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Cerebral Embolic Activity in a Patient during Acute Crisis of Takayasu's Arteritis

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Key Words

Takayasu's arteritis · Transcranial Doppler · Embolic activity · Cerebral ischemia

Abstract

Takayasu's arteritis is a disease that affects large vessels and may cause neurological symptoms either by stenoses/occlusions or embolisms from vessels with an inflammatory process. Transcranial Doppler (TCD) ultrasound can provide useful information for diagnosis and monitoring during the active phase of the disease. Cerebral embolic signals can be detected by TCD and have been considered a risk factor for vascular events. We report a patient in whom TCD ultrasound was used to monitor cerebral embolic signals during the active phase of the disease. This case report suggests that embolic activity in Takayasu's arteritis may represent disease activity, and its monitoring may be useful for evaluating the response to therapy.

Introduction

Takayasu's arteritis (TA) is a chronic inflammatory disease of unknown etiology which can affect the aorta and its branches, including the pulmonary and coronary arteries [1, 2]. The main neurological features of TA are headache, visual disturbance, seizure, transient ischemic attack, cerebral infarction or hemorrhage, and syncope [3]. Digital angiography has been considered the procedure of choice to characterize the involvement of vessels in terms of lesion extension along the vessel and severity of arterial narrowing, despite other imaging modalities such as computerized tomography, magnetic resonance and ultrasound with Doppler studies which also provide clinically important information [2].

Vascular signs and symptoms of TA originate either by arterial stenoses/occlusions or embolisms from vessels with an inflammatory process. Cerebral embolic signals (ES)

have been considered as a risk factor for vascular events and may be monitored by transcranial Doppler (TCD) ultrasound [4]. We report a patient in the active phase of TA in whom TCD ultrasonography was used to monitor cerebral embolization during treatment.

Case Report

A 46-year-old female patient with a previous diagnosis of TA (4 years prior), who had been treated with immunosuppressants, presented with headache, transient episodes of left leg paresthesia, and vertigo. These episodes lasted 45 min, and her neurological examination was normal at admission. Laboratory findings such as erythrocyte sedimentation rate (ESR: 99 mm/h) and C-reactive protein (7.73 mg/l) suggested active phase of the disease. Magnetic resonance imaging showed hyperintense signals in T₂ and FLAIR sequences in the left frontal and occipital lobes without diffusion restriction (fig. 1). Serial TCD ultrasonography examinations of the intracranial vessels were performed before and after the TA treatment. The first TCD ultrasonography, performed 2 days before the treatment, revealed frequent cerebral emboli in both sides of the anterior and posterior intracranial circulation vessels. Echocardiography did not disclose a cardiac source of the emboli. Computerized tomography with angiography revealed diffuse aortic and bilateral carotid thickness. Treatment with corticosteroids for 5 days was initiated. Post-treatment TCD ultrasonography examination showed no detection of emboli. The patient had partial improvement of her symptoms after treatment.

Discussion

Transient ischemic attacks and stroke may be complications of TA due to the occurrence of the hemodynamic mechanism associated with stenosis/occlusion in the cerebral arteries and/or embolic material originating from the vessel region with inflammation [4]. In TA, the inflammation affects the aorta and extends into its branches [2, 3, 5]. During the acute phase, inflammatory infiltrates usually consist of lymphocytes, plasma cells, and giant cells and involve all layers of the arterial wall. Thrombi are frequently found distal to the sites of inflammatory changes. During the chronic phase, fibrous tissue replaces the damaged intima, media, and adventitia, and arterial stenoses may develop [3].

In a recent paper, Kumral et al. [4] studied 18 patients with TA and compared them with 100 age-matched healthy controls. The middle cerebral arteries were monitored by TCD ultrasound for at least 30 min. ES were present in 22% of the patients. ES were found in 30% of the patients with a higher ESR. Interestingly, 2 out of 3 patients (67%) who did not receive any treatment had ES, while only 2 out of 15 patients (13%) who received immunosuppressive and anticoagulant therapy before TCD ultrasonography monitoring had ES. The authors suggest that ES frequency was higher during the active phase of the disease, which was followed by ESR assessment, than during the silent chronic phase.

Our patient presented with clinical and laboratory findings indicative of disease activity. TCD ultrasonography revealed persistent cerebral embolic activity prior to treatment; however, after immunosuppressive therapy, no emboli were detected by TCD ultrasonography in the cerebral arteries. This fact reinforces the notion that embolic activity in TA may represent disease activity, and its monitoring may be useful for evaluating the response to therapy.

Disclosure Statement

The authors have no conflicts of interest.

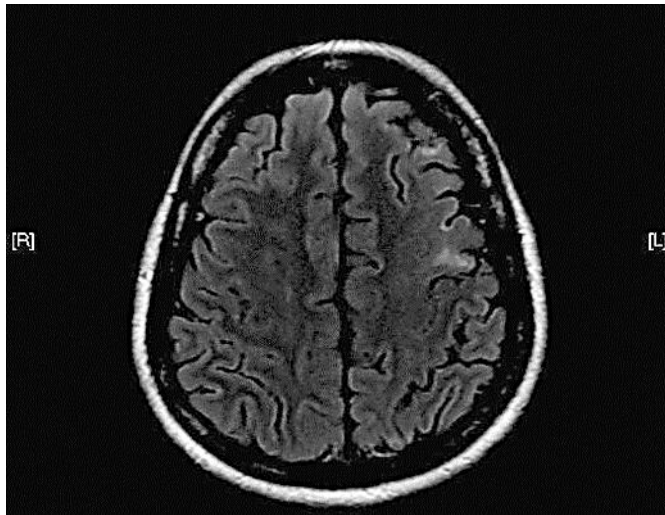


Fig. 1. MRI FLAIR sequence showing hyperintense signals in the left frontal and occipital lobes.

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