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VI cranial nerve paralysis after epidural anaesthesia

Paresia del VI par tras anestesia epidural

Dear Editor:

Lumbar puncture (LP) is a technique that is widely used by different specialists (anaesthesiologists, intensive care specialists, internists, oncologists, emergency room physicians, specialists in nuclear medicine, and, of course, by neurologists) for purposes of diagnosis and/or treatment. Despite the fact that it is an invasive procedure, it is nonetheless fairly safe, albeit not without possible complications, such as headache, lumbar pain, or temporary paraesthesia of a lower limb due to ipsilateral radicular irritation. Much less common are infectious or haemorrhagic complications, or the involvement of cranial nerves (CN). We report a case following epidural anaesthesia, as well as a review of the bibliography.

A thirty-five year old female without any history of interest came to the Emergency Room due to diplopia 6 days after epidural anaesthesia with an 18-gauge needle for the birth of her first child which took place without complication. She had never had an LP previously. The day after the anaesthetic procedure, she reported having a mild, oppressive-type headache located in the neck that was unmodified by postural changes. She did not present vertigo, nausea, or vomiting or any other type of additional symptom. She considered her headache to be trivial and probably related to the little sleep she was able to get and to the typical stress of being a mother for the first time; hence, she did not consult for the headache.

The general examination was normal and the only neurological finding was VI cranial nerve palsy on the right side; the rest of the examination was normal.

The general blood work (haemogram, biochemistry, coagulation), ECG, chest X-ray, and cranial scan were all normal. One week after the onset of symptoms, a cranial magnetic resonance (MR) was performed that revealed a small patch of cerebrospinal fluid (CSF) in the supra- and infratentorial subdural space (fig. 1A). The decision was made to adopt conservative treatment and 16 days after onset, the diplopia had disappeared. Two months after the first one, a new MR was performed that showed an integrum resolution of the subdural patch (fig. 1B); the patient was therefore, discharged.

In 1891, Quincke carried out the first LP to treat intracranial hypertension associated with tuberculous

meningitis. The first complication of this technique was described after Bier himself suffered it in 1898; it consisted of a post LP headache.¹ Other complications have subsequently been reported. One of the very unusual complications of LP is CN involvement,² particularly oculomotor involvement that manifests with diplopia.

In an outstanding review, Nishio et al.³ have recorded the reported cases, including the first one to be documented in 1930. The CN most commonly affected is the VI cranial

