Treatment of idiopathic intracranial hypertension with bariatric surgery☆,☆☆

Tratamiento de la hipertensión intracraneal idiopática con cirugía bariátrica

Dear Editor:

Idiopathic intracranial hypertension (IIH) is a disease typically manifesting in young female patients with obesity. Obesity is thought to play a central role in its pathophysiology. Treatment is classically based on weight loss, diuretics, and cerebrospinal fluid (CSF) shunting in refractory cases. Bariatric surgery has been proposed for the treatment of IIH associated with obesity, although the neurological literature includes scarce evidence on the subject. We present a case of IIH associated with obesity which was refractory to conventional treatment, and the clinical response after bariatric surgery.

Our patient was a 23-year-old woman with a history of morbid or class III obesity, (BMI: 42.7 kg/m^2 [weight: 112 kg; height: 162 cm]), dyslipidaemia, smoking, and treatment with oral contraceptives. She presented a 3-week history of oppressive frontal headache, amaurosis fugax in the right eye, binocular diplopia with bilateral paralysis of the sixth cranial nerve, and bilateral papilloedema. We performed a brain CT scan and CT angiography, with no abnormal findings. The brain MRI scan showed a dural ectasia of the optic nerves, a partially empty sella turcica, and a mild herniation of the cerebellar tonsils which we suspected was secondary to intracranial hypertension and not a type 1 Arnold-Chiari malformation (Fig. 1). Lumbar puncture opening pressure was 39 cm H_2O; CSF analysis results were normal. Visual acuity, visual field assessment, and optical coherence tomography yielded normal results. The patient was diagnosed with IIH. Treatment with oral contraceptives was suspended and treatment was started with acetazolamide at 750 mg/24 hours, as well as dietary and lifestyle changes. The patient initially presented a favourable response: migraine episodes reduced in frequency, and papilloedema and bilateral paralysis of the sixth cranial nerve disappeared.

However, her condition progressively worsened, and she had to be hospitalised twice and visit the emergency department numerous times due to refractory headache, with onset of a new bilateral papilloedema with preserved visual acuity, requiring 2 lumbar taps; the second tap revealed an opening pressure of 47 cm H_2O and resulted in no clinical improvement. She was then referred to neurosurgery for assessment of eligibility for CSF shunting. At 10 months after diagnosis, she had lost 12 kg and presented a BMI of 38.1 kg/m^2, and was therefore assessed by the endocrinology and nutrition department's high risk obesity unit; bariatric surgery was indicated and the neurosurgical treatment was postponed. She underwent a laparoscopic sleeve gastrectomy without complications. At 3 months after bariatric surgery, the patient had lost an additional 16 kg, with a BMI of 32 kg/m^2, showing clear signs of headache improvement, normal eye fundus, normal visual acuity, and improvement of the signs of intracranial hypertension in the follow-up MRI scan (Fig. 1). We therefore ruled out neurosurgical treatment. IIH resolution persisted after 12 months of follow-up.

Since 1986, several case reports and case series of IIH with obesity treated with bariatric surgery have been published, mainly in surgery journals.[1–9] A review article by Fridley et al.[10] reported a total of 62 cases (class IV evidence) in which signs and symptoms of IIH resolved in 92% of cases, with no significant complications associated with bariatric surgery. Furthermore, 11 cases have been reported in which neurosurgical shunting was unsuccessful and improvement was observed after bariatric surgery.[10,11] We did find it striking that these data have had so little impact in the field of neurology, with only 2 articles published.[8,12]

Weight loss continues to be the most effective treatment for obese patients with IIH. Its pathophysiological mechanism remains unknown. It has been hypothesised that obesity may cause an increase in intra-abdominal pressure, and secondarily intrathoracic pressure, consequently increasing central venous pressure, which would delay venous return and consequently CSF reabsorption.[9] Therefore, secretion of pro-inflammatory adipokines and such hormones as leptin by the adipose tissue is believed to play an important role.[13]

Regarding neurosurgical treatment options, we would like to point out that in cases where IIH is associated with type 1 Arnold-Chiari malformation, CSF lumboperitoneal shunt is not recommended, since it may even aggravate the condition; ventriculoperitoneal shunt is an alternative option.[14]

Despite the need for prospective and controlled trials with higher levels of evidence, those published to date suggest that bariatric surgery should be considered in patients with IIH and obesity,[15] especially before associated visual loss manifests. Furthermore, researchers suggest including IIH as a criterion for prioritising patients with obesity on bariatric surgery waiting lists, which are long in our setting. In practical terms, multidisciplinary management is recommended, with early referral of these patients to a specialised obesity unit.

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☆☆ This study was previously presented at the 12th National Congress of the Spanish Society for the Study of Obesity (SEEDO) in 2015, under the title ''Bariatric surgery in idiopathic intracranial hypertension: a case report''.

