwal et al.[12] showed that patients with TA have heightened humoral response to mycobacterial antigens, including the 65 kDa fraction, a heat shock protein that has also been found to be expressed in the arterial wall of TA patients. Recently, Soto et al.[13] identified in a case and control study a higher frequency of IS6110 and hupB gene sequences of MT and bovis in the aortic tissue of TA patients and in Tb compared to patients with atherosclerosis with important statistical differences suggesting that arterial damage could occur due to the previous infection with MT. Finally, the exact pathogenic sequence between TA and Tb remains to be elucidated.

The exact etiology of Takayasu arteritis is not known. A number of features suggest an autoimmune base while others raise the question that the aortitis may be an expression of tuberculin sensitization.[14]It is characterized histologically by an inflammatory cell infiltrate that affects all the layers of the arterial wall, especially the aorta and its major branches. Its incidence varies between 1.2 and 2.3 cases per million per year, and it is more common in Asians than in other racial groups. An exact epidemiological figure from our region is not available.[15]

Takayasu arteritis is a chronic vasculitis mainly involving the aorta and its main branches, such as the brachiocephalic, carotid, subclavian, vertebral and renal arteries, as well as the coronary and pulmonary arteries.[16] It induces clinically varied ischaemic symptoms due to stenotic lesions or thrombus formation, including blindness, cataract and/or retinal hemorrhage, pulselessness, aortic regurgitation and/or congestive heart failure due to dilatation of the ascending aorta. More acute progression causes destruction of the media of the arterial wall, leading to the formation of aneurysms and/or dissecting aneurysm or rupture of the involved arteries. [17] The presentation differs in different demographic locations while almost all patients in Japan, have ischemic disorders due to cervical lesions, presenting with dizziness, syncope, visual disturbance, faint or absent pulse, or differences in systolic blood pressure between arms in Western countries this disease usually present with absent pulses secondary to obstruction of subclavian or brachial arteries.[18] A causal relationship between TA and tuberculosis (TB) had been suggested. Both diseases show similar pathological changes in the form of chronic inflammatory lesions and, occasionally, granulomas in the arterial walls. The genetic relationship between these two diseases has not been reported to exist until now however, both diseases have been associated with human leukocyte antigen (HLA) alleles, cold agglutinins and cryoglobulins during the acute phase of the illness.[19] Mycobacterium tuberculosis has been implicated in the pathogenesis of Takayasu arteritis (TA). Recently, its 65 kDa heat shock protein (HSP) has been implicated in the pathogenesis of other autoimmune diseases. Patients with TA have heightened immune response to Mycobacterium tuberculosis antigens, in particular to its 65 kDa HSP, suggesting that this organism may have a role in the immunopathogenesis of this disease.[20] The co-occurrence of Takayasu Arteritis and tuberculosis pose a dilemma for treating pediatricians or physicians since most patients eventually receive AKT along with steroids or immunosuppression the improvement cannot be attributed to any one form of this drug therapy. However it is important not to ignore the need for co-administration of AKT otherwise immunosuppression caused by steroids in these patients may cause disseminated tuberculosis.

There are reports which describe the cases of complete symptomatic remission as well as the return of pulses simultaneous with anti-Tb therapy. The investigators so far have failed to isolate Mycobacterium tuberculosis in arterial lesions of either active or inactive TA but this does not necessarily exclude the possibility of a cross-reaction between mycobacterial and arterial antigens.

V. CONCLUSION

1) TA is a systemic vasculopathy which can progress to cause vital organ ischemia.
2) TB should be kept in mind during exploration of the etiopathology of TA mainly in a region where TB is common.
3) Anti-Tb therapy is rationale during the treatment of this disease.

