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LEFT VENTRICULAR HYPERTROPHY WITH FALSELY NORMAL BLOOD PRESSURE REVEALING TAKAYASU ARTERITIS

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ABSTRACT

We present the case of Takayasu's arteritis in a woman whose diagnosis was delayed and not suspected because of falsely normal blood pressure due to arterial stenoses. Echocardiographic examination identified concentric left ventricular hypertrophy of unknown cause.

We describe this interesting and instructive case with a short review of literature. This should refer to the necessity to remember this rare disease in our country too.

KEYWORDS: Takayasu arteritis, Left ventricular hypertrophy, hypertension.

INTRODUCTION

Systemic hypertension, including secondary causes, is commonly excluded in patients with normal blood pressure. Echocardiographic left ventricular (LV) hypertrophy (LVH) suggests some conditions, such as systemic hypertension, aortic stenosis, and rarely restrictive or hypertrophic cardiomyopathy. We report the case of a young woman with an LV hypertrophy developed due to a severe hypertension related to Takayasu's arteritis whose diagnosis was delayed and not suspected because of falsely normal BP due to arterial stenoses.

CASE REPORT

We are reporting a case of a 52-year-old, menopausal, non hypertensive woman who was hospitalized for an exertional dyspnea since 2 years with headache and intermittent upper limbs claudication.

Physical examination finds a regular rhythm, HR at 75bpm, a blood pressure at 90/70mmhg in the right arm where the radial pulse was very weak, while it was totally absent in the left arm, where the BP was unmeasurable. Femoral and foot pulses were felt. Crackles heard in both pulmonary bases. Auscultation finds a murmur in the right carotid path. An abdominal bruit irradiated to renal arteries was also detected. EKG showed sinus rhythm, with left ventricular hypertrophy with secondary repolarization disorders **(Figure 1)**.

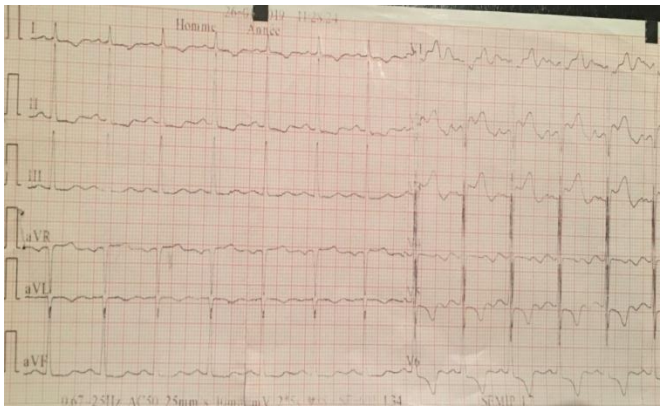

Figure 1: EKG showing left ventricular hypertrophy with secondary repolarization disorders.

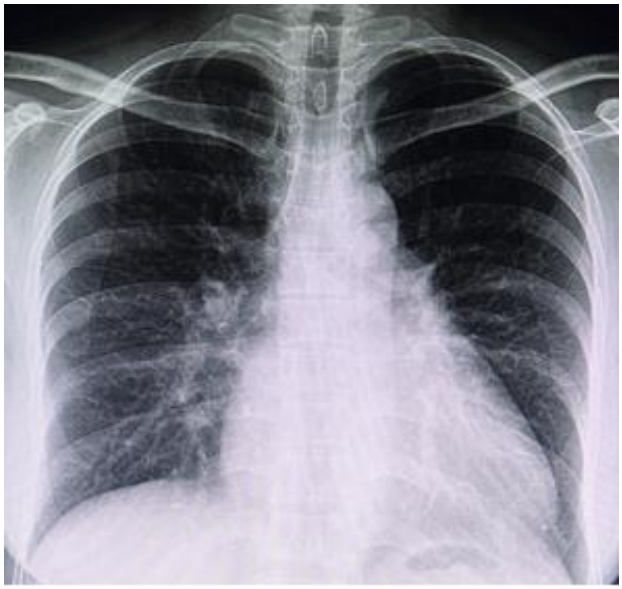

Figure 2: Thorax X-ray showing cardiomegaly whereas signs of left ventricular hypertrophy.

Transthoracic echocardiography revealed a concentric LV hypertrophy (wall thickness of 15mm), LVEF=55%, a restrictive mitral inflow pattern (E/A=2.3), enlarged left atrium with an area of 28cm², with no valvular abnormality or evidence of pulmonary arterial hypertension.

Laboratory tests shows signs of inflammation with a C-reactive protein at 51mg/l and an elevated erythrocyte sedimentation rate (first hour) at 48mm. Autoimmunity tests, including plasma antinucleus antibodies, Rheumatoid factor, Antineutrophil cytoplasmic antibodies, were negative.

