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LETTERS TO THE EDITOR

Epileptic seizure and lipoma of corpus callosum: cause or incidental finding

Crisis epiléptica y lipoma del cuerpo calloso: causa o hallazgo

Dear Editor,

Lipomas represent less than 1% of intracranial lesions.[1-4] Their association with epileptic crises has been reported.[1,3]

We report the case of a 21-year-old male who attended the emergency service for an episode of non-convulsive generalised epileptic crisis. The neurological examination was normal. The patient presented a history of headache, but not of epilepsy or psychomotor retardation. The cranial computed tomography (CT) revealed a midline mass with a density of -105 HU and peripheral calcification. Magnetic resonance imaging (MRI) study showed a homogeneous lesion in the anterior region with a hyperintense signal on T1 sequences (fig. 1), which was reduced in T2 (fig. 1) and vanished in fat-suppressed sequences (fig. 2), associated to partial agenesis of the corpus callosum. The electroencephalogram showed generalised paroxysmal activity with sharp waves, intensified by hyperpnoea unrelated to intermittent photic stimulation. Adequate correlation was found between the lipoma location and the clinical-electroencephalographic characteristics of the crisis, so the patient was diagnosed with epileptic crisis probably related to tubulonodular lipoma of the corpus callosum and partial agenesis of the latter.

Lipomas are located in the pericallosal region in the vicinity of the cerebral cisterns.[1-4] It has been suggested that they are the result of abnormal persistence and poor differentiation of the primitive meninges during the development of subarachnoid cisterns.[1,2,4,5]

The prevalence of symptomatic lipomas is controversial: some establish it at 60%,[4] while others advocate that the majority of lipomas are asymptomatic.[1,5] Epilepsy, headache, psychomotor retardation and alterations of the cranial nerves are the most common manifestations reported.[1,2,4,6] In 1980, Gastaut et al[7] determined the relationship between corpus callosum lipomas and epilepsy, its serious, frequent and early (before 15 years) character and its pathophysiology due to interhemispheric disconnection. Since then, several authors have reported the association between intracranial lipomas and epilepsy. However, few have found an adequate correlation between mass location and clinical and electroencephalographic characteristics of the epileptic crises that would help to consider the tumour as the cause of the crises.[3] In 2006, a review was conducted on 3,500 epileptic patients in search of lipomas. Only 5 cases were found and that correlation was found in only one of them,[3] therefore, only in this case was it considered that the crisis

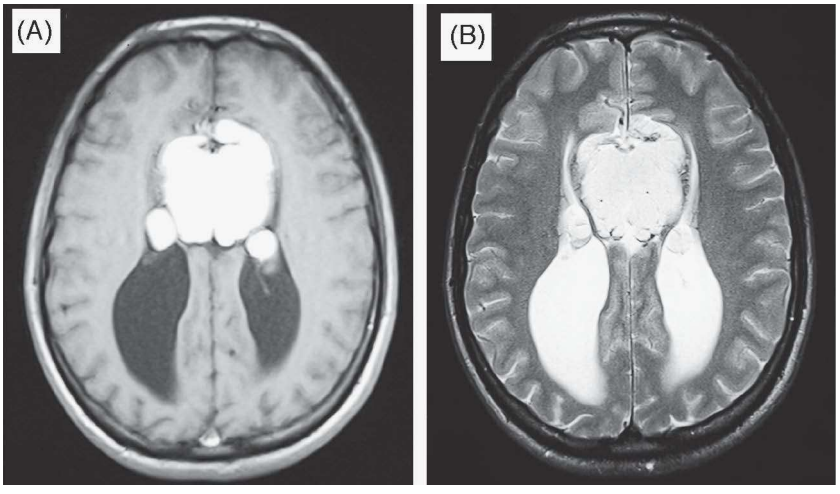

Figure 1 A: T1-weighted Magnetic resonance imaging (MRI) in the axial plane. B: T2-weighted MRI, in the axial plane.

