

where we were surprised by several images indicative of ependymal implants in both anterior horns of the lateral ventricles, hyperintense in T1 sequence and hypointense in T2 (Fig. 1) without contrast enhancement, which did not correspond to blood-borne deposits in the sequence of magnetic susceptibility. In the control dorsal MRI, there were signs of myelopathy with traces of haemosiderin; at the boundary between D9 and D10 segments, we found a 1 cm lesion located in the right posterolateral region of the spinal cord. Not captured by IVC, it was hyperintense in T1 and hypointense in T2, with radiological semiology characteristic of melanin (Fig. 1). The ependymal implants had doubled in size in the brain MRI after 1 month of evolution (Fig. 1), a fact that strengthened the diagnosis of rapidly progressing meningeal melanocytoma with seeding or a multifocal origin. Despite this, the patient remained clinically stable with not only her spinal cord lesion but also in her cognitive deficit during the 3 months that she stayed at our centre. In the end, she was sent to the oncology department of her reference hospital to be assessed for other treatment options.

Melanotic cells are derived from the neural crest during embryonic development; they are present in a healthy adult and are the origin for pigmented leptomeningeal tumours. Among these, melanocytoma originates in the spinal cord half the time.¹ Although these tumours generally have a good prognosis when treated with a complete resection or incomplete surgery plus adjuvant radiotherapy, according to the series of cases with evolution at 5 years, the percentage of local relapses is not negligible; neither is the leptomeningeal progression and seeding, which are less frequent.² In cases described on seeding, the primary location is in the posterior fossa, with implants at a spinal level.^{3,4} This can occur after a complete tumour resection and a period of clinically-stable years. This possibility was considered in the case presented, but it seemed improbable due to the short time of evolution between the surgery and the detection of cerebral lesions.

Despite the fact that in this case the pathological anatomy did not reveal malignancy, we saw rapid tumour progression, with a possible multifocal origin. This has been published on very few occasions; consequently, the prognosis is uncertain in this case. In references that have been found, it is attributed to a possible aggressive course and a

bad prognosis.^{5,6} Finally, we would like to stress that we had previously considered a histological review of this tumour in order to modify the various treatment regimens and improve the outcome.

References

1. Wang F, Li X, Chen L, Pu X. Malignant transformation of spinal meningeal melanocytoma. Case report and review of the literature. *J Neurosurg Spine*. 2007;6:451–4.
2. González-Tortosa J, Ferri-Ñíguez B, Ros de San Pedro J. Melanocytoma meningeo del ángulo pontocerebeloso: ¿Un tumor benigno? *Neurocirugía*. 2009;20:372–80.
3. Franken SP, Setz-Pels W, Smink-Bol M, Gijtenbeek JM, Nanda D, Van Der Maazen RW, et al. Unusual case of bifocal leptomeningeal melanocytoma in the posterior fossa with seeding in the spinal canal. *Br J Radiol*. 2009;82:e182–8.
4. Koch HJ, Roeder S, Zimmermann UW, Schäfer C, Villarrubia V, Kuchelmeister K, et al. Spinal and cerebral leptomeningeal seeding from a melanocytoma of the cerebello-pontine angle. *Wien Med Wochenschr*. 2005;155:360–4.
5. Ali Y, Rahme R, Moussa R, Abadjian G, Menassa-Moussa L, Samaha E. Multifocal meningeal melanocytoma: a new pathological entity or the result of leptomeningeal seeding? *J Neurosurg*. 2009;111:488–91.
6. Bydon A, Gutierrez JA, Mahmood A. Meningeal melanocytoma: an aggressive course for a benign tumor. *J Neurooncol*. 2003;64:259–63.

