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Case report

The role of ultrasound in the diagnosis of temporal arteritis

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ABSTRACT

Temporal arteritis (TA), also known as giant cell arteritis, is a chronic vasculitis of medium and large-sized blood vessels, in particular the main cervical branches of the aorta, with particular affinity to the temporal arteries and eye-supplying arteries. Temporal artery biopsy is still a gold standard for diagnosis, however in recent years colour duplex ultrasound examination has been proposed as a useful diagnostic screening tool in cases of TA suspicion. We report three cases of TA in which the ultrasonographical examination of the temporal arteries had a decisive role in the diagnosis.

1. Introduction

Temporal arteritis (TA), also known as giant cell arteritis, is a chronic vasculitis of medium and large-sized blood vessels, in particular the main cervical branches of the aorta, with particular affinity to the temporal arteries and eye-supplying arteries [1]. The involvement of the vertebral arteries, carotid arteries, the aorta itself and coronary arteries is less common [2].

The disease occurs in all cases in patients older than 50 years, the mean age at diagnosis being approximately 72 years [3].

Disease susceptibility has been associated with European descent, the highest incidence being found in Scandinavian countries and among Americans of Scandinavian descent [4,5].

The most feared complication of TA is visual loss, however in rare cases, stroke can occur (3–7% of cases) and is the leading cause of death in patients with TA [6,7]. Cerebral ischaemic events in patients with TA are mainly due to the involvement of the extradural vertebral and carotid arteries rather than to the vasculitic involvement of the intracranial vessels [6,8].

For the purpose of differentiating TA from other forms of vasculitis, the American College of Rheumatology formulated five classification criteria for TA: age ≥50 years at onset, localised headache of new onset, tenderness or decreased pulse of the temporal artery, erythrocyte sedimentation rate > 50 mm/h and biopsy revealing a necrotising arteritis. The presence of three of these five criteria is associated with 94% sensitivity and 91% specificity for the diagnosis of TA [9,10].

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Temporal artery biopsy is still a gold standard for diagnosis, however in recent years colour duplex ultrasound examination has been proposed as a useful diagnostic screening tool in cases of TA suspicion [6,11,12].

We report three cases of TA in which the ultrasonographical examination of the temporal arteries had an important role in the diagnosis.

The examinations were performed by the same skilled investigator using a Siemens Acuson Antares ultrasound device with a VFX13-5 MHz linear transducer. The B-mode, Colour mode and duplex settings were adjusted for best assessment of the vessels. The temporal artery trunk, frontal and parietal branches were scanned in the axial and longitudinal sections. The latter section was used to detect stenoses. Stenosis was considered to be present if the flow velocity was two times higher comparing to the velocity measured in the area before stenosis.

2. Case reports

2.1. *Case 1*

An 80-year-old woman with negative medical history was admitted with a 1-month history of bitemporal headache, jaw claudication, diplopia and progressively decreasing visual acuity (left > right) that had appeared one week before. Clinical examination revealed necrotic cutaneous lesions in the temporal and frontal region of the head and induration on palpation of the temporal arteries. Neurological examination evidenced decreased visual acuity bilaterally (0.1 left eye, 0.6 right eye) and right-sided abducens nerve palsy. Laboratory analysis was relevant for increased erythrocyte sedimentation rate (ESR), 95/1 hour, thrombocytosis (507000 mm^{-3}), positive C reactive protein, elevated fibrinogen level (682 mg/dl) and mild mixed dyslipidaemia. Cerebral MRI revealed only mild cerebral atrophy, lacunary infarctions and leukoaraiosis. Duplex ultrasound examination of the cervical arteries revealed no pathological lesions. Duplex examination of the temporal arteries described the presence of the hypoechogenic halo (the halo thickness was between 0.6 and 0.9 mm) sign in both frontal and parietal branches of the temporal arteries (Fig. 1). Based on this workup the diagnosis of temporal arteritis was established without biopsy and high dose corticosteroid treatment was initiated (1 mg/kg/day prednison) followed by prednisone tapering. The outcome was favourable excepting the left-sided visual impairment.

