

ered cord syndrome.⁸ Bladder dysfunction in tethered cord syndrome varies between 40% and 93% in cases,⁹ which can manifest itself as incontinence-urgency or stress incontinence (87.5%), difficulty in urinating (40%), feeling of incomplete emptying (33%) or pollakiuria (33%). With regard to the urodynamic pattern, there are differences between authors, although it seems that the most common is bladder hyperreflexia.⁸

The treatment for tethered cord syndrome is controversial because it mainly improves sensory motor deficits and pain. Some authors advocate spinal release after diagnosis, thus preventing the progression of neurological dysfunction. Other authors note that the initial improvements are due to post-surgical denervation, especially in cases of hypertonia and hyperreflexia returning to baseline at 6 months, or that all the treatment does is stabilise the neurological deficit and does not give back normal functioning. The improvement percentage in urological disorders varies in different series, with results around 25%.¹⁰ The time from the onset of the symptoms to that of surgery is an important factor for prognosis.^{8,10}

The differential diagnosis of tethered cord syndrome starting in adulthood is vast and is made up of different myelopathies, whether deficiency, inflammatory, cancerous or from multiple sclerosis. The presence of bone malformations in lumbosacral radiography can be present in up to 100% of cases in some series.⁸ That is why this technique should be carried out in cases of neurological dysfunction, as the presence of bone anomalies steers us towards tethered cord syndrome and would indicate that we should undertake magnetic resonance studies to confirm the diagnosis.

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Lacosamide, new antiepileptic with broad perspectives[☆]

Lacosamida, nuevo antiepiléptico con amplias perspectivas

Sir,

Lacosamide is a third-generation antiepileptic drug (AED) with a good pharmacokinetic and pharmacodynamic profile that has been recently approved as adjuvant therapy in difficult-to-control partial seizures, with or without secondary generalisation.¹ Its main application is in adjuvant therapy for refractory partial seizures² with a dual mech-

anism of action: inactivation of slow sodium channels and modulation of response to type 2 collapsin.^{1,2}

We present our experience with lacosamide in various epileptic and non-epileptic disorders in which we observed a favourable response.

We present a 48-year-old female patient suffering migraine without aura from the time she was 18 (2–4 headaches/month) and no other relevant history. She referred episodes of distorted vision followed by generalised seizure crises from age 20. She was following treatment with 200 mg carbamazepine every 8 h and 3000 mg levetiracetam, and was well controlled (1 crisis of distorted vision lasting various seconds per week). Neurological examination and neuroimaging were normal. The EEG showed focality of sharp waves in the right parietal area. The patient was diagnosed with partial seizures consisting of secondarily generalised metamorphopsia. She presented leucopenia secondary to carbamazepine, so this was changed to 100 mg lacosamide every 12 h; leukocytes returned to normal after the change. After 4 months without any seizures, the patient

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reported that she had not suffered any migraine headaches since she had started taking lacosamide.

Additionally, we present two cases of epilepsy that were difficult to control with drugs. Both followed multiple therapies in various combinations: the first case, with a frequency of 6 crises per month, was taking carbamazepine. This was replaced by lacosamide due to the presence of secondary hyponatraemia. The crises decreased to 2/month and sodium values returned to normal. The second case, with multifocal crises, followed treatment with 5 AEDs. After being admitted in a convulsive state, 3 drugs were stopped and lacosamide was started. A rapid control of seizures was achieved. After 5 months, the patient maintained this improvement, with 2 crises per month.

We present a fourth case of complex partial seizures in a patient who was being treated with oxcarbazepine, which was changed to lacosamide due to secondary hyponatraemia. The crises disappeared and natraemia returned to normal.

In Spain, there are very few case reports on experience with lacosamide after it was commercialised. It was used in these 4 patients in different situations: metamorphopsia, status epilepticus, complex partial seizures and drug-resistant epilepsy, highlighting its effectiveness in a case of migraine, of which there are no cases reported in the current literature. Known results were confirmed, but we also observed an adequate response in situations not yet listed. The case with migraine suggests that lacosamide may come to have a role as a neuromodulator.

Clearly, controlled studies are needed to demonstrate the efficacy of lacosamide in the prevention of migraine. However, positive results such as the ones described above are preliminary findings that support this drug as an alternative in this indication in the future, especially if we consider that it can be effective and well tolerated. It is important to note the limited potential for lacosamide interactions, as well as the few side effects described with its

use, generally mild and transient, and dose-dependent or unspecific.¹⁻⁵

These observations, along with the known risk-benefit profile of lacosamide, suggest that it may be safe and effective in the treatment of not only epilepsy, but also for migraines.

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