THIRD NERVE PALSY AS THE ONLY MANIFESTATION OF OCCULT TEMPORAL ARTERITIS

PARÁLISIS DEL TERCER PAR CRANEAL COMO ÚNICA MANIFESTACIÓN DE ARTERITIS DE LA TEMPORAL OCULTA

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ABSTRACT

Case report: Two patients (80 and 67 year-old) presented with diplopia for a sudden right 3rd cranial nerve palsy without other ocular or systemic symptoms related to temporal arteritis. Erythrocyte sedimentation rate and C-reactive protein levels were normal. Subsequent biopsy of the superficial temporal artery confirmed the diagnosis of temporal arteritis.

Discussion: Patients with ocular nerve palsy could have occult temporal arteritis. Aged patients with an acute ocular ischemic lesion, without elevated erythrocyte sedimentation rate, C-reactive protein levels and systemic symptoms, should raise a high index of suspicion for temporal arteritis (Arch Soc Esp Oftalmol 2009; 84: 395-398).

Key words: Third cranial nerve palsy, occult temporal arteritis, temporal biopsy, diplopia, sedimentation rate.

RESUMEN

Caso clínico: Se describen dos pacientes (80 y 67 años) con diplopía por parálisis del tercer par craneal sin otros síntomas de afectación ocular o sistémica de arteritis de la temporal. La velocidad de sedimentación globular y la proteína C-reactiva fueron normales. La biopsia de la arteria temporal superficial confirmó el diagnóstico de arteritis de la temporal.

Discusión: Pacientes con parálisis de los nervios oculomotores podrían tener una arteritis de la temporal oculta. En los ancianos con lesiones oculares isquémicas, sin elevación de la velocidad de sedimentación, con niveles normales de proteína C-reactiva y sin síntomas sistémicos, el índice de sospecha de arteritis de la temporal debe ser alto.

Palabras clave: Parálisis del tercer par craneal, arteritis de la temporal oculta, biopsia de la temporal, diplopía, velocidad de sedimentación.
INTRODUCTION

Temporal arteritis (TA) has a prevalence of 2.3 for every 100,000 inhabitants at age 60 and of 44.7 at age 90 (1). Ophthalmological findings in TA include the anterior segment, pupil dysfunction, retinal infarct, ischemic optic neuropathy, ophthalmoparesia, cortical blindness, visual field defects, amaurosis fugax and complex visual hallucinations (2). Eye motor palsy is an infrequent clinical expression of TA (1,2). Occult arteritis of the temporal vein is defined as an ocular involvement due to TA without systemic symptoms or signs (3). This paper reports to cases of third cranial pair palsy due to occult TA without increase of the globular sedimentation velocity with pathognomonic pathological anatomy..

CASE REPORTS

Case 1

An 80-year-old woman who visited the hospital with diplopia starting seven days earlier. The diagnostic was complete palsy of the right side third cranial pair with pupil involvement. She did not refer headache, jaw weakness or stiffness in the neck and joints. Her personal history was only significant for high blood pressure, while ophthalmological history lacked interest. The exploration of ocular motility in the left eye (LE) was normal. The right eye (RE) exhibited palpebral ptosis and limitation of movement in all gaze positions except abduction (fig. 1A-C). The reaction of the pupil to light in the left eye (LE) was of 3 mm to 2 mm, while in the right eye it was slow, from 5 mm to 4 mm (fig. 1D). No afferent pupil defect. The slit lamp exploration was typical of bilateral cataracts. The ocular fundus was normal. A cerebral magnetic angi-resonance failed to detect any pathology. In the lab studies, the global sedimentation rate (VSG) was of 28 mm/h, C-reactive proteins and 0.2 mg/dl (normal, <0.8 mg/dl) and platelet amounted to 175,000/ml3. The rheumatoid factor, antinuclear antibodies, anticytoplasm neutrophile antibodies, anti-receptor antibodies of acetylcholine and syphilis serology were negative. A TA diagnostic was considered and a biopsy of the temporal artery was performed. The pathological anatomy revealed stenosis of the arterial diameter with rupture of the internal limiting membrane together with inflammatory and giant cells (fig. 1E).

Case 2

A 67-year-old male without relevant personal history, who referred that while walking he suddenly noticed double vision and slight palpebral ptosis in the RE, which became total in the following hours. He didn’t have ophthalmological antecedents, did not refer weight loss, fever, jaw weakness or diminished eyesight. Visual acuity in both eyes was 1. The extra ocular motility in the LE was normal. The RE exhibited total ptosis with gaze limitation in all positions except abduction (fig. 2A-C). Pupil motility was normal in both eyes. The intra-ocular pressure was of 16 mmHg in RE and 18 mmHg in LE. Anterior and posterior biomicroscopic studies gave normal results. The lab tests were not significant with VSG of 21 mm/h, C-protein 0.3 mg/dl and platelet amounting to 125,000/ml3. The definitive diagnostic was made after a biopsy of the temporal artery which revealed occlusion with giant cells around the internal elastic membrane, which appeared broken and folded (fig. 2D).

DISCUSSION

In the first case described above, the history and the clinical assessment were compatible with complete palsy of the third cranial pair with involvement of the pupil, while in the second patient the pupil was not involved. Neither of them had symptoms or signs of temporal arteritis and the ophthalmological involvement was the only reason for visiting the emergency services. In patients aged 60 or over, the suspicion of temporal arteritis must be high (1,2). The clinical suspicion is based on a variety of symptoms including headache, fever, jaw weakness, stiffness in the back of the head, loss of weight and asthenia. The lab tests, including VSG and a hematological study could be useful (1,2). High VSG is a well established diagnostic criteria in TA. However, several studies describe patients with VSG between 5 and 30 mm/h and a positive biopsy for TA (4, 5). A biopsy of the superficial temporal artery is considered to be the most sensitive and specific test (1,3). In suspicious cases, a
Fig. 1: palsy of the third right cranial pair. Limitation of ocular motility with pupil dilatation (A-D). Biopsy of the left superficial temporal artery (hematoxilin-eosin) exhibiting occlusion of the arterial diameter with giant cells close to rupture in the internal elastic membrane with chronic inflammatory cells (E).

Fig. 2: right palpebral ptosis with gaze limitation in all positions except abduction (A-C). Hematoxilin-eosin of the temporal artery with stenosis of the interior light and multiple giant cells.
second biopsy should be considered. Hayreh et al (3) described 18 cases of occult TA in which the patients had no sign or systemic symptom of TA and the values of VSG and reactive C protein were relatively low. The patients described above attended the emergency services due to diplopia as the only ophthalmological clinical sign and did not exhibit any systemic signs or symptoms to consider TA. This could lead us to suspect a microvascular etiology, even more so because one of them had high blood pressure. The differential diagnostic includes compression of the nerve due to an aneurysm microvascular ischemia, inflammation, tumor, infiltration, infection, self-immune disorders, ocular migraine and temporal arteritis. The fact that all tests were negative and that temporal arteritis is a proficient simulator of other diseases led us to perform a temporal artery biopsy. Accordingly, in elderly patients TA should be suspected and other possible expressions of the disease should be analyzed. It is not infrequent to find in the medical literature (neurology, rheumatology, ophthalmology) reports that the diagnostic of TA was confirmed in the light of negative results of all other tests.

Normal VSG and reactive C-protein may indicate a more localized disease in patients with an occult TA and therefore without systemic symptoms (3). The TA diagnostic is necessary not only for preventing a disease with a high ocular morbidity but also to prevent severe systemic expressions of a treatable disease.

REFERENCES