Giant cell arteritis: A reversible cause of oculomotor nerve palsy

Artérite à cellule géante (maladie de Horton) : une cause réversible de paralysie oculomotrice

Case report

A 83-year-old female patient was hospitalized in November 2012 because of headaches, unilateral red eye and decreased visual acuity. She had previously experienced for 10 months asthenia, anorexia and 9 kg weight loss. Her past medical history was significant for hypertension, Raynaud's phenomenon and Alzheimer disease.

Clinical examination revealed an afebrile patient with stable hemodynamic parameters and normal cardiovascular auscultation. She had pupil-sparing unilateral third nerve palsy, with ptosis and adduction palsy of the right eye (figure 1). Testing of other cranial nerve was normal. Temporal and mandibular arteries were pulsatile but hardened. No murmur was found on subclavian or humeral arteries.

Biological revealed hypochromic microcytic anemia (10.5 g/dl), with elevated C-reactive protein level (46.2 mg/l), erythrocyte sedimentation rate (56 mm/h) and platelet count (513,000/μl). A cerebral angiography was performed and revealed a 5.1-mm wide and 5.8-mm long saccular aneurysm of the right T carotid (figure 1). There were no sign of complication of this aneurysm. The ophthalmologic examination confirmed the presence of partial right third nerve palsy with binocular diplopia and pupillary sparing. Hess screen test demonstrated partial impairment of the right third nerve with paresis of the right medial rectus and inferior rectus muscles. Finally, a temporal artery biopsy confirmed the diagnosis of giant cell arteritis (GCA).

The patient was treated with systemic corticosteroid therapy at 0.7 mg per kg per day dosage. After discussion with neuroradiology team, we decided to defer intravascular treatment of the aneurysm, because of its probable inflammatory etiology.

Patient outcome improved rapidly after a few days. The oculomotor third nerve palsy recovered completely and biological parameters began to normalize.

At 3 months, general examination noticed a significant improvement, with a 4-kg weight gain and the absence of any ophthalmological or neurological symptoms. Laboratory tests returned to normal. Cerebral angiographies were performed in April 2013 and January 2014, and showed stabilization of the aneurysm of the right T.

Discussion

Ophthalmologic symptoms usually occur in 20–30% of patients [1]. Arteritic anterior ischemic optic neuropathy is the most common ocular finding in GCA. Third nerve palsy is an extremely rare manifestation of CGA[2,3]. GCA can cause a third nerve palsy by a muscular necrosis of extraocular muscles[3], but also through an inflammatory aneurysm of the carotid [4]. In our case, the clinical and biological improvements after treatment with corticosteroids suggest that the intracranial aneurysm was, in part, inflammatory.

GCA may appear with highly variable clinical forms, making diagnosis difficult. We report here a case of unilateral third nerve palsy secondary to intracranial aneurysm explained by GCA that was reversible after corticosteroid therapy. In front of an intracranial aneurysm, clinician should evoke GCA and look

![Figure 1](A) Clinical observation of third nerve palsy, with right eye ptosis and right eye adduction palsy. Initial (November 2012) brain computed tomography (B) and cerebrovascular three dimensional computed tomographic angiography (C) showing saccular aneurysm of the right T carotid. (D) Follow up brain computed angiography (January 2014) showing a stability of the aneurysm (black arrow)
for the presence of elevated inflammatory markers on biological feature.

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References


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