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## Differential diagnosis of flaccid paralysis in paediatric medicine☆

## Diagnóstico diferencial de parálisis flácida en pediatría

*Dear Editor:*

Guillain-Barré syndrome (GBS), an acute inflammatory polyneuropathy, is currently considered the most frequent cause of flaccid paralysis in children.[1] It is characterised by acute areflexic paralysis with albuminocytologic dissociation and it is considered a neurological emergency.[2—4] Differential diagnosis of GBS offers a wide range of possibilities; the most important diseases to rule out are spinal canal compression, transverse myelitis, botulism, and cerebellar ataxia.[1] Monitoring clinical progression and running complementary tests are the keys to definitive diagnosis.

We describe the case of a previously healthy 8-year-old boy who presented abdominal pain radiating to the back, subsequently associated with weakness in the lower limbs. The patient was referred due to suspicion of GBS. No recent history of infections or fever was reported. At admission, he presented a 20-hour history of ascending muscle weakness, paraesthesias, pain in the lower limbs, and decreased vesical sensation. He was afebrile upon physical examination, with stable ventilation parameters, Glasgow coma scale score of 15, symmetrical decreases in upper limb strength (4/5), and strength ratings of 2/5 in the right leg and 1/5 in the left. The patient reported significant pain when moving his legs, and plantar reflexes were flexor bilaterally. He also exhibited patellar tendon and Achilles tendon areflexia (1/4). Sensitivity could not be properly assessed because the patient did not cooperate. No abdominal or cremasteric reflex could be evoked. He did not present tremor, dysmetria, or truncal ataxia.

Results from the first round of complementary tests were as follows: normal haemogram and serum electrolyte study; cerebrospinal fluid (CSF) analysis contained 281 mg/dL proteins, 61 mg/dL glucose, 10 red blood cells/$mm^3$, and 0 leukocytes/$mm^3$. We requested a neurological evaluation and an emergency magnetic resonance imaging (MRI) study of the spine given the suspicion of spinal compression syndrome. Craniospinal MRI with contrast showed an intramedullary tumour extending from D5 to D9 and syringomyelia with beaded cavities proximal to the tumour. The study revealed central cord signal alterations with a nearly holocordal spread and compatible with oedema. The solid component of the tumour showed increased uptake (Fig. 1). Differential diagnosis uses MRI to distinguish between ependymoma and glioma. Brain MRI results were normal. The patient underwent an emergency surgical procedure which achieved partial resection of the mass. The histological study classified it as an anaplastic ependymoma. In the following days, the patient reported increased strength and mobility in the upper and lower limbs. He is currently able to stand with assistance and is receiving chemotherapy.

When GBS is suspected, various entities must be ruled out. One of the most important is spinal canal compression, which can also cause sudden-onset symmetrical flaccid paralysis accompanied by arreflexia in the initial stage of spinal shock. However, it may also present at a later stage with normoreflexia or hyperreflexia. Spinal cord compression requires emergency decompressive surgical treatment.

Lumbar pain, asymmetric paralysis, sensory level, and persistent bladder or intestinal dysfunction are findings which require aetiologies other than GBS to be considered.[5,6] We should highlight that in presence of sudden flaccid paralysis and the previously described

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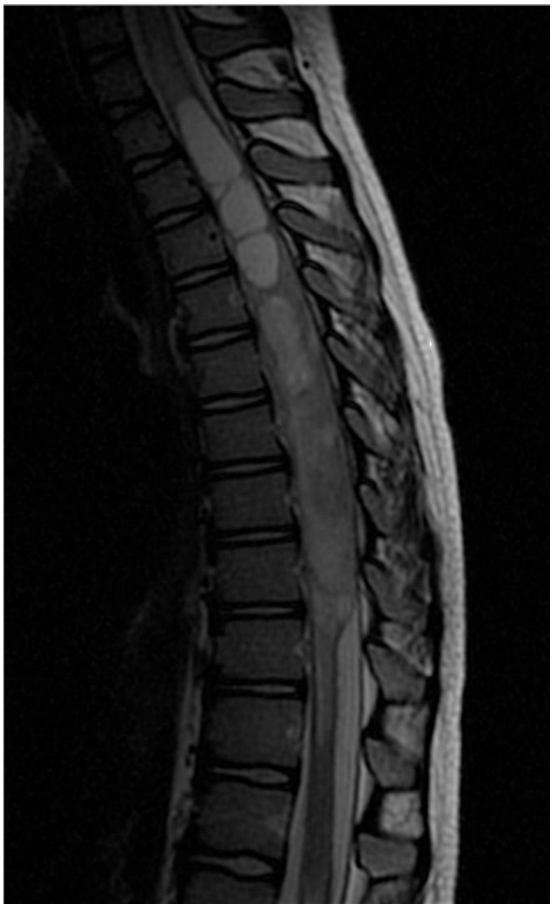

**Figure 1** Craniomedullary magnetic resonance imaging study. Sagittal T2-weighted sequence before contrast administration showing an intramedullary tumour from D5 to D9, and syringomyelia proximal to the tumour.

characteristics, a spinal MRI should be performed to rule out compressive diseases of the spinal cord, such as traumatic lesion, haemorrhage, intramedullary abscess, tumour, and transverse myelitis.[1,5–7] In our case, the diagnosis of GBS was doubtful because of presence of abdominal pain radiating to the lumbar region at symptom onset, bladder symptoms, the asymmetric pattern of weakness, and absent cremasteric and abdominal reflexes. These symptoms led doctors to the correct diagnosis and treatment. Furthermore, the patient's elevated protein levels were another source of doubt, since this finding suggests of an entity other than GBS. However, we did not find any literature citing the maximum protein levels in GBS and we believe that this value should always be scrutinised closely.

## Conflicts of interest

This study has not been presented at any of the SEN's Annual Meetings or any other meeting or congress. It has not received any funding, whether public or private. The authors have no conflicts of interest to declare.

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# Spinal cord ischaemia after endovascular thoracic aneurysm repair☆

## Isquemia medular tras reparación endovascular de aneurisma torácico

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*Dear Editor:*

Spinal cord ischaemia, the most feared postoperative complication of thoracoabdominal aneurysm repair, is a frequent event with a high morbidity and mortality. Its estimated incidence is between 2.7% and 9.5%.[1] Symptoms vary and may appear in both early and late stages. Recovery may be partial or complete. Postoperative management of spinal cord ischaemia is based on measures favouring spinal cord perfusion, mainly haemodynamic optimisation and drainage of cerebrospinal fluid (CSF).[1]

We present the case of a 70-year-old man with a history of arterial hypertension (AHT), type 2 diabetes mellitus,