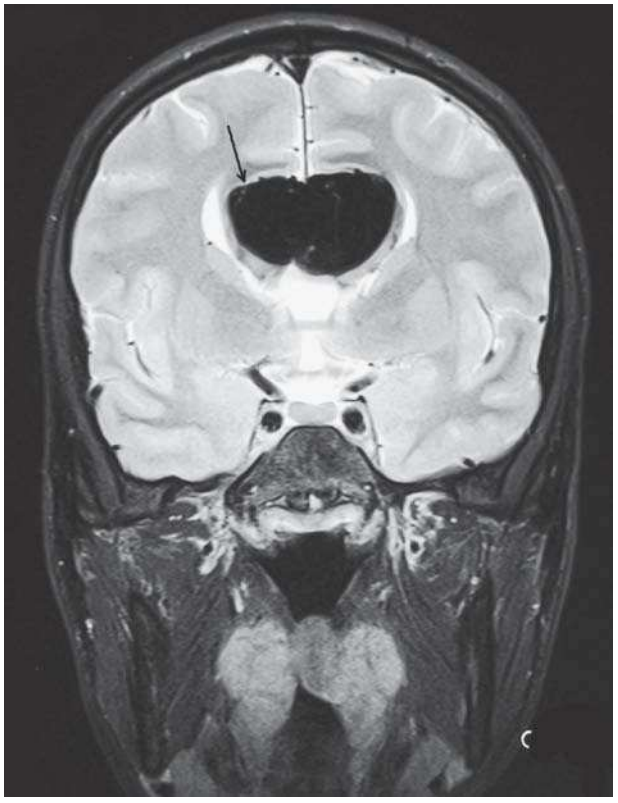

Figure 2 Fat-suppressed T2-weighted MRI, in coronal plane.

stemmed from the lipoma. The relationship between lipoma and epilepsy could be incidental[3] and less frequent than expected,[3] according to initial studies.[7] A prevalence of epilepsy around 20% has been reported in a series of lipoma cases in adults and around 5% in another similar series with paediatric patients. In the adult series, the predominant symptom was headache,[5] in the paediatric series, it was psychomotor delay.[6]

Lipomas of the corpus callosum may be tubulonodular or curvilinear. The first type, more frequent and voluminous, is often anterior and frequently associated with malformations.[2] Curvilinear lipomas tend to be posterior, smaller and asymptomatic.[2] A connection with brain malformations has been described in half of corpus callosum lipomas,[2,4] and their agenesis/dysgenesis stands out. Furthermore, the coexistence of vascular abnormalities[1] and cortical dysplasias[1] (by interference with the development of the cerebral cortex) has also been reported.

Neuroimaging studies often provide a certain diagnosis. The CT shows a lobulated extra-axial mass with a density between —50 HU and —100 HU that is frequently calcified,[1,2,6] has no oedema and is not enhanced by contrast. Calcification is more common in lipomas of the corpus callosum than in other locations[2]. The MRI shows a homogenous mass with hyperintense signal in T1, which decreases in T2 and vanishes in sequences with fat suppression.[1-4,6] It does not present paramagnetic contrast enhancement; however, its administration can reveal a venous malformation.[1] Angio-MRI can detect arterial abnormalities.[1] A differential diagnosis should carried out, especially with dermoid cysts and teratomas.[1,2] Dermoid cysts, hyperintense on T1, present a heterogeneous signal on T2 with some mass effect;[1,2] teratomas present a more heterogeneous signal and may uptake contrast.[1]

Neurosurgical excision is contraindicated[1,3-5] due to significant vascularization and adherence to adjacent tissue (which hinder resection without injuring the adjacent parenchyma), lack of growth (when expansion with prolonged steroid therapy is described)[1] and the lack of mass effect. Placing drainage or administering antiepileptic drugs may be necessary in cases of hydrocephalus or epilepsy.[4,5]

In conclusion, intracranial lipomas can be the cause of epileptic crises. However, the higher prevalence of lipomas in the epileptic population[3] should be considered. When a lipoma is identified in a patient who complains of epileptic crises, it is advisable to seek an alternative explanation, especially in cases with little correlation between the mass location and the clinical and electroencephalographic characteristics of the crisis. Fat-suppressed sequence MRI provides a security diagnosis. The correct diagnosis has prognostic and therapeutic implications since neurosurgical excision is contraindicated, in contrast to the management of most intracranial masses.

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