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Temporal lobe encephalocele, a subtle structural lesion that can be associated with temporal lobe epilepsy☆

Encefalocele temporal, una lesión estructural sutil que puede asociarse con epilepsia temporal

Dear Editor,

Anterior temporal lobe encephalocele, or temporal pole encephalocele, has recently been associated with refractory temporal lobe epilepsy.[1,2] Temporal lobe encephalocele may be congenital or acquired and can occur in any supratentorial region of the cranium, although the anterior region of the middle cranial fossa is most frequently affected.[3] These are subtle lesions, and high-field MRI including specific sequences is essential for diagnosis.

We present the case of a 49-year-old man presenting episodes of disconnection from the environment, incoherent speech, and oral automatism, lasting 1-2 minutes. After the episodes, he remained disoriented for 30 minutes, with repetition of questions. In the neurological examination he presented a good level of consciousness and was oriented in time, space, and person. He showed no memory or language alterations. Examination of the cranial nerves only revealed mild facial droop. No other remarkable findings were observed in the neurological examination.

Results from a head CT scan were considered normal and 1.5T MRI revealed no alterations.

An electroencephalogram showed very mild abnormalities in the left temporal region, consisting of bursts of slow delta and theta waves that, although diffuse, presented increased amplitude in this region; and mild background slowing with hyperventilation, which was more evident in the left temporal region. No associated epileptiform anomalies were observed.

Treatment with carbamazepine was started, and seizures were controlled. At 5 months of follow-up, the patient's family reported memory failures that did not interfere with his daily activities.

Six years later, the patient presented an episode of disconnection, and we performed a 3T MRI scan with an epilepsy protocol including high-resolution T2-weighted coronal and axial sequences. The study revealed a small left temporal encephalocele with herniation of the brain parenchyma through a bone defect in the left temporal pole (Fig. 1).

Detecting structural alterations in the brain MRI study improves the chances of controlling seizures after surgery in patients with refractory focal epilepsy.[1,2] However, a significant percentage of patients do respond to pharmacological treatment,[1] as did our patient.

It should be noted that in 20%-30% of patients with normal MRI findings, subtle lesions may go undetected if analysis of the images is not guided by the clinical context.[1]

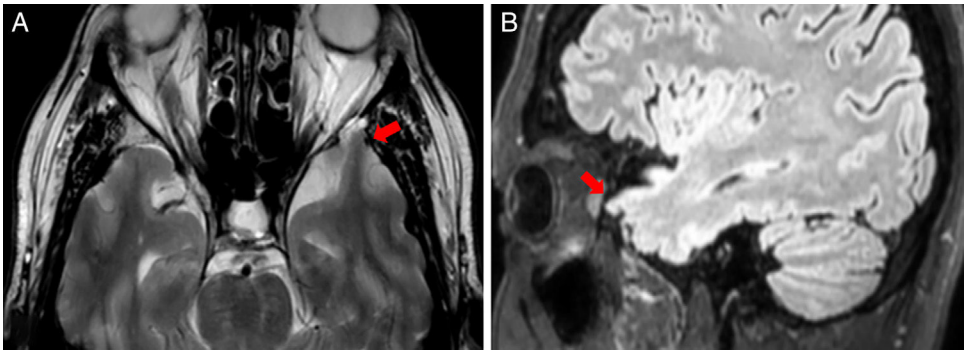

Figure 1 MRI scan: axial T2-weighted (A) and sagittal T2-weighted FLAIR sequences (B), showing a small herniation of the brain parenchyma in the anterior pole of the left temporal lobe through an anterior bone defect on the base of the cranium; this finding suggests encephalocele.

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Our patient presented complex partial seizures and mild involvement of the left temporal region, detected in the electrophysiological study and coinciding with the lesion identified by the high-field MRI; this led us to correlate both findings.

Focal epilepsy associated with encephalocele is an infrequent condition,[1–3] although its incidence may be higher than expected as the cause is frequently not identified in imaging studies of epilepsy.[1,2] This is explained by the difficulty of detecting small, isointense structural lesions to the parenchyma, as well as the limitations of MRI in studying bone defects.[1,4]

It is useful to acquire high-resolution coronal and sagittal sequences in studies of temporal epilepsy in which no alterations are detected; these can be specifically reviewed to identify subtle anomalies in the temporal pole.[4] Temporal lobe encephalocele is characterised as a herniation of the most rostral part of the temporal lobe through a bone defect on the base of the cranium, surrounded by a column of cerebrospinal fluid.[1]

The surgical procedures proposed for treating patients with refractory epilepsy include lesion resection and anterior temporal lobectomy with or without hippocampectomy,[2] although the optimal surgical strategy to follow remains controversial.[1]

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Isolated demyelinating pseudotumour: a focal, monophasic autoimmune encephalitis?☆

Lesión seudotumoral desmielinizante aislada: ¿encefalitis focal monofásica autoinmune?

Dear Editor:

Pseudotumoural lesions simulate central nervous system tumours in clinical and radiological evaluations.[1–3]

We present the case of a 28-year-old woman with no relevant history who attended our department due to subacute, progressive onset of right hemiparesis and dysarthria as the clinical manifestations of a pseudotumoural lesion. A baseline MRI study (Fig. 1) showed a heterogeneous lesion affecting the left corona radiata and centrum semiovale, with no perilesional oedema, mass effect, or gadolinium uptake. Blood analysis, lumbar puncture (with oligoclonal banding), blood and cerebrospinal fluid (CSF) cultures, CT scan, full-body PET/CT, and echocardiography yielded normal or negative results. We requested an immunodeficiency and autoimmunity study (systemic, anti-neuromyelitis optica antibodies, anti-myelin oligodendrocyte glycoprotein antibodies, onconeuronal antibodies, and neuronal surface antibodies), PCR to detect VZV and HSV in CSF, serology tests for HIV, HCV, HBV, CMV, syphilis, Toxoplasma gondii, and Borrelia burgdorferi; QuantiFERON®; and PPD skin test, all results were negative. After ruling out infectious causes, and having obtained radiological data suggesting an inflammatory pseudotumour, we started immunosuppressive treatment (Fig. 1B,C), administering 2 pulses of 1 g of intravenous methylprednisolone and one cycle of intravenous immunoglobulins (IVIg). After 5 weeks, with no therapeutic response and continuing clinical and radiological progression (Fig. 1C) (increased lesion size and contrast uptake), a brain biopsy (Fig. 2) showed findings compatible with an inflammatory demyelinating process,[1,4] with no histological data of malignancy. Treatment with rituximab achieved progressive clinical improvement. A brain MRI scan (Fig. 1D) performed one month later showed decreased lesion size and no contrast uptake. The patient was discharged with a diagnosis of inflammatory pseudotumour. She remains under follow-up by the neurology department, and at the time of publication has presented no further episodes.

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