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Transcutaneous electrical nerve stimulation to treat neurogenic bladder due to transverse myelitis☆

Neuroestimulación eléctrica transcutánea (TENS) en vejiga neurógena secundaria a mielitis transversa

Dear Editor:

Percutaneous sacral nerve stimulation with implantable electrodes is currently one of the most promising treatments for the management of neurogenic bladder,[1] while transcutaneous electrical nerve stimulation (TENS) has been shown to be effective for treating idiopathic overactive bladder.[2–4] Our group recently reported on the effectiveness of TENS administered at home for paediatric patients.[5] Transverse myelitis is a very rare cause of neurogenic bladder in children, with myelodysplasia being the most frequent cause.[1] The good outcomes observed with TENS administered at home to treat idiopathic overactive bladder led us to consider its use in paediatric patients with neurogenic bladder not caused by myelodysplasia.

We present the case of an 8-year-old girl treated over the last 5 years at our hospital's paediatric urodynamics unit due to neurogenic bladder secondary to transverse myelitis, with onset at 2 years of age. Prior to the onset of transverse myelitis, she presented adequate bladder and bowel control, having completed toilet training. Sequelae included absence of deep tendon reflexes in the lower limbs, urinary incontinence, recurring urinary tract infections, and constipation with encopresis. At 5 years of age, she was referred to our unit due to persistence of the urinary symptoms and bowel incontinence.

After proper management of the bowel symptoms, we performed urodynamic testing and evaluated the severity of urinary symptoms using the Paediatric Lower Urinary Tract Symptoms Score (PLUTSS; range 0–34).[6] A cystometry study revealed bladder capacity of 90 cc (expected capacity for her age: 240 cc); detrusor hyperreflexia with contractions of 100 cm H_2O, causing leakage; detrusor sphincter dyssynergia; and considerable urinary retention (Fig. 1). The patient scored 28 points on the PLUTSS. Treatment was started with anticholinergic drugs (oxybutynin) and intermittent urinary catheterisation, and the patient showed slight improvements in urodynamic parameters (functional bladder capacity [FBC] of 120 cc, and hyperreflexive contractions at 70 cc of filling with 50 cm H_2O of detrusor pressure) and clinical symptoms (PLUTSS = 27) (Fig. 2); no changes were observed one year after treatment onset. Due

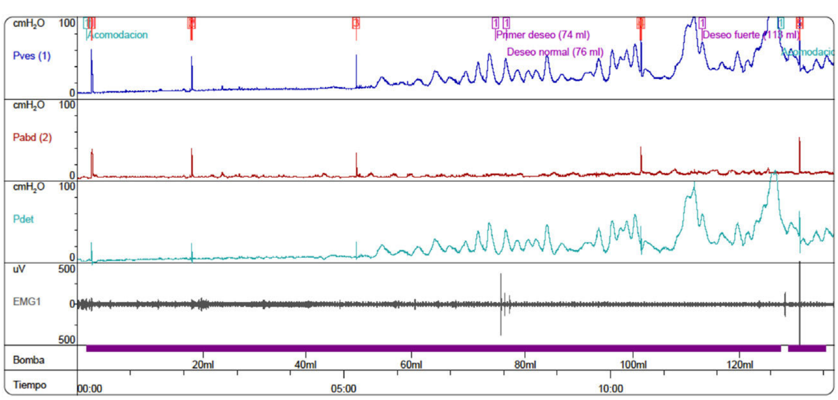

Fig. 1 Baseline urodynamic study: detrusor hyperreflexia with contractions of up to 100 cm H_2O of detrusor pressure (Pdet), causing leakage (not recorded in the image). Subsequent uroflowmetry testing revealed detrusor sphincter dyssynergia and considerable urinary retention.

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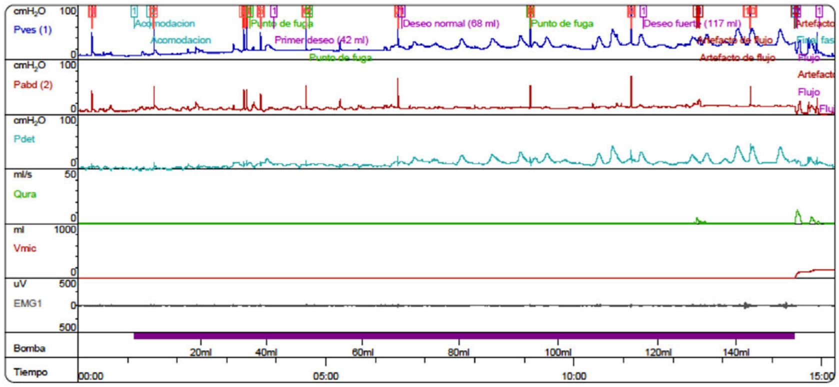

Fig. 2 Urodynamic study performed one year after onset of anticholinergic treatment, showing hyperreflexive contractions of up to 50 cm H_2O of detrusor pressure (Pdet), causing leakage.

