Temporal lobe parenchyma herniation: An etiological cause of headache or coexistence?

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Abstract

This report illustrates a patient presenting with a unilateral headache at whom brain magnetic resonance imaging showed temporal lobe parenchyma herniation. In our patient, there were no clinical or neuroimaging findings of increased intracranial pressure (ICP) that might lead to a headache. On the other hand, considering the dynamic nature of headache in our patient, we cannot exclude a mechanism of possible episodic ICP increments that might be related to encephalocele. We think that the detailed illustration of this case may present substantial perspectives for further deliberations regarding the headache pathophysiology.

Key words: Headache; Pathophysiology; Temporal lobe parenchyma herniation

Introduction

Spontaneous temporal lobe encephaloceles can be defined as brain herniations of middle cranial fossa origin that are not associated with traumatic, inflammatory, neoplastic, or iatrogenic conditions.1 The mechanism of brain parenchyma herniations, as it is in the classic encephaloceles, is not clear.1 However, it has been suggested that non-union of ossification centers in bones or variations of bone thickness may cause brain tissue herniation by the pressure of the brain tissue or cerebrospinal fluid (CSF).2 These lesions may be recognized during either childhood or adulthood. However, the clinical impact of this entity is unclear, in some circumstances, the differential diagnosis may be challenging. In this report, we illustrate a patient presenting with headache at whom cranial magnetic resonance imaging (MRI) showed temporal lobe encephalocele. Via the clinical presentation of this report, we will draw attention to this radiological sign and discuss the possible clinical impacts.

Case Report

A 34-year-old male patient applied due to a headache that had emerged over the last one year period. He suffered from depressive symptoms at that term and a headache had emerged following acute sinusitis. The headache was not accompanied by nausea and vomiting. Sensitivity to light and sound was not defined. Remarkably, he stated lachrymation attacks during the headache, however, no other signs suggesting autonomic cephalalgias were present. The patient stated constant headache localized at forehead that he scored 3 points on the numeric pain rating scale. However, several times a week, the severity of the headache intensified for a few hours up to 7 points on the right forehead. The fundus examination was within normal limits and the other neurological exam was normal. Cranial MRI showed temporal lobe parenchyma herniation into the right transverse sinus (Figure 1). Of note, no neuroimaging signs of increased intracranial hypertension were present. Taken together, the headache was categorized under the head of ‘headache unspecified’. Considering the coexisting depressive symptoms, amitriptyline 10 mg was initiated that provided moderate improvement in the subacute period.
Discussion

The majority of temporal encephaloceles are probably asymptomatic. Although there are reports associating some clinical symptoms with temporal encephaloceles, many of them remain undiagnosed or they are recognized coincidentally on MRIs. On the other hand, lesions causing dural venous sinus filling defects including dural sinus thrombosis, arachnoid granulations and tumors are more commonly encountered in clinical practice, may frequently resemble encephaloceles. Therefore, establishing the diagnosis of temporal encephaloceles may be challenging. It is isointense to brain parenchyma on all sequences by magnetic resonance imaging, surrounded by a cerebrospinal fluid rim and is seen to be contiguous with brain tissue on the image.

Interestingly, symptoms of headache, dizziness, syncope, and imbalance have been reported in cases with temporal lobe parenchyma herniation, however, the causal associations remain unclear. In these reports, the clinical presentation and the headache characteristics of the patients are not defined in detail constituting the main limitations. In our patient, the headache was not worse in the morning or it did not differ according to the position that might suggest an increased ICP. Besides, papilledema was absent. Taken together, there was no clinical or neuroimaging sings of increased intracranial pressure (ICP) that might lead to a headache in our patient. On the other hand, considering the dynamic nature of headache (it intensified for a few hours up to 7 points), we cannot exclude a mechanism of possible episodic ICP increments that might be related to encephalocele. However, amitriptyline (a medication not influencing the CSF dynamic) provided moderate amelioration in headache also reducing this presumption. The results of the future reports including the detailed examination of individuals with temporal lobe parenchyma herniation may provide crucial perspectives in this regard. Besides, the clarification of this possible association may also present substantial perspectives regarding the unknown
pathomechanisms underlying many subtypes of headaches as well as the group of headache currently categorized as ‘headache unspecified’.

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References
