experience minor sequelae, frequently in the form of seizures, headache, and hemiparesis.[14] In the case of VST, morbidity and mortality rates are correlated with baseline Glasgow Coma Scale scores. Indicators of a good prognosis are lack of damage to the parenchyma, older age, involvement of the lateral or sigmoid sinuses, and the possibility of receiving anticoagulants.[15]

ICs, although rare in paediatric patients, are associated with high morbidity and mortality rates. Performing an emergency CT scan is essential for diagnosis and early treatment since it can help prevent future complications and sequelae. This process should be managed by an interdisciplinary team including neuropaediatricians, otorhinolaryngologists, neurosurgeons, intensive care specialists, and microbiologists.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Marchiafava-Bignami disease triggered by poorly controlled diabetes mellitus☆,☆☆

Diabetes mellitus mal controlada como desencadenante de un caso de enfermedad de Marchiafava-Bignami

Alcoholism and malnutrition are the main causes of Marchiafava-Bignami disease (MBD). We present a case of MBD in which poorly-controlled diabetes mellitus is suggested as the aetiopathogenic mechanism.

Our patient, a 57-year old diabetic woman, had been hospitalised on several occasions due to hyperglycaemic episodes, with no history of previous alcoholism or malnutrition. She was admitted due to a 1-month history of memory loss, confusion, delirium, and gait disorder. The examination revealed bradypsychia and disorientation in time, space, and person; as a sign of interhemispheric disconnection, she showed left-sided ideomotor apraxia when attempting to follow directions. Blood analysis revealed a glycaemic level of 474 mg/dL and glycated haemoglobin of 11.9%. Brain magnetic resonance imaging (MRI) showed anomalies in the corpus callosum, more pronounced in its central region and appearing as hyperintensities in T2-weighted, FLAIR (Figs. 1 and 2), and diffusion sequences. The patient received treatment with intravenous thiamine (300 mg/day for 3 days) followed by oral thiamine (300 mg/day for 1

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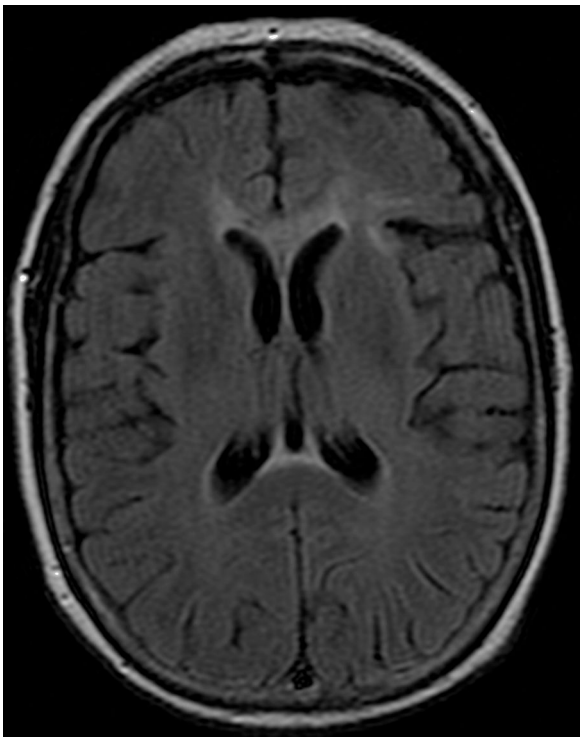

Figure 1 MRI FLAIR sequence (axial slice) showing anomalies in the genu and splenium of the corpus callosum.

