Bilateral vertebral artery occlusion without stroke secondary to giant cell arteritis

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An 84-year-old male patient was admitted to our department because of sudden vision loss in the left eye. He also complained of general malaise, headache, jaw claudication, dizziness and weight loss. The ophthal-mological examination revealed significantly decreased visual acuity in the left eye (perceived hand motion), the optic disc was pale and swollen, without haemorrhages, the arteries were narrowed.

The neurological examination revealed the absence of deep tendon reflexes in the lower extremities, mild postural instability and wide based gait. Brain computed tomography (CT) scan indicated moderate cerebral and cerebellar atrophy. The duplex ultrasound (US) examination showed at the level of the carotid bifurcations mild atherosclerotic changes and a dark area ("halo sign") (Figure 1) that ran circumferentially around the vascular lumen of the V1 and V2 segment of the vertebral arteries (VA), with a diameter of 1.0–1.5 mm and indirect signs suggestive of distal occlusion. The halo sign was absent at the level of superficial temporal arteries. The CT angiography of the cervical ar-

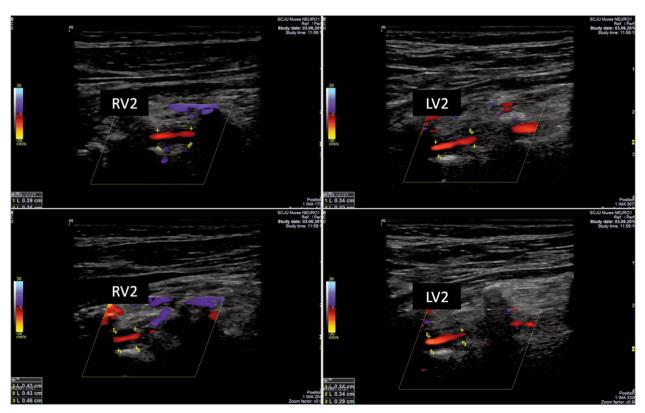


FIGURE 1. Duplex ultrasound examination. Hypoechoic "halo" sign in both vertebral arteries (RV2- right vertebral artery V2 segment, LV2-left vertebral artery V2 segment)

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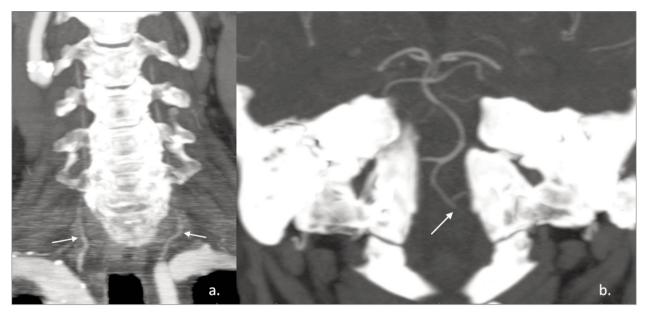


FIGURE 2. CT angiography, coronal MIP (maximum intensity projection) images: significant narrowing of both vertebral arteries (white arrow, a.); occlusion of both vertebral arteries in the in the V3 segment, reopacified just proximal to PICA (white arrow, b.)

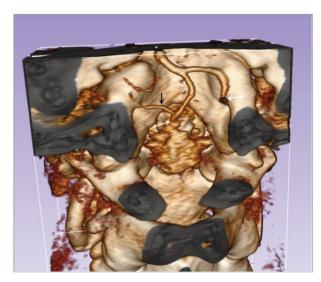


FIGURE 3. 3D reconstruction CT angiography. Occlusion of both vertebral arteries in the V3 segment (white arrows), reopacified proximal to PICA (black arrow)

teries revealed significant narrowing of both VA from the origin and bilateral occlusion at the V3 segment. The V4 segment was reopacified proximal to posterior inferior cerebellar artery (PICA) (Figure 2, 3).

Laboratory investigations revealed elevated erythrocyte sedimentation rate (80 mm/hr) and C-reactive protein level, without any other relevant changes in

the haematology and biochemistry workup. The patient fulfilled 3 of 5 classification criteria for giant cell arteritis (GCA). High dose steroid therapy was initiated with 1 g methylprednisolone, once a day for three days, followed by oral steroid treatment with a starting dose of 48 mg of methylprednisolone, followed by tapering.

The patient's visual acuity slightly improved after one week of treatment (counting fingers at 10 cm). The constitutional symptoms improved significantly. When discharged, the patient was on oral steroid, and empirically, because of high stroke risk dual antiplatelet therapy, with 100 mg of aspirin and 75 mg of clopidogrel was initiated. The neurological status of the patient was stable at the three-month and six-month follow-up.

VA involvement is not unusual in GCA, but bilateral vertebral artery occlusion (BVAO) secondary to this affection is a very rare morphological entity and only few cases have been published in the literature. Most of the published cases presented with acute stroke and the mortality rate at three months was $80\%^2$.

US examination has become an important tool in the diagnosis of GCA, revealing the suggestive hypoechoic arterial wall thickening. According to several publications the diagnosis of GCA may be established in patients with typical clinical signs and the presence of halo on US, without biopsy.^{3,4}

Spared temporal arteries may be present in 40% of patients with GCA. In such cases, the presence of a halo sign in other large arteries, such as VA, may be the main clue for the diagnosis of GCA.^{4,5}

The rarity of this case lies in the reasonably good outcome, with slightly improved visual acuity and no further neurological complications at the three-month and six-month follow-up and the absence of temporal artery involvement, despite the severe VA affection with bilateral occlusion.

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