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Case Report

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Familial Hemiplegic Migraine Mimicking a Brain Lesion in a Child: A Case Report

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Abstract

Familial hemiplegic migraine (FHM) is a rare autosomal dominant subtype of migraine with aura that may present with acute focal neurological deficits in children, closely resembling serious intracranial pathologies such as brain tumors or vascular malformations. We report a previously healthy 9-year-old boy who presented with acute-onset frontal headache followed by progressive left-sided hemiparesis and paresthesia. Initial brain computed tomography raised concern for a possible brain mass, although no vasogenic edema or mass effect was reported. Subsequent brain magnetic resonance imaging (MRI) showed no structural abnormalities, with mild asymmetry of the lateral ventricles interpreted as a normal anatomical variant. Notably, there was no evidence of a space-occupying lesion, ischemia, or parenchymal abnormality. The patient's neurological deficits completely resolved within 48 h with supportive care. A positive maternal history of similar episodes prompted genetic testing, which confirmed a pathogenic variant in the ATP1A2 gene (consistent with familial hemiplegic migraine type 2 (FHM2)), establishing the diagnosis of familial hemiplegic migraine. Familial hemiplegic migraine should be considered in the differential diagnosis of acute hemiparesis in children. Early recognition can prevent unnecessary invasive investigations and guide appropriate management and family counseling.

Key words: Brain Diseases, Case report, Familial hemiplegic Migraine, Hemiparesis, Pediatric

Introduction

Familial hemiplegic migraine (FHM) is a rare but clinically significant subtype of migraine characterized by transient motor weakness accompanied by aura and headache. Unlike common migraine, FHM has a strong genetic basis and can present with neurological symptoms that overlap with other serious central nervous system conditions, including stroke, encephalitis, and space-occupying lesions, making accurate diagnosis challenging. The similarity of FHM to a brain tumor or other structural brain lesion is particularly problematic in pediatric emergency settings, where acute hemiparesis often triggers an urgent search for a mass or vascular abnormality. FHM is classified into three major subtypes—FHM1, FHM2, and FHM3—associated with pathogenic variants in CACNA1A, ATP1A2, and SCN1A genes, respectively, which alter ion channel function and neuronal excitability (1, 2).

In the pediatric population, hemiplegic migraine constitutes a particularly rare and diagnostically complex entity. Studies suggest that pediatric hemiplegic migraine often presents with prolonged neurological symptoms such as aphasia, seizures, and transient hemiparesis, which can mimic acute encephalopathy or space-occupying lesions on neuroimaging (3, 4). The paucity of disease-specific epidemiologic data further complicates early recognition, and there is frequently a substantial delay between symptom onset and definitive diagnosis (5).

Recent case reports highlight the diagnostic pitfalls associated with FHM. For example, a 2024 study reported an adolescent with FHM2 due to an ATP1A2 pathogenic variant who presented with

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recurrent hemiplegia, headache, and persistent hyperperfusion on MRI, features initially suggestive of vascular or structural pathology (6). Similarly, pediatric patients with atypical initial manifestations such as acute encephalopathy have been documented, underscoring the broad clinical spectrum and the potential for misdiagnosis (4).

Unlike FHM, a true brain tumor in children typically presents with progressive headache, nausea, vomiting, and signs of increased intracranial pressure (ICP) rather than acute isolated hemiparesis without mass effect or vasogenic edema on imaging. Given these complexities, differentiating FHM from brain tumors and other space-occupying conditions is critical in pediatric neurology. Careful integration of clinical history, family pedigree, genetic testing, and advanced neuroimaging is essential to avoid unnecessary interventions and to guide appropriate management strategies.

Case presentation

A 9-year-old previously healthy boy presented with sudden-onset frontal headache associated with a sensation of heaviness on the left side of the body. Shortly after headache onset, he developed paresthesia and weakness of the left upper limb, followed by involvement of the left lower limb. The neurological deficits progressed over several hours, resulting in an inability to move the left side of the body.

There was no history of fever, recent infection, trauma, seizure, loss of consciousness, or prior similar episodes. On examination, the patient was alert and oriented. Neurological examination revealed left-sided hemiparesis without cranial nerve involvement or signs of increased intracranial pressure (such as papilledema or bulging fontanelle).

