

Case Report

Minor Stroke as First Manifestation of Disease in a 60 Years Old Woman Diagnosed with Takayasu Arteritis

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- Doppler ultrasound

Abstract

Takayasu arteritis usually affects young women between 10 and 40 years old, while is uncommon diagnosis after 50 years old. Symptoms at onset are often subacute and aspecific and this can be the reason for delay in diagnosis. Less frequently the first manifestation of disease can be a stroke or a transient ischaemic attack. We report here a rare case of a woman with a past medical history of tuberculosis infection, diagnosed with Takayasu arteritis at the age of 61 years old, with a minor stroke as first manifestation of disease. She presented to the emergency department for a sudden onset episode of facial weakness and difficulty with speech regressed in about 10 minutes. She was admitted in Stroke Unit where a Colour Doppler ultrasonography of the sovra-aortic branches was performed and it showed an occlusion from its origin of the right common carotid artery and severe thickening and luminal narrowing of left CCA wall with ipsilateral ICA occlusion from its beginning. Antiplatelet and high dose statin was introduced, she was discharged from Stroke Unit after 3 days and a Rheumatologic consultation and follow up was arranged. Past medical history of the patient was interesting as she referred a pulmonary and cutaneous Mycobacterium tuberculosis infection when she was 12 and 22 years old. Relationship between Tuberculosis infection, both latent and active, and Takayasu arteritis is suggested in many studies but so far no causal relationship between the two conditions could be established. New studies should be done in the future in order to understand mechanisms at the basis of this relationship. At the moment, a practical advice could be tuberculosis infection screening for TAK patients before initiating immunosuppressive therapies, especially in high prevalence TB infection countries.

INTRODUCTION

Takayasu arteritis (TAK) is a large vessel vasculitis which affects aorta and sovra-aortic primary branches. Incidence is about 3 cases over a million in Europe and etiology is still unknown. It affects in about 80% of cases young women usually between 10 and 40 years old [1] and diagnosis after 50 years old is uncommon. TAK has often a sub-acute onset and this can be the cause of delay in diagnosis. In the early stage of the disease patients present usually a specific symptom as weight loss, fatigue, arthralgia and myalgia or low-grade fever [2,3]. A common feature of the disease is asymmetric reduction or absence of arterial pulses, often of the radial artery, which lead to a reduced blood pressure usually in one arm. Hypertension can be a feature as well, caused by renal arteries narrowing.

Later can be observed limb claudication, syncope related to the subclavian steal syndrome, and sometimes strokes, caused by hemodynamic impairment or thromboembolic mechanism in carotid or vertebral arteries stenosis [4-6]. Studies reported that about 10-20% of patients with TAK experienced an ischemic stroke or a TIA [7]. Diagnosis of TAK is often made in a later stage of the disease, as we said before, and is based upon suggestive clinical features and imaging of aorta and sovra aortic branches (Doppler ultrasound, Magnetic resonance angiography

and computed tomography angiography, Positron Emission Tomography). No diagnostic laboratory tests are available for diagnosis, but CRP and ESR, as markers of systemic inflammatory process, are often increased, even if normal inflammatory markers values should not deter TAK diagnosis.

CASE PRESENTATION

A 61 years old female with arterial hypertension treated with an ACE inhibitor, and a previous pulmonary (1970) and cutaneous (1980) tuberculosis (she has no medical document about it), presented to the emergency department complaining a sudden onset episode of facial weakness and difficulty with speech spontaneously regressed in about ten minutes. Patient underwent a Computed Tomography (CT) scan negative for acute vascular lesions and an EKG that was normal. A Stroke Unit consultation was request.

On presentation, she was alert, oriented and collaborative, and no focal neurologic signs were found on physical examination. Blood pressure was 180/77 mmHg at the right arm and 135/70 at the left arm, radial pulse was stronger at the right arm than the left one. She said her pulse has always been different between the two arms but she never underwent any medical investigation for that. Blood tests showed mild alteration of inflammatory

markers: RPC was 1,51 mg/dl and ESR was 38 seconds. Complete blood count, PT, renal and liver function were normal. High cholesterol levels were found: total cholesterol was 255 mg/dl, LDL 182 mg/dl, HDL 55 mg/dl, VLDL 18 mg/dl, Apolipoprotein B was 117 mg/dl, Apolipoprotein A1 was 155 mg/dl with a ApoB-ApoA1 ratio of 0,75 mg/dl. Lpa was not increased: 28 mg/dl. A Colour Doppler ultrasonography of the sovra-aortic branches was performed and it showed an occlusion from its origin of the right common carotid artery (CCA), with a partial recanalization of ipsilateral internal carotid artery (ICA) from the external carotid artery (ECA). Severe thickening and luminal narrowing of left CCA wall was reported as well with an ipsilateral ICA occlusion from its beginning. All those features were confirmed after a Computed tomography angiogram (CTA). Transcranial colour Doppler Ultrasound (TCCD) reported normal flow and velocity on the middle cerebral artery (MCA) and on the anterior cerebral artery (ACA) bilaterally, with very high velocities on the posterior cerebral arteries (PCA) and activation of both communicant posterior artery (PCoA). Cardiac ultrasound didn't show pathological features. A brain magnetic resonance was performed showing a mild right insular restriction of diffusion at DWI with a FLAIR hyper intensity in the same region consistent for a subacute ischemic area. Antiplatelet and high dose statin therapy was introduced with Acetylsalicylic acid (ASA) 300 mg and atorvastatin 80 mg. Patient underwent an aortic CTA as well with reporting wall thickening of ascendant aorta and arch, and a narrowed lumen of the discendent aorta until renal arteries. Patient was discharged from Stroke Unit after 3 days and a Rheumatologic consultation and a Positron Emission Tomography (PET) were arranged. PET didn't show features surely ascribable to a large vessels vasculitis. After one month patient was asymptomatic and she was prescribed to continue ASA 100 mg per day and high dose statin (Figures 1-5).

