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Visual Diagnosis

Unexpected Neuroimaging Findings in an Adolescent With First Seizure Presentation

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This 17-year-old male patient presented following a witnessed tonic-clonic seizure episode associated with acute alcoholic intoxication. No symptoms of raised intracranial pressure were reported. Past medical history including birth history was unremarkable. The patient is in the final year of high school, and social history revealed no deficiency in cognitive or social development. Neurological and ophthalmologic examinations were normal. Brain magnetic resonance imaging (MRI) revealed severe narrowing of the cerebral aqueduct and noncommunicating hydrocephalus without periventricular interstitial edema. Phase-contrast cerebrospinal fluid (CSF) flow study showed high-velocity flow through the aqueduct with a peak velocity of 18.9 cm/second. Chronic structural deformities were present, and specifically there were intraparenchymal diverticula arising from the temporal horns. Increased T2 fluid-attenuated inversion recovery signal intensity of the left hippocampus and the pulvinar of the left thalamus were present

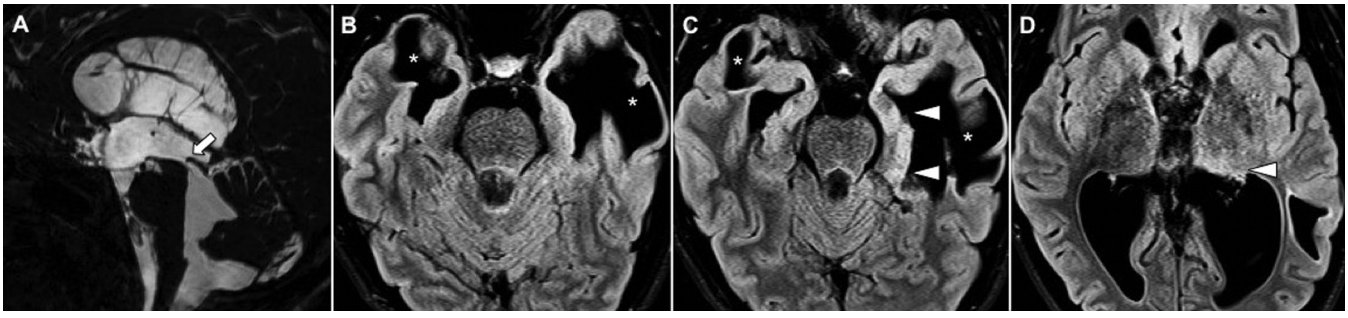

FIGURE. MRI high-resolution sagittal T2-weighted sequence (A) shows severe stenosis at the upper portion of the cerebral aqueduct (arrow) in a background of noncommunicating hydrocephalus. Fluid-attenuated inversion recovery (FLAIR) images (B-D) show pronounced dilatation of the lateral and third ventricles with intraparenchymal ventricular diverticula arising from the temporal horns of lateral ventricles (B and C, asterisk). The ventricular diverticulum on the right is limited to the temporal lobe anteriorly, whereas the more extensive left ventricular diverticulum extend into the inferior parietal lobule (asterisk). Increased T2 FLAIR signal intensity of the pulvinar of the left thalamus (C, arrowheads) and the left hippocampus (D, arrowhead) are consistent with peri-ictal changes.

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and consistent with peri-ictal findings (Fig). Aside from this provoked seizure event, the patient was otherwise well. He remained seizure free without medication on 12-month follow-up. MRI at 12 months showed no change in the ventricular morphology, and CSF diversion procedure was not indicated.

Discussion

Aqueductal stenosis is a common cause of noncommunicating hydrocephalus, which has a bimodal age distribution occurring in either the first year of life or in early adolescence.[1] Structural adaptations to impaired CSF circulation can be classified into acute and chronic phases. MRI findings of structural deformities in chronic hydrocephalus include stretching of the commissure fibers and focal enlargement of the third ventricle with associated compression of the optic chiasm and periaqueductal gray matter. An intraparenchymal diverticulum is a rare and extreme form of structural deformity. Intraparenchymal diverticula are caused by CSF dissection through weak points of the ependyma, which can lead to displacement into white matter tracts. Temporal horn intraparenchymal diverticula of the lateral ventricles are the most common site of involvement.[2,3] The prevalence of epilepsy associated with aqueductal stenosis is approximately 15%, with a stronger predilection for both temporal lobe and generalized seizures.[4] We presume that the impaired CSF flow, alteration in CSF pressure, and the aforementioned structural deformities may have lowered the patient's seizure threshold in addition to his acute alcohol intoxication. Despite the dramatic neuroimaging findings, our patient had no focal neurological deficit or further seizure episodes. This patient highlights not only the extreme structural deformities in chronic hydrocephalus but also the remarkable ability of the brain to adapt.

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