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Renal failure as a first sign of tethered cord syndrome in the adult[☆]

Insuficiencia renal como debut de un síndrome de médula anclada en el adulto

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Dear Editor,

Tethered spinal cord syndrome is a congenital anomaly resulting from abnormal development of the neuraxis, in which the medullary cone is fixed at a level lower than normal.¹ It can be produced by different pathological entities such as the thickening of the *filum terminale*, intradural lipomas, diastematomyelia, dermal sinus fibre tracts, and sacral dysgenesis and acquired pathologies such as secondary adhesions to the closure of myelomeningocele.² Clinically, it is characterised by neurological symptoms, urogenital dysfunction and orthopaedic sequelae. It is usually

diagnosed in childhood, but it sometimes remains asymptomatic until adulthood.³ The symptoms can appear either spontaneously or after triggered by a trauma. Presentation with isolated urological dysfunction is very rare.⁴

A 43-year-old male presented with a personal history of migraine without aura and gastric dyspepsia with a normal panendoscopy. He did not present a family history of neurological or urological disorders. He referred to fatigue symptoms that evolved over several months, and in the blood test his GP had performed, he was found to have renal failure with normal potassium levels (urea 186 mg/dl, creatinine 5.7 mg/dl). He presented problems on urinating of a least 6 months' evolution. He had no sensory motor deficit, back pain, sexual dysfunction, amyotrophy or skin or orthopaedic disorders.

The general examination with heart and lung auscultation was normal. Abdomen with distended bladder. No cutaneous stigmata from congenital defects. Lower limbs with no pathological findings. Neurological examination: normal cortical functions and cranial nerves. Motor: symmetric and conserved tone and strength. Symmetric stretch reflexes +++/++++. Plantar cutaneous flexor reflexes. Sensitivity, cerebellum and walk, with no changes.

Blood count, coagulation, vitamin B₁₂, folic acid, anti-nuclear antibodies, liver enzymes, thyroid hormones, rheumatic tests and serum protein with no significant changes. Abdominal ultrasound: kidneys with grade III bilateral hydronephrosis with cortical thinning, predominantly on the left side with no cause for filial obstructive. Antegrade pyelography: permeable urinary tract. Lumbosacral radiography: sacral dysgenesis. Urodynamic study: detrusor hyperreflexia and urethral sphincter dyssynergia compatible with neurogenic bladder. Lumbosacral magnetic resonance (Fig. 1): meningocele with tethered cord, terminal lipoma and posterior sacral dysraphism.

The patient received treatment with oxybutynin 2.5 mg each 8 h. A right nephrostomy was also carried out, with a gradual decline of creatinine, although the levels were still raised when he was discharged (2.4 mg/dl). The neurosurgical team suggested lipoma excision surgery and untethering the spinal cord, but this was rejected by the patient because of a lack of guarantees that the surgery would be successful. As well as his treatment with oxybutynin, the patient had self-catheterisations as an out-patient. We have not been able to follow-up the patient, as he did not return to successive revisions.

Sagittal magnetic resonance of the thoracolumbar spine is the technique of choice to diagnose tethered spinal cord syndrome.⁵ The presence of a thickened *filum terminale* with a diameter greater than 2 mm and/or a conus medullaris located below the L1–L2 has been established as radiological diagnostic criteria, and these should be accompanied by symptoms for a definitive diagnosis.

In cases where it seems no trauma was triggered, it has been postulated that neck flexion and extension movements over years would produce a movement towards the top of the spinal cord and this is the most damaged part of the conus medullaris.⁶ The physiopathological mechanisms involved were impairment of microcirculation and mitochondrial oxidative metabolism.⁷

Spinal cord malformations frequently coexist with vertebral anomalies. This leads to postulating the existence of a pluripotential caudal eminence whose disturbance or deficiency would provoke the different anomalies, or even an alteration of the mesoderm from which these organs derive.

Whilst children tend to consult for orthopaedic problems, in adulthood neurological and urological manifestations are the most frequent. In a case study review undertaken in our country, urological disorders accounted for three-quarters of referrals to the neurosurgeon for teth-