DISCUSSION

Ischaemic stroke is a not rare event in patients with TAK [8], but it is not frequent as first manifestation of disease. In literature, anyway, some cases are reported of stroke as first manifestation of TAK in young patient, but no case are reported as first manifestation in patients older than 60 years [9]. In literature a lot of major stroke are reported, as we said before, mainly in young patients, with internal carotid artery or middle cerebral artery involvement resulting in big ischemic lesion at CT scan and severe symptoms. Our patient had although a minor stroke with mild and transitory neurological symptoms. She had no significant symptoms of TAK until she was 61 and that's why she never had diagnosis before. She only occasionally complained arthralgia and myalgia, not surely attributable to TAK. She presumably had an indolent course of disease, and chronic arterial wall alterations lead to collateral circles outgrowth. Lot of literature can be also found about relationship between TAK and tuberculosis infection [10,11]. TAK is indeed common in high incidence areas for tuberculosis, and both disease share the pathology of granulomatous lesions [12]. Many authors suggested a latent *Mycobacterium tuberculosis* infection in patient developing TAK, and studies has been conducted using tuberculin test, Quantiferon-TB and purified protein derivative (PPD) [13]. A systematic review about association between TAK and latent or active tuberculosis infection has been published in

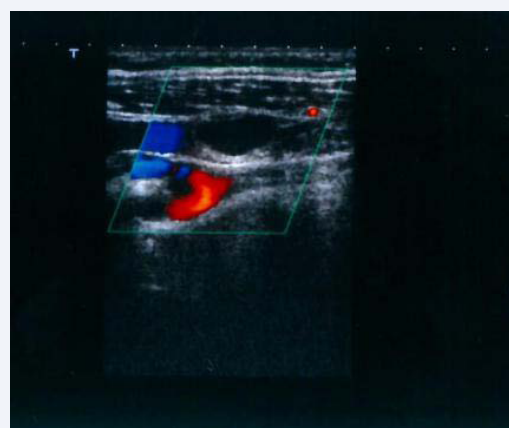


Figure 1 Right common carotid occlusion with distal internal carotid rehabilitation from the external right carotid (EchoDoppler ultrasound).



Figure 2 Left common carotid artery wall thickening (Echo-Doppler ultrasound).

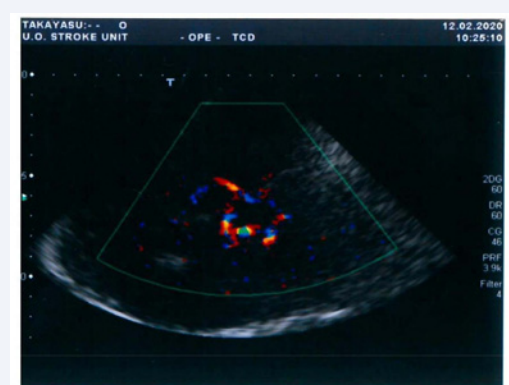


Figure 3 Transcranial Doppler ultrasound showing communicant posterior arteries activation.

2019, and authors found 8 studies assessing latent tuberculosis infection and its relationship with TAK. In those studies the most commonly used method to detect *Mycobacterium tuberculosis* was PPD, unless the cut-off point for positivity was variable.

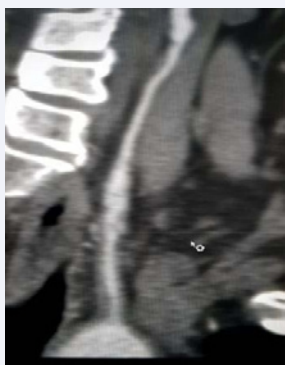


Figure 4 Left common arterial carotid (Computed Tomography angiogram).

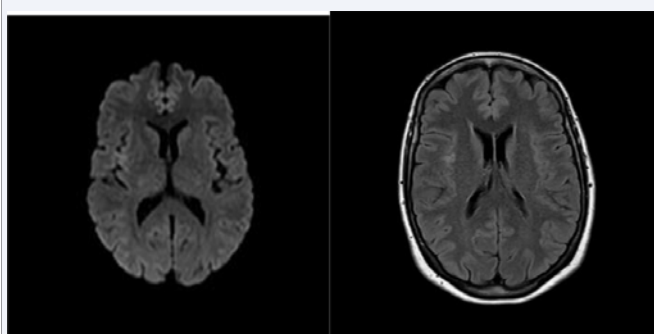


Figure 5 Right insular mild diffusion restriction (left) and mild hyperintensity at FLAIR (right) in the same region (MRI).

Prevalence of PPD positiveness ranged from 20 to 82%. Different results were found even in studies assessing arterial biopsy or autopsy. Those conflicting data brought authors to conclude that, although many studies show a high prevalence, no causal relationship between latent tuberculosis infection and TAK could be established [14]. Our case report represents a new proof of a possible connection between the two diseases, even if many efforts should be done in the future in order to understand mechanisms at the basis of this relationship. A practical advice, as suggested in Pedreira's review, could be tuberculosis infection screening for TAK patients before initiating immunosuppressive therapies, especially in high prevalence TB infection countries.

CONCLUSION

This is a rare case of a minor stroke as first manifestation of TAK vasculitis in a woman older than 60 years. When she was 20 years old she had tuberculosis pulmonary and, ten years later, cutaneous infection; a possible latent tuberculosis infection

could explain TAK development since a relationship between the two conditions is reported in many studies. More trials should be done, anyway, in order to confirm this association and to understand mechanisms below.

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