Given the acute focal neurological presentation, the initial differential diagnosis included an ischemic stroke, intracranial mass lesion, and demyelinating disorder. Although brain tumor was considered, the absence of signs of increased ICP made a space-occupying lesion less likely; however, neuroimaging was urgently performed to rule out a structural cause. Brain computed tomography performed during neurosurgical evaluation suggested a possible space-occupying lesion, but no vasogenic edema, mass effect, or hydrocephalus was reported, and the finding was considered nonspecific. The patient was referred for further evaluation with magnetic resonance imaging.

Brain MRI (including T1, T2, and FLAIR sequences in axial, coronal, and sagittal planes) demonstrated no evidence of a mass lesion, ischemia, or parenchymal abnormality. Specifically, there was no mass effect, no abnormal enhancement, and no vasogenic or cytotoxic edema. Mild asymmetry of the lateral ventricles was noted and interpreted as a normal anatomical variant.

The patient was admitted for observation and treated conservatively with hydration and supportive care. Over the subsequent 48 hours, both headache and neurological deficits resolved completely, with no residual weakness.

Further history revealed that the patient's mother had experienced recurrent migraine attacks associated with transient unilateral weakness in previous years. Based on the clinical presentation, complete reversibility of symptoms, normal neuroimaging, and positive family history, familial hemiplegic migraine was suspected. Genetic testing confirmed a pathogenic variant in the ATP1A2 gene, consistent with familial hemiplegic migraine type 2 (FHM2).

Discussion

Familial hemiplegic migraine is a rare but important cause of acute hemiparesis in children. The condition is characterized by migraine attacks accompanied by reversible motor weakness and may present with dramatic neurological symptoms that closely resemble stroke, intracranial mass lesions, or encephalitis. In the present case, the acute onset of hemiparesis without prior warning signs initially raised concern for a brain tumor or vascular event. However, several features argued against a structural lesion: (1) absence of signs of increased intracranial pressure, (2) lack of vasogenic edema or mass effect on CT and MRI, and (3) complete resolution of deficits within 48 hours without any specific therapy.

In pediatric patients, the acute onset and progression of focal deficits often necessitate urgent neuroimaging to exclude life-threatening conditions. Although MRI findings are typically normal in FHM, transient cortical changes have been reported in some cases. The absence of structural abnormalities, combined with complete symptom resolution, should raise suspicion for a functional etiology such as migraine. When a child presents with acute hemiparesis, the primary differential diagnoses should include ischemic

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stroke, hemorrhagic stroke, vascular malformation, demyelinating disorders (e.g., acute disseminated encephalomyelitis), and hemiplegic migraine, rather than primarily a brain tumor, which typically presents subacutely with signs of increased ICP (7).

Family history plays a critical role in diagnosis, and genetic testing can provide definitive confirmation. Early identification of FHM is essential to prevent unnecessary invasive procedures, reduce healthcare burden, and allow appropriate counseling regarding prognosis and recurrence risk. Specifically, identifying the ATP1A2 mutation in this case (FHM2) carries clinical implications, as different FHM subtypes may have varying degrees of severity, age of onset, and associated neurological features such as seizures or cerebellar involvement.

This case also highlights that the term "brain tumor mimic" may be too restrictive; "brain lesion mimic" is more accurate, as FHM can mimic various structural or vascular lesions, not only tumors. Furthermore, the incorrect imaging figures from the original submission (which showed pelvic and cervical spine MRI) have been corrected, and only appropriate brain MRI sequences are referenced. All authors confirm that the correct images are available upon request from the corresponding author.

Conclusion

This case highlights familial hemiplegic migraine as an important diagnostic consideration in children presenting with acute focal neurological deficits and headache. Awareness of this entity among pediatric neurologists and emergency physicians can facilitate prompt diagnosis, avoid misinterpretation as intracranial pathology, and guide optimal patient management. Genetic confirmation (including specification of the mutation type) and careful attention to the absence of mass effect or signs of increased ICP are key to avoiding unnecessary interventions.

Ethics Approval and Consent to Participate

Ethical approval was not required for this retrospective case report in accordance with institutional guidelines. Written informed consent for participation in this case report was obtained from the patient's parent or legal guardian.

Consent for Publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parent or legal guardian.

Data Availability Statement

All data generated or analyzed during this study are included in this published article. No additional datasets were generated.

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Authors' Contribution

A.M and N.F were responsible for the study conception and design, clinical management of the case, data acquisition, analysis and interpretation, manuscript drafting, and final approval of the submitted version.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Declaration of Generative Artificial Intelligence (AI) in Scientific Writing

The authors declare that generative artificial intelligence tools like DeepSeek were used solely for language, editing, and to improve grammar and clarity.

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