Arachnoid cysts and absence epilepsy
An evidence or a coincidence?

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While evaluating a cohort of 22 patients with absence epilepsy, we found two cases of 46- and 41-year-old females (Patient 1 and 2), who presented arachnoid cysts in the choroid fissure. The Patient 1 presented eyelid myoclonic epilepsy, early onset (11 years old) and no family history. Patient 2 presented juvenile absence epilepsy, characterized by sudden and brief impairment of consciousness, also early onset (14 years old) and positive family history. During video-electroencephalography (EEG) monitoring, Patient 1 presented flickering after eye-closure in bright light room and consciousness impairment during some of the flickering (Fig 1). Photic stimulation increased the frequency of eyelid myoclonic absence seizures. The ictal register showed spike-wave discharges of 4 Hz with bilateral projection (3 seconds interval) and anterior prevalence. The video-EEG of the Patient 2 showed 2.5- to 3 Hz generalized spike and wave discharges. Both patients underwent brain magnetic resonance (MR) imaging that showed well defined lesions compressing the hippocampus, presenting high signal on T2-weighted images and low signal on T1-weighted images, with no contrast enhancement, both located in the right choroid fissure, sizing 1.9 cm (AP) × 1.6 cm (L) × 0.9 cm (T) in Patient 1 and 1.8 cm (AP) × 1.8 cm (L) × 1.2 cm (T) in Patient 2 (Fig 2).

The relation between arachnoid cysts and absence seizures was not previously discussed in literature. In addition, there are some theories supporting that a cortical focus could play a leading role in the origin of generalized spike wave discharges¹. Meeren et al.¹ suggested that primary generalized epilepsies, including absence epilepsy, are the expression of a cortical abnormality. They argued that the initial leading spike appears first in a circumscribed area of the perioral region of the somatosensory cortex, which has a low threshold for spike generation. The spike rapidly spreads over the cortex, thus giving generalized appearance to the
discharges. However, to the best of our knowledge, the only report of a focal lesion associated with absence seizures was presented by Nakanishi et al. They reported a case of typical absence epilepsy in a 34-year-old woman without history of seizures, who had hypoparathyroidism and hyperostosis frontalis interna causing mild compression over the frontal lobes. The authors suggested that the compression of the superior medial frontal lobes by the hyperostosis could be deeply involved in the development of spike-wave stupor in the patient.

Corroborating the cortical focus theory and previous reports of a focal lesion associated to absence epilepsy, these cases suggest that arachnoids cysts in the hippocampus region might be associated with absence epilepsy, but further studies should be conducted in order to define whether that association is causal or casual.

REFERENCES

Fig 2. [A] Axial T2-weighted MR image of case one shows a hyperintense cystic appearing lesion in the right choroid fissure, which is compressing the head and body of the hippocampus. Axial T2- and coronal T1-weighted [B, C] images of case two demonstrate an image with similar aspects of the previous case, presenting high signal on T2-weighted image and low signal on T1-weighted image. The image findings in both cases suggest the diagnoses of arachnoids cysts in the choroid fissure.