Open Access Library Journal
2023, Volume 10, e10019
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Respiratory Distress Revealing Takayasu's Disease: Case Report

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How to cite this paper: Zerouati, K., Rkain, M. and Babakhoya, A. (2023) Respiratory Distress Revealing Takayasu's Disease: Case Report. *Open Access Library Journal*, **10**: e10019.
https://doi.org/10.4236/oalib.1110019

Received: March 16, 2023
Accepted: April 25, 2023
Published: April 28, 2023

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Abstract

Takayasu's arteritis is a chronic inflammatory, autoimmune, granulomatous disease of the aorta and its major branches, at their origin, resulting in dilatation, occlusion, stenosis and/or aneurysm formation of these arteries. The disease usually begins in adulthood but pediatric involvement is very rare and the diagnosis is late. We report the case of a 13-year-old child admitted for respiratory distress, whose detailed clinical examination and CT angiography confirmed the diagnosis.

Subject Areas

Cardiology, Emergency & Critical Care, Pediatrics

Keywords

Heart Failure, Respiratory Distress, Takayasu's Disease

1. Introduction

Takayasu's disease (TD) is a vasculitis of large vessels affecting the aorta, its main branches and pulmonary arteries. The diagnosis of TD is often difficult and delayed in children because of the large clinical polymorphism and lack of specific biological criteria [1].

Takayasu's disease remains rare in children, its clinical aspects are variable whose diagnosis is based on vascular imaging, this clinical case describes a 13-year-old child who presented with respiratory distress revealing Takayasu's disease after clinical investigation.

2. Case Report

A 13-year-old male child from a non-consanguineous marriage, with no person-

al medical history and with a notion of tuberculosis contusion, presented to the emergency department for dyspnea and asthenia from 3 weeks before his admission aggravated by apparition of respiratory distress.

The clinical examination upon his admission found:

- High blood pressure at 140/95 mmHg,
- A decrease in femoralpulse,
- Signs of heartfailure: cough and congestion in thelungs, visible swelling of the legs, ankles, abnormally fastbreathing, shortness ofbreath, tired
- Right basithoracic fluid effusionsyndrome.

The biological assessment showed an inflammatory syndrome: withe blood cells 15,400/mm^3, lymphocytes 850/mm^3, and C-Reactive-Protein CRP of 50 mg/l.

Chest X-ray showed bilateral alveolar opacities and right pleurisy of moderate abundance. Thoraco-abdominal CT showed thickening of the bilateral peri-alveolar septas associated with pleural effusion and thickening of the aortic walls with narrowing of its caliber and thrombosis of the right renal artery.

ETT has shown left ventricular hypertrophy.

The association HTA, inflammatory syndrome, thickening of the aortic wall and stenosis of the right renal artery led to the diagnosis of takayasu disease.

The patient was treated with a bolus of corticosteroids in combination with antihypertensive therapy.

The evolution was marked by the installation of acute ischemia of the right lower limb and then of the left inferior limb, the angioscanner objectified a stenosis of the common femoral artery, the child benefited from a fogartisation, complicated by bilateralblindness

The patient presented cardiac arrhythmias followed by cardiac arrest.

The child died after 10 days of his admission.

3. Discussion

At the epidemiological level, there is a higher prevalence in Asia, South America and North Africa [1].

The association of TD and tuberculosis, particularly in Africa, is not yet clearly established [2].

More recently, immunological data has made it possible to highlight the important role of lymphocytes and heat shock proteins (Hsp60) [3].

Clinically, the disease begins in an acute form in the child, with more severe general manifestations. However, the delay between the first symptoms and the diagnosis is four times greater than that found in adults [4].

The macroscopic study of the material obtained during vascular surgery shows thickened and fibrous vessels, which may be the site of stenosis and/or aneurysms. Microscopic examination shows a segmental panarteritis with a predominantly meso-supportive pattern [5].

The disease evolves classically in two phases separated from an asymptomatic period. The first, called “preocclusive”, is characterized by the presence of general

signs, rheumatological manifestations, and sometimes a table of granulomatosis and by a biological inflammatory syndrome [6] [7]. The second, called "occlusive" is insidious, resulting in the occurrence of ischemic clinical manifestations.

It can evolve over several years and lead to renovascular arterial hypertension by unilateral or bilateral stenosis of the renal arteries but is sometimes consecutive to coarctation of the aorta [8].

This dogma is nevertheless questioned by the large proportion of patients who have, at the outset, general signs and ischemic vascular manifestations. Renal artery involvement in children is present in 25% to 75% of cases. Inaugural hypertension is the most common mode of disclosure in children and may be complicated by malignant hypertension, hypertensive encephalopathy, or even heart failure [9] [10].

The four most frequently affected arterial territories are the thoracic (48%), abdominal (48%), right (52%) and left (48%) renal arteries. Different diagnostic criteria of Fiessinger *et al*. and the American College of Rheumatology are available [11] [12].

In 2006, the diagnostic criteria were modified and adapted to pediatrics [13].

MRI is a non-invasive, non-radiative examination that visualizes both the wall and the vascular lumen. Positron emission tomography (PET scan) could be a future examination, to visualize the presence of hyper-metabolic foci in the arterial wall [14].

The progressive follow-up of the disease is ensured by noninvasive radiological examinations [15] [16].

MRI angiography has taken a prominent place in the detection of early-onset attacks showing T2-weighted sequence-mediated parietal edema [14].

In practice, panaoretic angio-MRI and Doppler ultrasound have become the gold standard for patient monitoring. There is no specific biological test for MT and abnormalities observed during routine examinations are only a direct or indirect reflection of the underlying inflammatory syndrome. Antinuclear antibodies and rheumatoid factors are sometimes positive but always at low levels.

The ANCA search is negative.

The realization of an HLA typing is of no diagnostic interest.

The medical treatment of TM is currently not based on any controlled trials and has no consensus in pediatrics. In the acute phase, vascular lesions may be reversible. Corticosteroids at 1 or 2 mg/kg/day are proposed as first-line therapy. In most series of the literature, the control of the disease under a corticotherapy alone seems insufficient in 50% of the patients. A second-line immunosuppressive treatment is then necessary: methotrexate, azathioprine, mycophenolate mofetil, cyclophosphamide.

Recently, Ozen *et al*. [17] in a preliminary work report a remission obtained in three of the four patients treated from the start by a combination cyclophosphamide and induction corticosteroids followed by a relay by methotrexate. With an evolutionary decline of one to seven years, no serious adverse events were noted.

If second-line treatment fails, mycophenolate mofetil [18] or anti-TNF [19] [20] can be discussed with interesting responses reported.

Recently, the use of an IL-6 receptor antagonist has been shown to be effective in a 20-year-old patient with refractory MT [21]. HTA is sometimes severe and difficult to stabilize.

In the acute phase, angiotensin converting enzyme inhibitors are not recommended until arterial blood pressure is established because of the risk of acute renal failure. Anticalcics will be preferred [22]. When arterial stenoses are fixed, angioplasty will be proposed, sometimes renal artery replantation or, in extreme cases, nephrectomy [23].

4. Conclusions

In our case, Takayasu's disease can be discovered fortuitously after respiratory distress or heart failure, even if the patient has not reported any particular history.

Management is made complex by the absence of effective diagnostic criteria, validated therapeutic strategies and reliable activity criteria.

It is essential to continue studies on the physiopathology of the disease, which will make it possible to propose more effective diagnostic criteria and to develop new therapeutic strategies.

Conflicts of Interest

The authors declare no conflicts of interest.

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