2.2. *Case 2*

A 65-year-old woman with medical history relevant only for osteoporosis was admitted with a 2-month history of generalised headache, jaw claudication, pain and stiffness in the neck, shoulder and hip girdles and weight loss (3 kg in 1 month). Clinical examination revealed tender and thickened temporal arteries bilaterally, with right predominance, decreased pulse at this level and tenderness at the level of cervical spine and shoulder girdles. The neurological and ophthalmological examinations were normal. The laboratory workup was relevant for elevated ESR (76/1 hour) and mild normochromic anaemia. Duplex ultrasound examination of the temporal arteries revealed a hypoechoic halo sign in all branches of the temporal arteries (halo thickness between 0.3 and 1.1 m) (Fig. 2) and stenosis at the level of the left temporal artery trunk. Based on these findings, the diagnosis of temporal arteritis was established and treatment with corticosteroids was initiated (24 mg Medrol/day). The outcome was favourable, 2 weeks after the initiation of the corticotherapy the patient was asymptomatic.

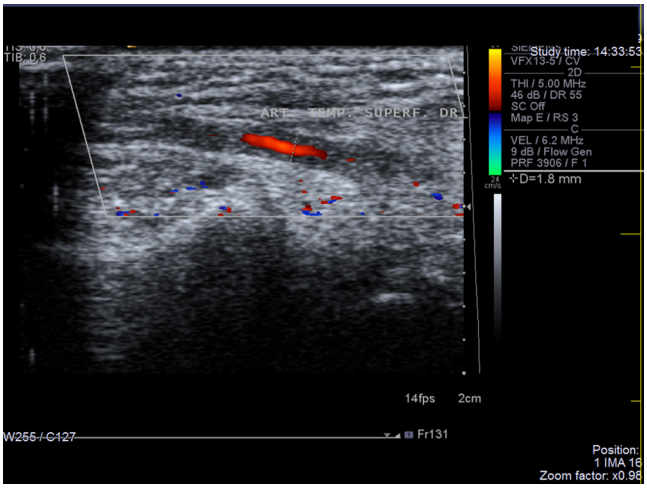

Fig. 1 – Case 1. Temporal arteritis in a 80-year-old woman. Colour duplex ultrasound examination of the temporal artery, longitudinal section, showing pronounced hypoechoic mural thickening ('halo sign').

2.3. *Case 3*

A 72-year-old hypertensive woman, without any other relevant findings in her medical history, was admitted for sudden onset vertigo, disequilibrium, nausea and vomiting, hiccups presented on awakening. Clinical examination was relevant for higher blood pressure values (156/100 mmHg). Neurological examination revealed a clinical picture suggestive of a right-sided Wallenberg syndrome. Cerebral MRI described a right-sided dorsal lateral medullary infarction. The laboratory workup was relevant only for thrombocytosis (667000 mm^{-3}); ESR, PCR and Fibrinogen were not performed. Cervical duplex ultrasound examination evidenced no stenotic lesions at the level of the carotid arteries and a significant hypoechoic mural thickening of the right vertebral artery in V1 and V2 level (the maximal thickness was 3.6 mm) (Fig. 3a). The first impression of the ultrasonographer was that this was a vertebral artery dissection, but the ultrasonographical examination of the temporal arteries revealed the same hypoechoic mural thickening (between 0.7 and 1.1 mm), that was suggestive for arteritis (Fig. 3b). Temporal artery biopsy revealed a granulomatous process with multinuclear giant cells and confirmed the diagnosis of temporal arteritis. The patient was treated with high dose corticosteroids (16 mg Dexamethasone/day, 10 days), followed by prednisone tapering, antiplatelets and antihypertensive medication with good outcome.