BIBLIOGRAPHY

[1]. Brunner J, Feldman BM, Tyrrell PN, Kuemmerle-Deschner JB, Zimmerhackl LB, Gassner I, et al. Takayasu arteritis in children and adolescents. Rheumatology (Oxford) 2010;49:1806–14. [PubMed]
[2]. Weiss PF. Pediatric vasculitis. PediatrClin North Am. 2012;59:407–23. [PMC free article] [PubMed]
[3]. Duzova A, Türkmen O, Cinar A, Cekirge S, Saatci U, Ozen S. Takayasu's arteritis and tuberculosis: A case report. ClinRheumatol. 2000;19:486–9. [PubMed]
[4]. Pantell RH, Goodman BW., JrTakayasu's arteritis: The relationship with tuberculosis. Pediatrics. 1981;67:84–8. [PubMed]
[5]. Robles M, Reyes PA. Takayasu's arteritis in Mexico: a clinical review of 44 consecutive cases. ClinExpRheumatol 1994;12:381–8. [PubMed]
[6]. Nooshin D, Neda P, Shahdokht S et al. Ten-year investigation of clinical, laboratory and radiologic manifestations and complications in patients with Takayasu's arteritis in three university hospitals. Malays J Med Sci 2013;20:44–50. [PMC free article] [PubMed]
[7]. Soto ME, Del Carmen Avila-Casado M, Huesca-Gomez C et al. Detection of IS6110 and HupB gene sequences of Mycobacterium tuberculosis and bovis in the aortic tissue of patients with Takayasu's arteritis. BMC Infect Dis 2012;12:194 doi:10.1186/1471-2334-12-194 [PMC free article] [PubMed]
[8]. Kroot EJ, Hazes JM, Colin EM et al. Poncet's disease: reactive arthritis accompanying tuberculosis. Two case reports and a review of the literature. Rheumatology (Oxford) 2007;46:484–9. doi:10.1093/rheumatology/kel268 [PubMed]
[9]. Hahn D, Thomson PD, Kala U, Beale PG, Levin SE. A review of Takayasu's arteritis in children in Gauteng, South Africa. PediatrNephrol. 1998;12:668–75. [PubMed]
[10]. Arnaud L, Haroche J, Mathian A, Gorochov G, Amoura Z. Pathogenesis of Takayasu's arteritis: A 2011 update. Autoimmun Rev. 2011;11:61–7. [PubMed]
[11]. Arnaud L, Cambau E, Brocheriou I, Koskas F, Kieffer E, Piette JC, et al. Absence of Mycobacterium tuberculosis in arterial lesions from patients with Takayasu's arteritis. J Rheumatol. 2009;36:1682–5. [PubMed]
[12]. Aggarwal A, Chag M, Sinha N, Naik S. Takayasu's arteritis: Role of Mycobacterium tuberculosis and its 65 kDa heat shock protein. Int J Cardiol. 1996;55:49–55. [PubMed]
[13]. Soto ME, Del Carmen Ávila-Casado M, Huesca-Gómez C, Alarcon GV, Castrejon V, Soto V, et al. Detection of IS6110 and HupB gene sequences of Mycobacterium tuberculosis and bovis in the aortic tissue of patients with Takayasu's arteritis. BMC Infect Dis. 2012;12:194. [PMC free article] [PubMed]
[14]. Takayasu M. A case with peculiar changes of the retinal central vessels (in Japanese). ActaSocOphthalJpn 1908;12:554–5.
[15]. Sato EI, Hatta FS, Levy-Neto M, Fernandes S. Demographic, clinical, andangiographic data of patients with Takayasu arteritis in Brazil. Int J Cardiol.1998 Oct 1;66Suppl 1:S67-70;
[16]. Yamada I, Shibuya H, Matsubara O, Umehara I, Makino T, Numano F, Suzuki S.Pulmonary artery disease in Takayasu arteritis: angiographic findings. AJR Am JRoentgenol. 1992 Aug;159(2):263-9.
[17]. Lee GY, Jang SY, Ko SM, Kim EK, Lee SH, Han H, Choi SH, Kim YW, Choe YH, KimDK. Cardiovascular manifestations of Takayasu arteritis and their relationship tothe disease activity: analysis of 204 Korean patients at a single center. IntJCardiol. 2012 Aug 9;159(1):14-20.
[18]. Strachan RW. Pre-Pulseless and Pulseless Takayasus' Arteritis. Postgraduate Medical Journal. 1966;42(489):464- 468.
[19]. Khemiri M, Douira W, Barsaoui S. Co-occurrence of Takayasu arteritis and

tuberculosis: Report of a Tunisian pediatric case. Annals of Pediatric Cardiology. 2016;9(1):75- 78.

[20]. Pantell RH, Goodman BW., JrTakayasu arteritis: The relationship with tuberculosis. Pediatrics. 1981;67:84–8