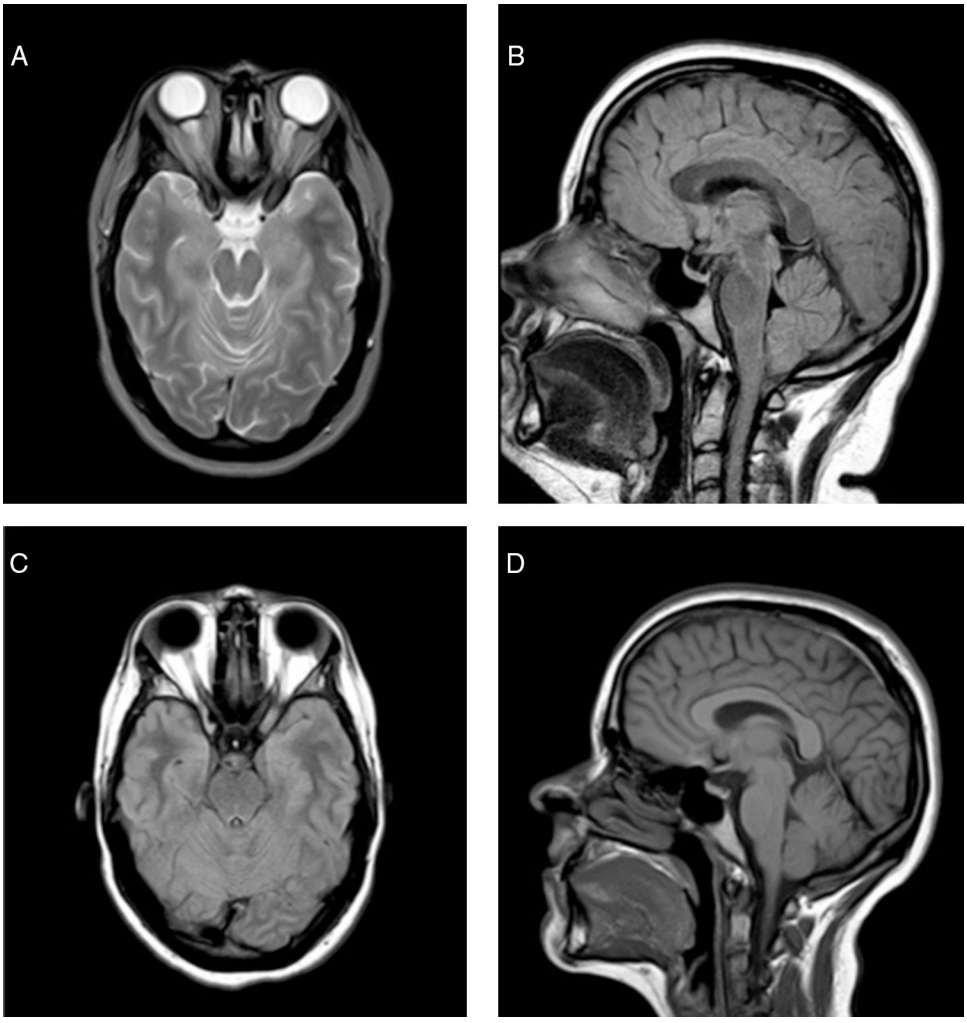

Figure 1 Baseline brain MRI: (A) Axial T2-weighted sequence, showing ectasia of the optical nerve sheath, with prominence of both optic discs with signs of IIH. (B) Sagittal FLAIR sequence, showing the partially empty sella turcica. The cerebellar tonsils are mildly herniated, reaching the level of the foramen magnum. Brain MR image obtained 6 months after bariatric surgery. (C) Axial FLAIR sequence, showing resolution of the orbital findings. (D) Sagittal T1-weighted sequence, showing slight retraction of cerebellar tonsils in comparison with the previous study.

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J.R. Pérez-Sánchez[a,*], M. Arnoriaga Rodríguez[b], F. Díaz-Otero[a], I. Bretón Lesmes[b]

[a] *Servicio de Neurología, Hospital General Universitario Gregorio Marañón, Madrid, Spain*
[b] *Servicio de Endocrinología y Nutrición, Hospital General Universitario Gregorio Marañón, Madrid, Spain*

* Corresponding author.
E-mail address: javineuro@hotmail.com
(J.R. Pérez-Sánchez).

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Temporal crescent syndrome vs arcuate scotoma☆

Síndrome de la media luna vs. escotomas arciformes

Dear Editor,

It was with great interest that we read the article by Camacho-Velasquez et al.[1] describing a case of left monocular temporal scotoma, which was attributed to temporal crescent syndrome of ischaemic origin.

The case report includes the results of the Humphrey visual field test for the left eye, which shows arcuate scotomas crossing the midline in the inferior visual field.

Temporal crescent syndrome is a visual field defect caused by lesions to the most anterior part of the primary visual cortex. The most striking feature of the syndrome is that patients exhibit monocular scotomas despite the lesion being retrochiasmatic.[2]

Visual field defects in this syndrome are located between 60° and 90° in the temporal hemifield of the eye contralateral to the lesion; they cannot therefore be detected with the Humphrey visual field test as this technique usually focuses on the central 30° of the visual field.[2]

Defects can nonetheless be seen with confrontation visual field testing (when the examination begins at the periphery); graphical representation requires a technique including the whole visual field (e.g., Goldmann perimetry).

In the case presented by Camacho-Velasquez et al., if the scotoma is monocular, as the authors state, it should be attributed to an artefact or a prechiasmatic lesion as it involves the central 30° of the visual field. On the contrary, if it is secondary to a transient ischaemic attack, a congruent visual field defect should be detected in the contralateral eye.

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J.M. Losada Domingo*, I. Caballero Romero

Servicio de Neurología, Hospital Universitario Cruces, Barakaldo, Bizkaia, Spain

* Corresponding author.
E-mail address: josemaria.losadadomingo@osakidetza.eus
(J.M. Losada Domingo).

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