no alterations. We also performed a lumbar puncture and CSF showed normal biochemical results.

We initiated treatment with thiamine at high doses during hospitalisation (500 mg IV every 8 hours for 2 days, 500 mg IV every 24 hours for 5 days) according to the recommendations of the latest guidelines on treatment of Wernicke encephalopathy. The dose was subsequently changed to 300 mg/24 hours orally and maintained after discharge. From treatment onset, clinical symptoms improved progressively. Our patient was transferred to a medium- to long-stay centre to convalescence and undergo rehabilitating therapy. One month later, in a follow-up visit in our clinic, we observed that she had improved significantly: her speech was normal, she showed no diplopia or oculomotor alterations, and she was able to walk independently.

We present a case of Wernicke syndrome with an atypical radiological presentation, although cases with cerebellar involvement have already been described in the literature.[5] Some believe cerebellar involvement may be present in more than half of the cases in autopsy studies.[6] In almost all of published cases, cerebellar lesions are associated with altered signals in typical locations.[1,7,8]

This case highlights the absence of the MRI alterations pathognomonic for this entity, and the importance of early diagnosis and treatment of a patient at risk for thiamine deficiency and a suspicious clinical profile, regardless of the locations of altered MRI signals.

Conflicts of interest

The authors have no conflicts of interest to declare, and have not received any funding for this study.

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Is acute otitis media always banal? Clinical cases and review of intracranial complications☆

Otitis media aguda, ¿una enfermedad siempre banal? Complicaciones intracraneales: casos clínicos y revisión

Dear Editor:

Acute otitis media (AOM) is one of the most frequent reasons for visits to the paediatrician. While usually regarded as benign, AOM may also cause severe complications.

We present 3 clinical cases of AOM (Table 1) and provide a literature review of the intracranial complications (IC) of these infections.

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ICs arise when inflammation extends to adjacent structures. The most frequent ICs are brain abscess, meningitis, venous sinus thrombosis (VST), and epidural and/or subdural abscess. The first 2 of these are typical in paediatric patients. Incidence of ICs, which decreased considerably with the advent of antibiotics, is estimated at 0.13% to 1.97%.[1,2]

In patient 1, the coagulation study revealed predisposition to thrombotic events. Prothrombin G20210A, or Factor II mutation, is associated with increased plasma prothrombin levels and activity, whereas the C677T mutation causes a thermolabile variant of MTHFR, leading to increased blood homocysteine levels which promote thrombosis and atherosclerosis.[3]

Patients 2 and 3 had mastoiditis. Dissemination of the infection via the emissary veins and direct erosion of the skull and the dura mater are thought to play a role in the development of brain abscess.[4]

The form of presentation differs depending on the type of IC. The main types of VST (lateral and/or transverse VST) are associated with intracranial hypertension,[5,6] whereas seizures or paresis are more likely in patients with deep vein or cortical vein thrombosis.[7] Patients with brain abscess most frequently present with non-specific

Table 1 Description of patients.

	Patient 1	Patient 2	Patient 3
Age (years)/sex	5/M	7/M	11/M
Family history	None of interest	None of interest	Father experienced sudden blindness of unknown aetiology at the age of 18
Personal history	48-hour history of bilateral AOM, oral amoxicillin	Recurrent AOM, 72-hour history of fever, oral antipyretics	2-week history of AOM, oral amoxicillin for 7 days
Symptoms and clinical findings	Instability, somnolence, dizziness, vomiting. GCS: 15	Fever, holocranial headache, 2 self-limiting episodes of aphasia, dysarthria. GCS: 15 Hyperaemic left tympanic membrane	Fever, left-sided hemicranial headache, facial paralysis, dysarthria. GCS: 15
Cranial CT scan	Thrombosis of the right jugular bulb, SS, and RTS	Abscess/empyema in the right frontal area, oedema at the site of the lesion. Occupation of left-side mastoid cells	Subdural hypodense lesion in the left convexity, mass effect, ring-shaped contrast uptake. Occupation of left-side mastoid cells
Brain MRI scan	Thrombosis of the right jugular bulb, both SS, and the right TS	Subdural collection measuring 3×2 cm in the left anterior frontal region, white matter oedema, slight midline shift. Dura mater and arachnoid enhancement	
Treatment	LMWH + cefotaxime (IV)	Cefotaxime + vancomycin + metronidazole (IV)	Cefotaxime + vancomycin + metronidazole (IV)
Complementary tests	Heterozygosity for prothrombin G20210A (Factor II mutation) and C677T mutation (thermolabile MTHFR). Normal homocysteine levels	No findings	No findings
Progression	LMWH for 6 weeks. Warfarin until completing 6 months of treatment	IV antibiotics for 4 weeks. Bilateral papilloedema on day 10, disappearing at 2 months	ICHT on day 10. CT scan revealed an abscess in the left temporal and frontal regions: surgical drainage and irrigation with gentamicin were necessary. IV antibiotics for 4 weeks
Sequelae	None	None	A single episode of generalised seizures. No AED

AED, antiepileptic drugs; GCS, Glasgow Coma Scale; LMWH, low-molecular-weight heparin; ICHT, intracranial hypertension; MTHFR, methylenetetrahydrofolate reductase; AOM, acute otitis media; MRI, magnetic resonance imaging; SS, sigmoid sinuses; CT, computed tomography; TS, transverse sinus.

symptoms: headache and fever occur in 50% to 80%, with lower percentages of vomiting and altered levels of consciousness.[8] Antibiotics can mask initial signs and symptoms, which can hinder early diagnosis and treatment. In several published case series,[9,10] numerous patients with ICs secondary to AOM had been treated with antibiotics; the highest percentage was 42.9% in the study by Migirov et al.[9]

No causal pathogen was isolated in any of the patients. According to the literature, mixed flora including *Pseudomonas* spp. and *Proteus* spp. are the most commonly reported in VST. The most frequent organisms in abscesses are *Streptococcus* (60%-70%), gram-negative anaerobes (20%-40%), and to a lesser extent, *Enterobacteriaceae*, *Staphylococcus aureus*, and fungi.[11]

There is no consensus on the management of paediatric brain abscess; most of the available evidence is based on studies of adult populations. Conservative treatment is recommended when the patient is stable and the lesion is small, well-vascularised, and located in the cortical area, as in patient 2. This approach requires close follow-up with imaging studies.[8,11]

Recent review articles on VST treatment in children (grade IB) recommend starting treatment with sodium heparin or low-molecular-weight heparin (LMWH), followed by oral anticoagulants or LMWH for at least 3 months, even in the presence of local haemorrhage. Surgery is limited to patients who do not respond to conservative treatment.[12,13]

ICs usually have a favourable prognosis. Some 80% of patients with an abscess either recover completely or

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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