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LETTERS TO THE EDITOR

VI cranial nerve palsy following epidural anaesthesia☆

Paresia del VI par craneal tras anestesia epidural

Dear Editor:

It was with great interest that we read the article published by E. Durán Ferreras analysing the complications of epidural anaesthesia with reference to a case in which VI cranial nerve palsy (CNP) developed.[1]

The doctor presents an excellent review of the history of complications that have been described after lumbar puncture (LP), the physiopathological mechanisms that may be involved, and a description of the most common clinical presentations. The article suggests conservative treatment measures including rest, hydration, and analgesia. In many cases, this approach delivers spontaneous resolution of symptoms at some point between 2 weeks and more than 3 months. It also describes use of epidural blood patches which give mixed results, depending on how soon they are used after symptom onset.

We recently treated a patient whose clinical description was similar to that in the cited article. She had an excellent response to treatment with an epidural blood patch given 72 hours after onset of diplopia.

This 35-year-old female received an epidural during childbirth; on the following day, she experienced an intense generalised headache that intensified in a vertical position and improved upon lying down, accompanied by nausea and pain in the nuchal area. Doctors immediately employed conservative treatment measures with rest, hydration, and analgesics (metamizole + codeine, NSAIDs); the patient improved somewhat, and the doctors decided to discharge her. The patient was readmitted 5 days later due to recurrence of a similar headache and appearance of double vision when gazing to the right. Examination showed right VI CNP with no other changes. It was at that time that our department was contacted and the patient referred for care in our centre. An MRI scan (Figs. 1 and 2) revealed findings compatible with cerebrospinal fluid hypotension; bilateral subdural hygromas were present. As the headache and diplopia persisted despite hydration and analgesia, we consulted with the anaesthesiology division and decided to treat with a 20 cc autologous blood patch at 72 hours after onset of VI CNP. The patient's headache and diplopia both improved gradually in the 24 hours following the procedure. The patient was discharged 72 hours after treatment, at which time the diplopia had abated and she was able to stand without experiencing further headaches.

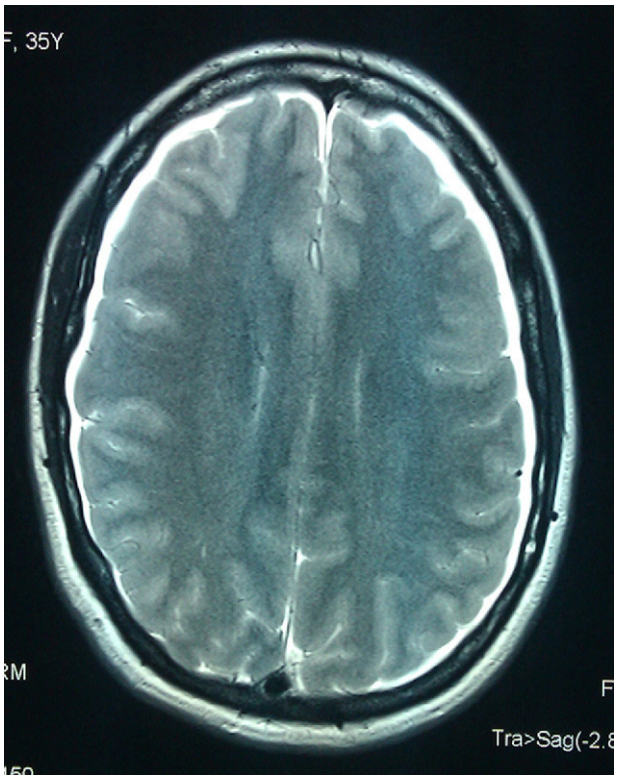

Figure 1 MRI FLAIR sequence. Homogeneous dural enhancement.

Although most published cases report complete and spontaneous resolution of VI CNP after LP within several weeks or even months after onset of the deficit, there are also other cases that do not progress well and require more aggressive treatment, including surgical treatment.[2,3] Diplopia and headache are debilitating symptoms that limit patients' daily life activities and require use of the most effective treatment possible. The literature states that autologous blood patches are only effective for cases of

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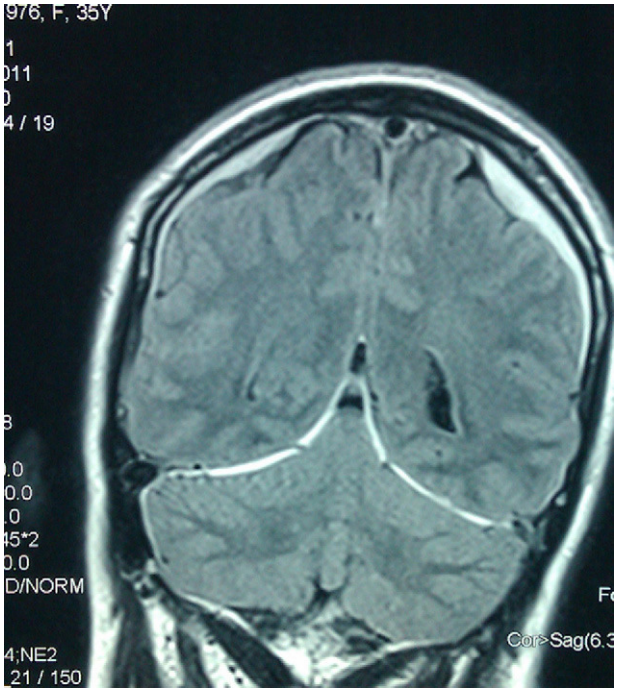

Figure 2 MRI FLAIR sequence. Bilateral subdural hygromas.

post-LP headaches and diplopia in which treatment is initiated in the first 24 hours after VI CNP appears.[4–6] The interesting feature of this case is that treatment delivered excellent results even though the epidural blood patch was administered 72 hours after palsy onset.

In conclusion, we would like to suggest considering treatment with an epidural blood patch even when more than 24 hours have passed since the onset of CNP. This treatment may significantly accelerate the patient's recovery.

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Painful polyneuropathy secondary to prolonged treatment with linezolid: Presentation of a case☆

Polineuropatía dolorosa secundaria a tratamiento prolongado con linezolid: a propósito de un caso

Dear Editor:

Polyneuropathy is a common disease that requires an exhaustive aetiological study. Even so, the cause goes undiscovered on some occasions. Toxic neuropathies represent a small percentage of this group's diseases; some cases may be reversible, which makes identifying them all the more important.[1] At present, the appearance of new drugs such as linezolid means that we have to be especially alert in order to detect potential neurotoxic side effects that had not been described before those drugs were marketed.

Our patient was a 24-year-old female smoker (20 cigarettes per day) with no other history of drug use. She was being treated with paroxetine for depressive disorder and used a vaginal ring; there was no other relevant medical or surgical history. She was referred to the hospital by her primary care doctor, who had observed positive Mantoux and bacilloscopy in a contact tracing study; her partner had recently been diagnosed with pleuropulmonary TB. Upon admission, the patient was completely asymptomatic and reported no fever, wasting syndrome, or respiratory problems. We began treatment with isoniazid, rifampicin, and pyrazinamide. After checking for good tolerance to the treatment (the patient only showed a slight increase in uric acid caused by the pyrazinamide) and a negative bacilloscopy, the patient was discharged. Since the sputum culture was positive for *Mycobacterium chelonae*, tuberculostatic treatment was replaced with clarithromycin, ethambutol, and linezolid dosed at 1200 mg daily. Three months after the treatment modification, she was readmitted for symptoms of dyspepsia, diarrhoea, and major asthenia. Analytical tests revealed normocytic

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