VISUAL ACUITY LOSS, THE INITIAL SYMPTON OF A SPINAL CORD NEOPLASM

DISMINUCIÓN DE AGUDEZA VISUAL, PRIMER SÍNTOMA DE UN TUMOR MEDULAR

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ABSTRACT

Case report: A 46-year-old woman, who presented with right visual acuity loss, was found to have papilledema, and subsequently shown to have ventricular dilatation in a cerebral Magnetic Resonance Imaging (MRI) assessment. Elevated protein levels were found in the cerebrospinal fluid. Spinal MRI revealed the presence of a spinal cord neoplasm. After surgical removal of the tumor, which turned out to be a neurilemmoma, the patient’s visual acuity was restored.

Discussion: The ocular presentation and the relationship between intracranial hypertension and spinal tumors are discussed. Likewise, the importance of considering the various causes of papilledema is emphasized (Arch Soc Esp Oftalmol 2008; 83: 437-440).

Key words: Papilledema, Spinal cord neoplasms, Intracranial hypertension, neurilemmoma, hydrocephalus.

RESUMEN

Caso clínico: Se presenta el caso de una mujer de 46 años con disminución de agudeza visual en el ojo derecho y papiledema, con dilatación ventricular en la Resonancia Magnética Nuclear (RMN) cerebral. En la punción lumbar se detectó hiperproteinorraquia. La RMN medular reveló la presencia de una neoplasia de la médula espinal. La paciente recuperó la agudeza visual tras la extirpación quirúrgica del tumor, que resultó ser un neurilemmoma.

Discusión: Se comenta la presentación ocular y la fisiopatología de la hipertensión intracraneal en el tumor espinal. Asimismo se destaca la importancia del diagnóstico etiológico del papiledema.

Palabras clave: Papiledema, neoplasias de médula espinal, neurilemoma, hipertensión intracraneal, hidrocefalia.
INTRODUCTION

Brain tumors and pseudotumor cerebri are frequent causes of papilledema. Papilledemas secondary to spinal cord neoplasms are not frequent, but they have been reported (1). A description is provided below of a patient with a spinal cord neoplasm which involved papilledema and a loss of Visual Acuity (VA).

CASE REPORT

A 46 year old female reporting decreased vision in her right eye for one year prior to visit to Ophthalmology Outpatient Dept. No report of headaches, transitory darkening, or double vision episodes. No previous treatment with lithium, tetracyclines, vitamin A, or corticosteroids. No weight variation in the last year.

Neurological examination was normal. The ophthalmic test showed a corrected visual acuity of 20/25 for the right eye, and 20/20 for the left eye. The slit lamp anterior pole examination revealed no abnormalities in intraocular pressure and extrinsic and intrinsic ocular motility. The ocular fundus examination showed a discrete blurriness, and elevation of both papillas, with no hyperemia or bleeding, with a larger aedema on the nervous fibre layer.

Fig. 1: Ocular fundus image for the right eye. The papilla is elevated and slightly blurred.

Fig. 2: Humphrey 24-2 Perimetry for both eyes. The blind spot has increased, and there is a lower nasal defect for both eyes.
for the right eye (Fig. 1). The ecograph revealed no drusen on the optic nerve. Exploration of the visual field with Humphrey perimeter (Carl Zeiss Meditec, Inc., Dublin, California) programme SITA 24-2 showed an increase of the blind spot with a lower nasal defect in both eyes (Fig. 2).

The MRI showed a moderate dilatation of the supratentorial ventricular system, with no objective evidence of space-occupying injuries. After these findings, and with no contraindication, a lumbar puncture was performed to determine the cerebrospinal fluid (CSF) pressure and to ascertain the origin of hydrocephalus, which revealed a high opening pressure of 38 cm H₂O (normal values 5-18 cm H₂O); the CSF analysis showed very high protein values of 794 mg/dL (normal values 15-50 mg/dL).

An MRI was then taken of the spinal cord. A large intradural, polylobulated mass, excentric to the spinal cord was detected. The mass exerted a mass effect on the spinal cord, it was highlighted with contrast, and was located from T8 to T11, with a size of 8 x 2 cm (Fig. 3). Surgery was performed, with a laminectomy and excision of the lesion. The anatomical report confirmed a neurilemoma.

The patient recovered from visual problems within three months of surgery. Perimetry and optic nerve head were practically normal, and some residual gliosis was present. The encephalic ventricular dilatation was resolved within six months.

**DISCUSSION**

Descriptions have been provided of spinal cord neoplasms with ocular involvement only, but these are not common. Only seven cases have been found in the literature (1,2). The isolated symptoms with a slight loss of VA for the right eye and slight papilledema might have suggested pseudopapilledema by drusen. However, after checking for alteration of the visual field, and discarding optic nerve drusen with the ecography, an MRI test was prescribed, followed by a lumbar puncture, and a spinal cord MRI, which allowed diagnosis.

Spinal cord neoplasms rarely produce papilledema. Arseniy Maretsis reviewed 289 cases, and only 3 showed papilledema (1,3). When papilledema is produced 40% of cases correspond to ependimomas, with only 11% neurilemomes, also known as schwannomes (1). Symptoms usually include local pain and paresthesias (1,4). When these are present with papilledema, cephalalgia and loss of vision are more frequent (1,3).
Different mechanisms have been suggested to explain hydrocephalus with intracranial high pressure and papilledema associated with spinal cord neoplasms. Local obstruction may be present for high spinal cord neoplasms (4), whereas thoracic, lumbar, and sacrum neoplasms might be related to high protein levels in CSF. This would lead to obstruction of the drainage, and a subsequent inflammation of the leptomenynges (1-3,5). Another mechanism attributed to low spinal cord neoplasms would imply a reduction in lumbar-sacral elastic reserve, leading to a mechanical obstruction of the cerebrospinal fluid at that level, with its corresponding increase in pressure (1-4). The hyperproteic CSF and the large size of the tumor for the patient described might have compressed the epidural venous drainage. This would explain the high intracranial pressure (4).

As a conclusion, the importance of an etiological diagnosis of papilledema and the need for medular MRI must be outlined for patients suffering from papilledema, together with brain MRI with ventricular dilatation and increased protein levels in CSF, even with no medullary symptoms.

REFERENCES