INTRACRANIAL HYPERTENSION AND CRANIAL SINUS STENOSIS

HIPERTENSIÓN INTRACRANEAL Y ESTENOSIS DE LOS SENOS CRANEALES

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ABSTRACT

Clinical case: Cranial sinuses stenosis can appear in patients with idiopathic intracranial hypertension. Neuroimaging techniques revealed a right transverse sinus stenosis. As the pressure gradient between both sides of the stenosis was small and response to conservative treatment good, angioplasty was not indicated.

Discussion: Lateral sinus stenosis in patients with idiopathic intracranial hypertension is prevalent. It is not clear whether these stenoses are the origin of, or secondary to, cerebrospinal fluid pressure increases. Some cases refractory to conservative treatment may respond to angioplasty with stent placement (Arch Soc Esp Oftalmol 2008; 83: 619-622).

Key words: Idiopathic intracranial hypertension, intracranial sinus stenosis, pseudotumor cerebri.
INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a syndrome of unknown etiology which develops with increase of intracranial pressure in the absence of hydrocephalia and masses, with a normal composition of cerebrospinal fluid (CSF). The common pathogenic factor seems to be an increase of intracranial venous pressure, which would increase resistance to the absorption of CSF with the ensuing elevation of intracranial pressure (1).

Imaging tests have described stenosis of lateral cranial sinuses which increase venous pressure. The nature and relevance of this stenosis is not clear. This paper describes an IIH case associated to stenosis of the right transversal sinus.

CASE REPORT

A 35-year old male patient who visited the emergency section due to highly intense bilateral frontal parietal cephalea beginning 8 hours earlier, which did not change with Valsalva maneuvers or different positions and partially receded with analgesics. Relevant history included an IIH episode treated with oral acetazolamide and naproxen with clinical recovery and disappearance of the papilledema.

The general and neurological exploration was normal, with a body mass index of 22 Kg/m². The ophthalmological exploration revealed a corrected visual acuity of 0.7 in both eyes. Intrinsic ocular motility, exploration of the anterior segment and intra-ocular pressure (IOP) were normal.

The funduscopic exploration showed bilateral papilledema (fig. 1). Fluorescein angiography (FA) revealed bilateral peripapillary leak since early times. The nervous fiber layer study (with the fast RNFL strategy) by means of optical coherence tomography (Stratus OCT; Carl Zeiss Meditec, Dublin, CA) showed a thickening of the fiber layer in the right eye (RE) and a thickness at the high end of normality in the left eye (LE).

Orbitary echography, computerized axial tomography and automatic perimetry yielded normal results.

A lumbar punction was carried out, showing an opening pressure of 30 cm of water, with the composition and serology of CSF being normal.

The analytical coagulation and determination of coagulation and ANCAs, ANAs, PCR, FR, ASLO, thyroid hormones and anti-thyroid antibodies were negative.

After a Nuclear Magnetic Resonance (NMR) and phlebo-resonance, the presence of cranial sinus thrombosis was discarded, finding a segmentary stenosis of the right lateral sinus (figs. 2 and 3).

The patient was treated with acetazolamide and naproxen with clinical improvement being observed. After a venography (fig. 4) and manometry of cranial sinuses a long stenosis of the transverse sinus was observed, with a pressure gradient of 7 mmHg, of small size and similar to the contralateral stenosis. For this reason and considering the clinical improvement, dilatation with angioplasty and stent placement was discarded.

Fig. 1: Retinography in acute phase of both eyes showing a bilateral disc edema.
DISCUSSION

Until a short time ago, the IIH diagnostic was based on Dandy’s modified criteria (1) (Table I), which required, as image proof, a normal cranial tomography. It has been known for some time already that cranial sinus thrombosis developed with intracranial hypertension. Recently, the percentage of cases coursing with intracranial hypertension without other neurological symptoms was estimated at 37%. These cases could be confused with IIH if the adequate imaging tests are not made. In 2002, new diagnostic criteria were established, requiring a Nuclear Magnetic Resonance and phlebo-resonance in non-typical cases (1) (Table II) to discard cranial sinus thrombosis. NMR reveals stenotic injuries in the lateral venous sinuses of IIH patients with a significantly greater frequency than in healthy subjects. These stenoses, observed in up to 90% of patients with IIH (2,3), have been studied with manometry and phlebography, finding a pressure gradient at both sides with increased proximal venous pressure.

The possibility that this type of stenosis could be the cause of IIH is a very attractive proposition because it gives rise to a cure by means of dilatation with angioplasty. The authors who support this theory base their assumption on the fact that other

Table I. Modified Dandy diagnostic criteria

- Clinical symptoms of intracranial hypertension (headache, nausea, vomiting, temporary loss of vision, papillary edema)
- Absence of neurological focality signs, excepting single or bilateral paralysis of the VI cranial pair
- Increased CSF pressure with normal chemical and cytological composition
- Normal or small symmetrical ventricles, initially assessed with ventriculography and at the present time with tomography
injuries which develop obstruction of sinus flow, such as thrombosis or extrinsic compression, exhibit IIH clinical signs (1) which is resolved when the obstruction disappears. In addition, there are IIH cases efficiently treated by means of dilatation of the stenosis and placement of a stent (4).

Yet other authors postulate that stenoses appear as a result of the elevation of CSF pressure which is transmitted to the intracranial space, collapsing the least resistant venous wall. This theory is based on described cases in which stenosis disappeared after a derivation of CSF and the ensuing pressure reduction. In addition, not in all cases in which stenosis is dilated exhibit improvements (5).

Probably, either of said theories does not exclude the other. There are cases in which sinus stenosis are the cause of IIH and therefore dilatation would be the treatment of choice. Also, other cases in which an unknown initial cause increases CSF pressure and collapses the sinus walls thus producing stenosis would be more adequate for CSF derivation. At this time we do not have diagnostic data allowing us to differentiate between both conditions and thus establish the most adequate etiopathogenic treatment.

REFERENCES