Echo-Doppler of the supra-aortic vessels revealed a stenosis of both subclavian arteries, tighter on the left, with diffuse stiffness of the walls of the carotid arteries. Thoraco-abdominal angiography CT showed Staged calcified parietal plaques on the thoracic and abdominal aorta extending to the primary iliac arteries. PET CT was performed to assess inflammatory activity observing extensive calcifications along the wall of the aorta and primary iliac arteries.

All those arguments were compatible with the diagnosis of TAKAYASU arteritis. The patient was put under furosemide and immunosuppressive treatment (Methotrexate, corticosteroids), with a good clinical and biological evolution after a 6 months follow-up.

DISCUSSION

Takayasu's arteritis is a chronic inflammatory disease of unknown etiology that involves aorta and its major branches,[1,2] including coronary arteries,[3,4] and usually affects young females in their second and third decades. It occurs worldwide and no race seems to be immune.[5] The *HLA-B5* genetic locus is linked with susceptibility[6]

The diagnosis is delayed and commonly made when organ damage has been developed.[1] Vessel wall inflammation may cause organ ischemia due to arterial stenosis or occlusion leading to clinical manifestations. It may lead to segmental stenosis, occlusion, dilatation or aneurysm formation[7,8,9] Many patients manifest for a long time with nonspecific symptoms (fatigue, malaise, weight loss, fever…). Limb claudication, absent pulses, and BP discrepancies are typical symptoms of large-vessel vasculitis. Inflammatory cell infiltration suggests that cell-mediated autoimmunity plays an important role in the pathogenesis of TA.[10] The patients often have arterial hypertension, which is frequently associated with renal artery stenosis (which was not the case for our patient). The disease can affect the cardiovascular system (cardiomyopathy, valvular disease, aortic root disease, coronary disease or constrictive pericarditis).

Our case highlights the need for cardiac review in patients with TA, in this sense, it illustrates how, as a consequence of a continuous increased afterload due to severe hypertension, severe LV hypertrophy may develop.

The laboratory data are typical for generalized inflammation (slight leukocytosis and mild anemia, elevation of the C-reactive protein, thrombocytosis and elevation of erythrocyte sedimentation), and are necessary for assessing disease activity. Other laboratory data (antinuclear antibodies, rheumatoid factor, antineutrophil cytoplasmic antibodies, etc.) are very often within normal limits.[5]

Radiographic methods can also be useful in diagnosing TA. Arteriography is the standard for detecting vessel disease. Color Doppler sonography can show stenosis of the arteries (as in our case).[7,8] Transthoracic echocardiography can reveal cardiac disorders (LV hypertrophy in our case). Currently, magnetic resonance

imaging (MR) is a very useful non- invasive examination. It reveals vessel wall oedema in 94% of the cases.[11] PET/CT scan with 18F-fluorodeoxy- glukose (FDG) is a new method able to reveal deposits of inflammation in vessel walls (as in our case).

The American College of Rheumatology (ACR) provides criteria classifying patients with TA. They are not met in the early stages of the disease. It requires three of the following six criteria: age at the disease onset < 40 year, claudication extremities, decreased brachial artery pressure, blood pressure difference > 10 mmHg, a bruit over the subclavian arteries or aorta and abnormal arteriogram (arteriographic evidence of narrowing or occlusion of the entire aorta, its primary branches, or large arteries in the proximal upper or lower extremities).[12] The presence of three or more of these six criteria demonstrates a sensitivity of 90.5% and a specificity of 97.8%.

Corticosteroids in high doses are the primary treatment recommended for TA (as for our patient). Cytotoxic therapy is recommended for patients that are resistant to glucocorticoid treatment. The overwhelming majority of experience is with the use of methotrexate (MTX) (As for our patient). Anti-TNF therapy may bea useful adjunct to glucocorticoids.[12] Those treatments, with heart failure therapy, allow the boost of cardiac remodeling, prevent LV systolic and diastolic function deterioration, and improve quality of life and symptoms, although cardiac involvement in TA carries a worse prognosis.

Surgical or percutaneous transluminal angioplasty therapy remains an alternative for the revascularization of stenosed or occluded vessels.

In conclusion, we describe a young woman with an LV hypertrophy developed due to a severe hypertension related to Takayasu's arteritis whose diagnosis was delayed and not suspected because of falsely normal BP due to arterial stenoses.

CONCLUSION

Our case report shows the need to bear Takayasu arteritis in mind in our country as well, especially in young women, describing a our patient with a LV hypertrophy developed due to severe hypertension whose diagnosis was delayed and not suspected because of falsely normal BP due to arterial stenoses. Cardiac involvement in TA carries a bad prognosis. Medical therapy using corticosteroids and immunosuppressive treatments allows a remarkable amelioration of prognosis, symptoms and quality of life.

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