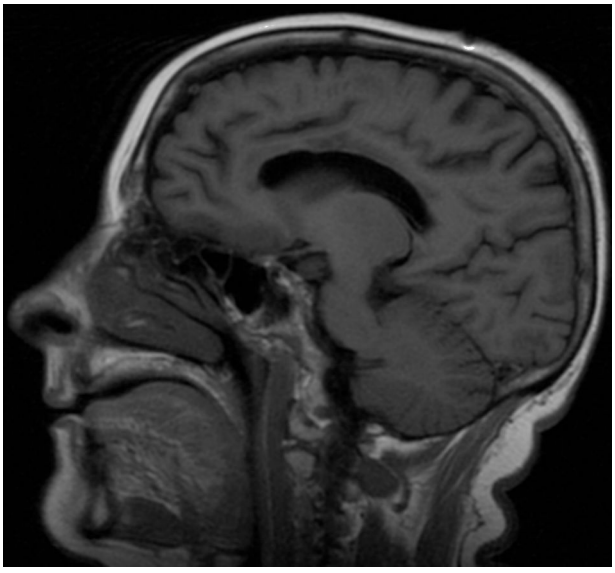

Figure 2 MRI FLAIR sequence (sagittal slice) showing diffuse and extensive necrosis of the entire corpus callosum.

month), and intravenous methylprednisolone (1 g/day for 5 days) followed by oral prednisone (60 mg/day) in decreasing doses for 6 months. Antidiabetic treatment was readjusted. Three months after treatment, her gait and ideomotor apraxia had improved but the cognitive sequelae persisted.

MBD is an infrequent disorder first described in 1903 by 2 Italian pathologists who performed autopsies on 3 alcoholic patients with rapidly progressing symptoms of neurological impairment.[1] A 2004 review article reported 250 cases of patients mostly ranging between 40 and 60 years of age, with no differences linked to ethnic group, sex, or geographical location.[2] Anatomical pathology studies described necrosis and demyelination of the corpus callosum sometimes including impairment of the semioval centre and the cerebral cortex. On the microscopic level, loss of oligodendrocytes with abundant macrophages can be observed.[3] The lesion mechanism may result from the direct toxic effect of alcohol or the indirect effect of electrolytic and osmotic changes, as in central pontine myelinolysis. It is thought to be linked to B_{12} and folate deficiencies in malnourished patients.[4,5]

Suzuki et al.[6] and Yadala and Luo[7] have recently described 2 cases of MBD in diabetic patients who were not alcoholic or malnourished. One of these studies presents a patient with no known diabetes, and therefore not being treated, whose glycated haemoglobin was 16%. The other case was a diabetic patient with glycaemia measurements ranging from 30 to 450 mg/dL and with glycated haemoglobin of 8.4%. These authors suggest that osmotic changes secondary to glycaemic fluctuations constitute the pathophysiological mechanism. These descriptions match those of our patient, who had been admitted repeatedly due to hyperglycaemic episodes.

There are 2 clinical variants of MBD; the acute form manifests with rapidly progressing decline and even death, and a subacute and chronic form, seen in our case, which progresses with dementia, extrapyramidal symptoms, incontinence, difficulty walking, and signs of interhemispheric disconnection.[8,9] Diagnosis is established using MRI, which shows hypointense lesions to the corpus callosum in T1-weighted sequences, and hyperintensities in T2-weighted and FLAIR sequences. Other findings include increased signal in DWI sequences and decreased signal in ADC maps, which is an early sign of MBD.[10] The acute form affects the genu and the splenium, while chronic forms primarily impair the truncus. Treatment consists of ceasing alcohol consumption, taking vitamin supplements and corticosteroids, and correcting glycaemia in poorly-controlled diabetics. Steroid treatment is prescribed for potentially reversible cytotoxic oedema, which can be identified as an alteration in the DWI and ADC sequences of MRI.[6] Prognosis ranges from death from acute forms to partial clinical recovery with treatment.[11,12]

We conclude that, in addition to classic risk factors, osmotic changes associated with glycaemic fluctuations, and possibly changes in other electrolytes or particles which alter normal blood osmotic concentration, can cause this disease. Diffusion sequence as a marker of a potentially reversible lesion can help in the selection of patients able to benefit from early steroid treatment in a context of hyperglycaemia.

Conflicts of interest

The authors have no conflicts of interest to declare.

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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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