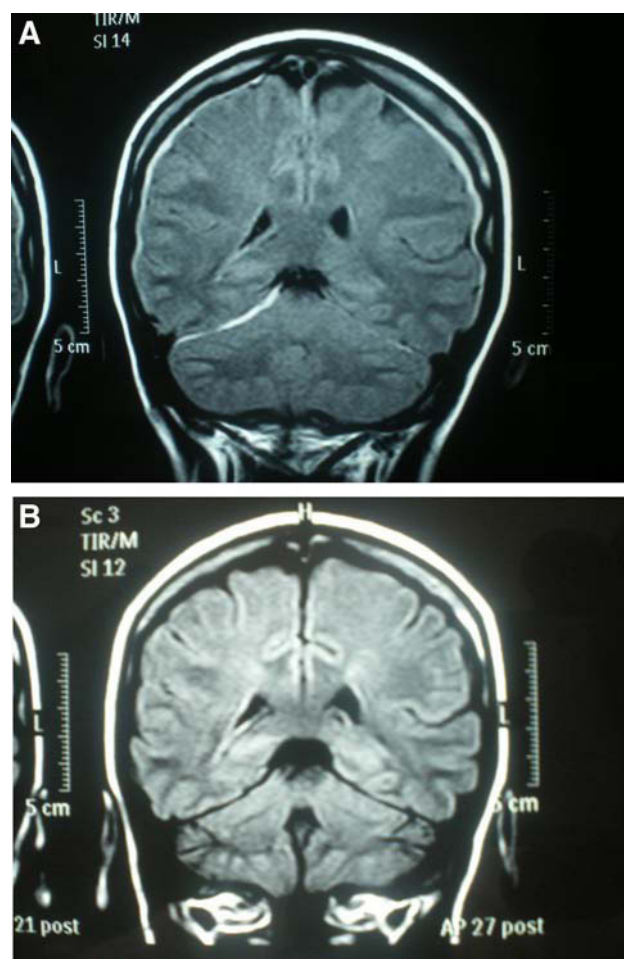


Figure 1 A) Cranial MRI. FLAIR coronal slice in which a CSF patch can be seen in the infra- and supratentorial subdural space. B) The same slice in the MRI performed 2 months later, documenting resolution of the patch.

nerve (external ocular motor nerve or abducens), as occurred in our case, given that it is the cranial nerve with the longest intracranial course and is affected by the presence of local compression or an increase or decrease in CSF pressure.^{3,4} The abducens may be affected unilaterally or bilaterally.³ We must point out that, except for the olfactory, the glossopharyngeal, and the vagal nerves, any CN can be affected.⁵ There have also been reports of injury to more than one CN^{3,6} and even combined symptoms in cranial nerves and long pathways.⁷ The involvement of CN generally takes place within the context of an intracranial hypotension syndrome (ICH) with the characteristic headache and other typical symptoms such as dizziness, nausea, or vomiting. However, in our case and in others⁵ the diplopia did not precede the headache.

Incidence data vary widely from one author to the next.^{2,3,5} The gauge of the needle and the LP target being the epidural or intradural space are factors that account for this difference. In the first case, the *dura mater* is not punctured except by mistake; hence, there is no possibility of ICH, given that there would be no exit orifice. Insofar as needle thickness or width is concerned, it must be remembered that the smaller the gauge, the thicker the needle and therefore the more likely it is to present liquorale hypotension. Thus, with a 16-gauge needle, 8 out of 839 patients presented palsy of the VI cranial nerve, and none of the more than 7,500 when the needle was 20- or 22-gauge.⁵

The pathophysiology of this process remains unknown, although the most widely accepted hypothesis is that ICH due to a dural CSF leak following LP would lead to the loss of the hydraulic suspension of the brain. This causes mechanical traction³ of painful intracranial structures, leading to headache² and eventual injury to the VI cranial nerve.³ Therefore, ICH plays a key role, whereas the LP does not. In fact, cases of VI cranial nerve palsy has been reported in patients with spontaneous ICH.⁸ It is possible that there is an individual predisposition, although there are patients in whom this has happened in their third instance of epidural anaesthesia, without incidence in the first two.⁹

Although there are cases in males³ as well, most of the patients reported were female, as in our case. ICH has always been considered to be more likely in women,³ possibly because they generally have a lower body mass index. It is unlikely for it to appear prior to puberty or from the age of 60 years onwards.

The temporary co-existence of a subdural patch of CSF together with the clinical symptoms and anaesthetic procedure leads us to think that both are related by coincidence. The subdural hygroma, also described by other authors,^{5,10} is due to the traction the brain exerts through its anchoring structures on the *dura mater*-arachnoid border, without rupturing the vessels, unlike a subdural haematoma.¹¹ Other findings reported in ICH are the descent of the brain, the decreased size of the subarachnoid cisterns, or gadolinium uptake in the meninges.¹¹

The clinical picture is not acute; rather, it tends to emerge on the fourth day post-procedure, with peak incidence taking place on day ten.³ In our patient, it appeared on the sixth day following the LP. Given that it sometimes occurs up to three weeks later, the patient or even a physician may fail to connect it with said technique

(particularly if not preceded by the headache characteristic of ICH syndrome, as in our case), causing the patient to consult with the ophthalmologist or emergency room. For this reason, these specialists must be alert and, when faced with ocular motor nerve involvement, above all, the abducens, and LP in the preceding days, should consider it as a possibility.⁵ The course of the condition tends to be benign, with resolution within a few weeks,³ as in our case, although on occasion, the symptoms have lasted for more than three months, and even as long as 7 months,⁵ 18 months,³ or 21 months,¹² or had become recurrent.¹³