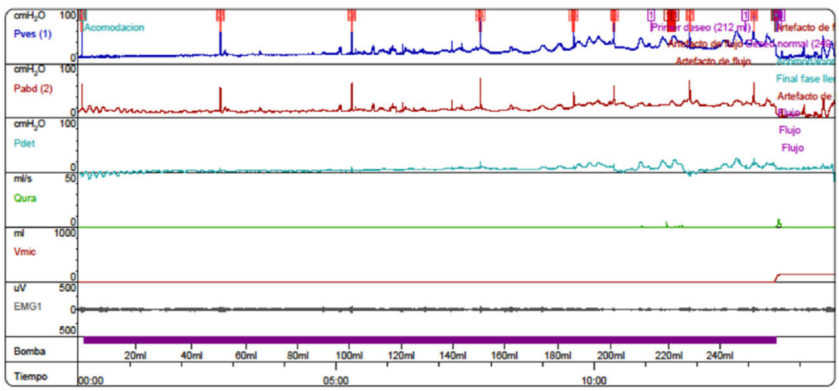

Fig. 3 Urodynamic study performed 3 months after onset of TENS: hyperreflexia had improved, with small-amplitude contractions at 200 cc of filling (FBC = 240 cc), with no leakage during the filling phase.

to refractory detrusor hyperreflexia, we proposed that TENS be added to the anticholinergic treatment before resorting to surgical treatment (enterocystoplasty or botulinum toxin injection). The family were trained to apply the stimulator (UroSTIM 2.0) to the sacrum (S2-S3) during consultations at our unit. Current was applied with a frequency of 10 Hz and a wavelength of 200 s. Intensity was regulated on a daily basis according to the maximum intensity tolerated without pain. Daily 20-minute sessions were performed while the patient was eating dinner or watching television. After 3 months of treatment, cystometry results (FBC = 240 cc, without leaks during filling; low-amplitude contractions; Fig. 3) and symptoms had improved (PLUTSS = 3).

TENS presents a high level of evidence for the treatment of paediatric idiopathic overactive bladder,[7] as shown by urodynamics study findings[8]; however, few studies have addressed its effectiveness for neurogenic bladder. Patients with myelodysplasia presenting lumbosacral involvement are ruled out as candidates for TENS; however, we believe the technique may be considered a non-invasive therapeutic option for neurogenic bladder secondary to transverse myelitis. Initial treatment response in our patient was very good, although these favourable results must be confirmed with long-term follow-up.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Multiple cerebral microbleeds associated with disseminated intravascular coagulation[☆]

Microhemorragias cerebrales múltiples asociadas con el desarrollo de coagulación intravascular diseminada

Dear Editor:

Cerebral microbleeds are one of the most significant manifestations of cerebral small vessel disease. However, this phenomenon may also be related with less common processes or conditions. For instance, cerebral microbleeds have been described as a complication of sepsis in patients presenting a combination of trigger factors, such as coagulation alterations or hypoxia.[1] MRI can be useful for the characterisation and differential diagnosis of these lesions. We present an atypical case of multiple cerebral microbleeds secondary to disseminated intravascular coagulation (DIC) in the context of cardiac arrest.

Clinical case

The patient was a 76-year-old woman diagnosed with atrial fibrillation, who was hospitalised to undergo ablation. As a complication of the procedure, she developed a cardiac tamponade, which required emergency surgery. During the operation, the patient presented cardiac arrest; pulse was recovered after 5 minutes of cardiopulmonary resuscitation and extracorporeal life support. A neurological examination performed 12 hours later revealed confusional state, spontaneous eye opening, normal pupillary reactivity, preserved brainstem reflexes, and mild paresis of the left arm, with no other focal neurological deficits. Blood analysis findings were compatible with DIC, with elevated D-dimer levels (7900 ng/mL), prolonged prothrombin time (19.1 s), a platelet count of 81×103 platelets/μL, and fibrinogen level of 0.84 g/L. No significant alterations had been detected in the blood analysis performed at admission.

Curiously, brain MRI (Fig. 1) revealed multiple punctiform hypointensities on susceptibility-weighted imaging (SWI) sequences, distributed across both hemispheres, the cerebellum, and the brainstem. These findings are suggestive of diffuse parenchymal microbleeds. No alterations were detected on other imaging sequences. These punctiform haemorrhages had minimal clinical impact, and the patient completely recovered after 4 days. A follow-up MRI study performed a week later revealed persistence of the parenchymal microbleeds, with no changes with respect to the previous study. Three weeks prior to the procedure, a head CT scan had also identified no relevant alterations.

Discussion

The peculiar imaging findings in this case are compatible with the development of multiple generalised cerebral microbleeds, with the underlying mechanism probably being DIC in the context of cardiac tamponade and subsequent cardiac arrest.

The processes most commonly associated with appearance of cerebral microbleeds are small-vessel diseases, including hypertensive arteriopathy and cerebral amyloid angiopathy.[2,3] Less frequent scenarios include moyamoya disease, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, posterior reversible encephalopathy syndrome, and radiotherapy-induced lesions.[3] All these causes were ruled out in this case due to the patient's clinical characteristics and the normal findings from previous studies, including a head CT scan and blood analysis. Air and fat embolisms share some

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