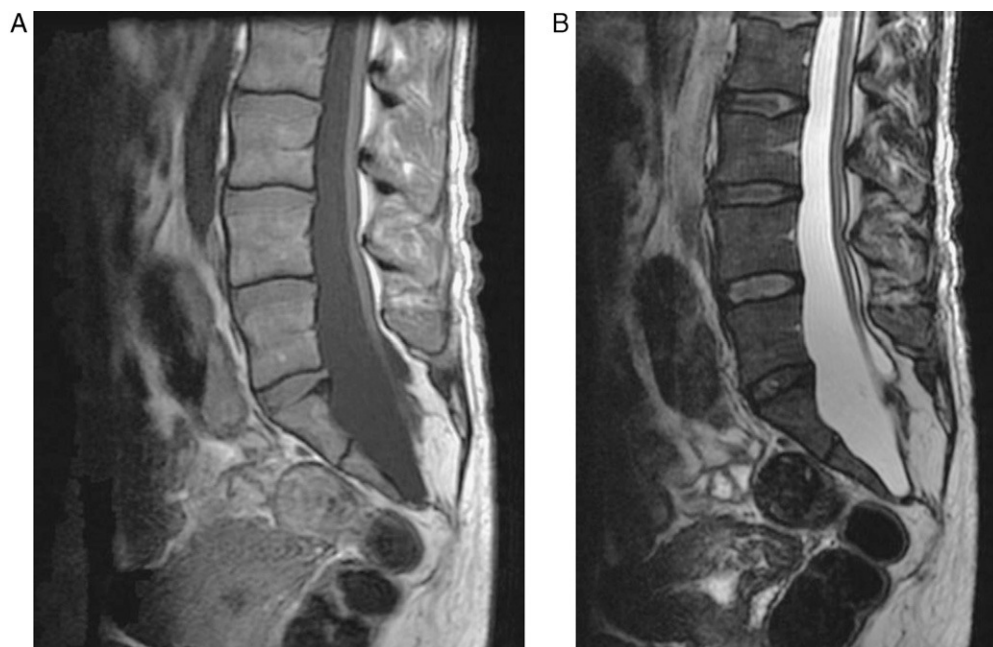


Figure 1 Sagittal plane lumbosacral MRI in T1 (A) and T2 (B) sequences: we see a meningocele with tethered cord, terminal lipoma and posterior sacral dysraphism.

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.

References

1. Lew SM, Kothbauer KF. Tethered cord syndrome: an updated review. *Pediatr Neurosurg*. 2007;43:236–48.
2. Agarwalla PK, Dunn IF, Scott RM, Smith ER. Tethered cord syndrome. *Neurosurg Clin N Am*. 2007;18:531–47.
3. Herrero-Valverde A, Martínez-SanMillán J, Heredero-Sanz J, Mora-Campillo J, Masjuan-Vallejo J. Amiotrofia progresiva de una extremidad como síntoma de presentación del síndrome de médula anclada con lipoma espinal. *Rev Neurol*. 2001;32:437–40.
4. Tareen B, Memo M, Cerone J, Bologna R, Flora R. Tethered cord syndrome in a 24-year-old woman presenting with urinary retention. *Int Urogynecol J*. 2007;18:679–81.
5. Pilo de la Fuente B, Corral Corral I, Vázquez-Miralles JM, Masjuan Vallejo J, Rodríguez del Barrio E. Síndrome de médula anclada del adulto. *Neurología*. 2007;22:201–5.
6. Gupta SK, Khosla VK, Sharma BS, Mathuriya SN, Pathak A, Tewari MK. Tethered cord syndrome in adults. *Surg Neurol*. 1999;52:362–9.
7. Filippidis AS, Kalani Y, Theodore N, Rekate HL. Spinal cord traction, vascular compromise, hypoxia, and metabolic derangements in the pathophysiology of tethered cord syndrome. *Neurosurg Focus*. 2010;29:1–4.
8. Chong C, Molet J, Oliver B, Parés P, Tresserras P, Bartumeus F. El síndrome de médula anclada: revisión casuística. *Neurología*. 1994;9:12–8.
9. Gutierrez-Baños JL, Martín-García B, Hernández-Rodríguez R, Portillo-Martín JA, Correas-Gómez MA, Del Valle-Schaan JI, et al. Síndrome de médula anclada del adulto. *Arch Esp Urol*. 1998;51:195–8.
10. Satar N, Bauer SB, Shefner J, Kelly MD, Darbey MM. The effects of delayed diagnosis and treatment in patients with an occult spinal dysraphism. *J Urol*. 1995;154:754–8.

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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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