As regards treatment, hydration^{2,11} and the use of epidural autologous blood patches have been proposed¹⁴ with good response, although this therapy is not always efficacious in treating palsy.¹² This divergence in outcomes may be due in part to the promptness with which treatment is initiated, ideally within the first 24 hours after clinical onset.¹⁴ Some patients have required surgical treatment to correct the ocular alterations.⁶ The wait-and-see attitude, as in our case, also allows the symptoms to resolve on their own,⁴ particularly if the ICH is mild. The size of the dural orifice (related to needle thickness) may also be involved. As so often happens in medicine, prevention is the ideal, using the smallest-calibre needles possible,^{5,15} since that will lead to a lower incidence of intracranial hypotension, which is responsible for complications¹⁵. Most authors do not report the gauge of the needle, although in some of those who do^{3,9} it has even appeared with a 25-gauge needle.^{3,5} An 18-gauge needle was used in our case.

As a conclusion, a possible cause of diplopia is CSF hypotension; consequently, this possibility should be considered (even if not preceded by the characteristic headache) when facing oculomotor palsy, above all if it involves the VI cranial nerve.

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Eagle syndrome or Stylohyoid syndrome?

¿Síndrome de Eagle o síndrome estiloideo?

Dear Editor:

We have read the article written by Cano et al. on Eagle syndrome and carotid dissection¹ with great interest. We therefore allow ourselves to make an observation due to the fact that the diseases caused by an elongated stylohyoid process generate great controversy with respect to the criteria used for their diagnosis, as well as differential diagnoses. According to Camarda et al.,² the classification for cervicopharyngeal pain associated to the elongation of the stylohyoid process is divided into three distinct entities: the first is Eagle syndrome per se, which requires the presence of neck surgery, such as tonsillectomy or trauma, together with the presence of clinical tenderness of the elongated stylohyoid process, plus an imaging examination revealing the elongation of the stylohyoid process to provide an accurate diagnosis.^{3,4} The second entity is stylohyoid syndrome, in which the patient reports symptoms, but there has been no prior surgery or trauma; nevertheless, imaging studies reveal the presence of an elongated stylohyoid process or ossification of the ligament, at a young age or adulthood (around 40 years of age). In the third entity, pseudostylohyoid syndrome, the patient describes the classical symptoms of Eagle syndrome, but there is no clear clinical or radiographic presence of any elongation of the stylohyoid process or ossification of the ligament, nor is there a history of prior surgery or trauma. The symptoms are the consequence of aging, causing the tissues to lose elasticity, often manifested in the form of tendonitis and inflammatory reactions at the level of the territory innervated by the glossopharyngeal nerve, which causes pain.⁵

What is particularly striking about the case reported is that the patient has not undergone tonsillectomy or any other neck surgery; nor has she suffered any prior trauma, thus establishing her diagnosis as stylohyoid syndrome, excluding Eagle syndrome in the differential diagnosis because it is characterized by the fact that the patients have undergone tonsillectomy or present trauma in the area,^{6,7} a fundamental situation for the manifestation of the characteristic symptoms displayed in the classical presentation. In the case reported, the patient indicates

the typical symptoms and the imaging studies document the existence of an elongated stylohyoid process, in addition to debut in adulthood (approximately 40 years of age).

It is important to point out that the elongated stylohyoid process does not necessarily translate into Eagle syndrome or stylohyoid syndrome, since most people who exhibit this anatomical variation refer no symptoms whatsoever. In those cases in which its presence gives rise to symptoms, the diagnoses possibly associated with an elongated stylohyoid process or calcification of the stylohyoid ligament must be based on an accurate clinical history and meticulous clinical examination, both physical and by imaging, including the utmost precision in recording and interpreting the data, in addition to consultations with specialized professionals who can collaborate in solving the case and thus, achieve a correct diagnosis and treatment of the condition affecting patients seeking a solution to their illness.

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