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LETTERS TO THE EDITOR

CSF fistula in a woman with a history of idiopathic intracranial hypertension

Fístula de LCR en una mujer con antecedente de hipertensión intracraneal idiopática

Dear Editor:

We report the case of a 25-year-old gypsy woman with a history of morbid obesity and idiopathic intracranial hypertension (IIH), diagnosed 10 years earlier. She had never been operated on and had no relevant family history. The diagnosis of IIH had been reached at our centre after a lumbar puncture showed an elevated cerebrospinal fluid (CSF) pressure (49cm in a water column), and after excluding other causes of intracranial hypertension using brain imaging (conventional magnetic resonance imaging [MRI] and angio-MRI) and laboratory tests, which were normal. She was treated with acetazolamide with a good clinical response during the first year (normal visual field and without headache). Subsequently, the patient voluntarily discontinued monitoring and treatment.

Two weeks before admission, the patient presented headache that started in the occipital region and radiated to the frontotemporal region. The pain was pulsating and was not accompanied by nausea, vomiting or photophonophobia. She also presented dizziness, and both pain and dizziness became worse with the Valsalva manoeuvre and when rising from recumbence. The patient also reported episodes of fever of up to 38.5°C and rhinorrhea in the last 2 months, which her family doctor had attributed to a sinus infection and treated with antibiotics (amoxicillin-clavulanate) and aerosols. Physical examination did not show any data of interest, except for a clear fluid excreted by the left nostril. The neurological examination was normal and showed no meningeal signs. Examination of the fundus highlighted signs of chronic bilateral papilloedema. Laboratory tests indicated no significant data in haematological, biochemical and endocrine tests. The fluid excreted by the patient through the left nostril was also analysed by reactive strips and in the laboratory, revealing glucose at 53mg/dl and total protein at 47mg/dl, without formed elements, compatible with CSF. A subsequent cranial computed tomography scan identified a discontinuity in the left cribriform plate of about 3-4mm, which extended to affect the lateral lamella. These findings were consistent with a CSF fistula (fig. 1). Cerebral MRI was not performed due to claustrophobia. The patient underwent bilateral nasal endoscopic closure and her subsequent evolution was satisfactory.

idiopathic intracranial hypertension, also known as benign intracranial hypertension or "pseudotumor cerebri", is a disease characterised by increased CSF pressure, with no clinical, radiological or laboratory evidence of intracranial condition.[1] It is often associated with female gender and obesity.[2] It is generally well controlled with medical treatment (acetazolamide), but in some cases it is necessary to resort to surgical treatments. These patients require close clinical follow-up since recurrence is frequent.[3] However, CSF fistula as a complication of IIH with long evolution is less common.[4,5] Multiple causal mechanisms have been proposed: increased CSF flow may break the arachnoid surrounding the olfactory nerve, thus accessing the cribriform plate;[6] it has also been speculated that high CSF pressure may erode the sphenoid at the level of the sella turcica, given the association of IIH with empty sella turcica syndrome.[7] The most frequent anatomical location of CSF fistulas by IIH is the cribriform plate,[3] as in our case. Diagnosis can be difficult and the assumption that headache in patients with a history of IIH is solely due to this disease may lead to diagnostic errors. Headache is a symptom that

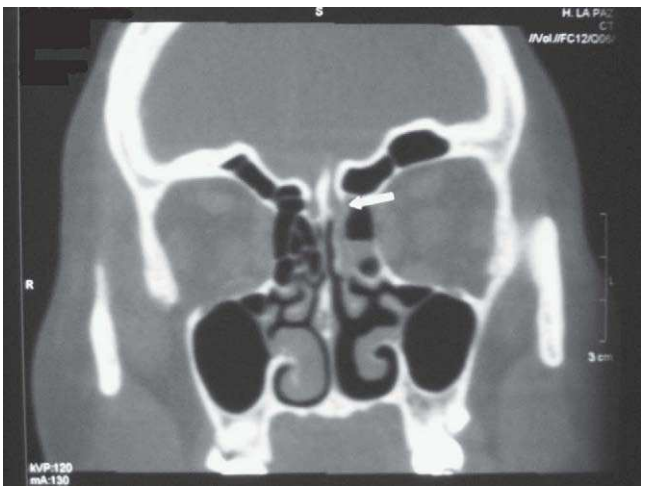

Figure 1 Cerebrospinal fluid fistula in left cribriform plate (arrow).

often appears in both IIH and in CSF fistulas. Typically, the headache associated with low CSF pressure worsens within 15min after sitting or standing and is often accompanied by neck stiffness, tinnitus, hearing loss, photophobia and nausea.[8] In our patient, we suspected that the headache was due to liquoral hypotension, in addition to liquorrhea, because the pain and dizziness worsened with standing. Lumbar puncture was not performed for the initial diagnostic suspicion, as it might have worsened the headache from intracranial hypotension.

In conclusion, IIH requires close monitoring because complications can occur. A CSF fistula, although rare, is an adverse event that requires early diagnosis and surgical treatment to prevent the development of meningitis or pneumoencephaly.[9]

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Corectopia as a neuro-ophtalmological sign of polyradiculopathy

Corectopia como manifestación neurooftálmica en un caso de polirradiculopatía

Dear Editor:

Corectopia is the manifestation of an eccentric, oval pupil. It is primarily described in mesencephalic pretectal lesions.[1] This alteration is the result of an incomplete injury of the pupillary fibres, which produces a selective inhibition of the iris sphincter tone.[1] We present the case of a patient with inflammatory demyelinating polyradiculopathy who presented corectopia.

The patient was a 32-year-old Caucasian male, with no relevant medical history, who attended the emergency service due to back pain of 15 days' evolution. He also reported intense pain in both sural regions and paresthesia in the soles of the feet and palms of the hands. The neuro-ophthalmic exploration revealed corectopia and anisocoria (right 3mm, left 2mm with ambient light; right 5 mm, left 4 mm with light deprivation) (fig. 1), both with photomotor reflex and accommodation, as well as a slight left peripheral facial palsy. The neurological examination revealed hyporeflexia (+/4+) in the lower extremities and hypoesthesia and allodynia in the soles and palms. Analytical studies, autoimmune profile and vitamins were normal. Serological testing was positive for cytomegalovirus. An electrocardiogram, chest radiograph and cranial and spinal and cranial magnetic resonance imaging showed no abnormalities. A cerebrospinal fluid (CSF) analysis showed mononuclear pleocytosis (70 cells; 90% lymphocytes; elevated protein, 80mg/dl) with normal CSF glucose. The electromyographic analysis showed a predominantly demyelinating sensorimotor polyradiculoneuritis. During his latest check-ups, the patient presented an obvious clinical improvement with partial recovery from the pupillary disorder.

Figure 1 Image showing a corectopia.