Case Report

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An Unusual Presentation of Sporadic Hemiplegic Migraine: A Case Report

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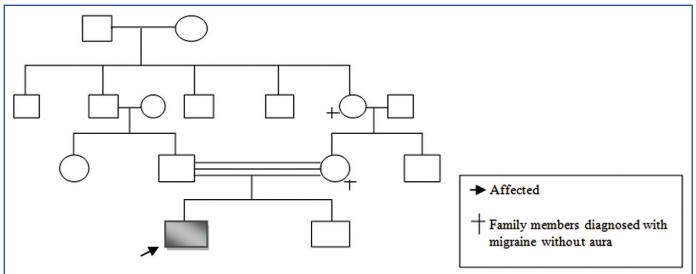
ABSTRACT

Sporadic Hemiplegic Migraine (SHM) is a rare subtype of migraine with recurrent attacks of reversible motor weakness alongside with visual, sensory and speech symptoms. It is classified as familial when a first- or second- degree relative has identical motor attack, and as sporadic when no such relative is affected. Patients with this subtype have been found to have a mutation in either *CACNA1A*, *ATP1A2* or *SCN1A*. In present case, a 13-year-old male, first born to a third degree consanguineous couple presented to the emergency department with severe headache lasting for an hour following which there was weakness in the right upper and lower limbs, blurring of vision in the left eye and deviation of angle of mouth to left followed by tonic movements of both upper limbs which lasted for five minutes. Child had frequent headaches for last six months and there was family history of migraine without motor involvement in mother and maternal grandmother. He was initially considered to have Transient Ischaemic Attack (TIA)/stroke. However, after initial stabilisation there was a dramatic neurological improvement within an hour of presentation hence the possibility of Hemiplegic Migraine (HM) was considered. Genetic testing was positive for *CACNA1A* mutation and hence the diagnosis of SHM was confirmed. The child was treated with Calcium Channel Blockers (CCB) and was put on aspirin prophylaxis. The child had no further episodes of hemiplegia post treatment, although there had been episodes of headaches with reduced intensity and duration. Parents were counselled regarding the possible prognosis and importance of follow-up for early identification of cerebellar signs and paroxysmal manifestations. Genetic counselling is recommended for future prenatal planning.

Keywords: Headache, Migraine with aura, Seizure, Stroke, Transient ischaemic attack, Weakness

CASE REPORT

A 13-year-old developmentally normal first born male of a third degree consanguineous couple was brought to the emergency department with complaints of severe headache lasting for an hour followed by weakness of right upper and lower limbs. Subsequently, tonic movement of both upper limbs were noted lasting for about five minutes. During the episode of headache, he also complained blurring of vision in the left eye and deviation of angle of mouth to the left. Parents did not give any history of trauma or fever. On further probing child had multiple episodes of severe headache in the past six months and were severe enough to affect day to day activities including school absence. But he never had motor weakness during these episodes. There was a strong family history of migraine in mother and maternal grandmother who are on treatment for last 10 years and two years, respectively. Similarly his younger brother also had history of recurrent headaches for a year but had not sought any medical advice. None of the above family members ever had any aura or motor weakness [Table/Fig-1].



[Table/Fig-1]: Family pedigree chart analysis.

At presentation, the child was irritable with a Glasgow Coma Scale score of 10/15. He was normothermic but exhibited tachycardia (heart rate: 120 beats/min), tachypnea (respiratory rate: 28 breaths/min), feeble peripheral pulses, cold extremities, and hypotension (blood pressure: 86/58 mmHg). In view of these findings, the child was considered to be in shock and was managed with appropriate fluid resuscitation using a normal saline bolus in the emergency department. Following initial stabilisation, he was transferred to the paediatric ICU for further management. Neurological examination revealed right-sided hemiparesis with an extensor plantar response on the right-side. Based on the clinical history and examination findings, differential diagnosis considered included TIA, acute ischaemic stroke, and seizure disorder. Empirical anti-oedema therapy with hypertonic saline (5 mL/kg administered over one hour) was initiated. The child showed marked clinical improvement within the next hour. The hemiparesis resolved within an hour and subsequent neurological examination was normal. There were no signs of meningeal irritation. Although the clinical semiology did not correspond to any specific seizure type, a seizure disorder could not be definitively excluded at that stage. Considering the child's past medical history and relevant family history, HM was also considered as a possible diagnosis.

All other routine investigations were within normal limits, including complete blood count, renal and liver function tests, and serum electrolytes (sodium, potassium, and calcium), except for a serum magnesium level of 1.6 mg/dL. In view of the possibility of seizures secondary to hypomagnesemia, intravenous magnesium was administered at a dose of 50 mg/kg. Electrocardiography demonstrated a mildly prolonged QTc interval, which normalised following magnesium correction. However, as the child returned to a neurologically normal state within one hour of presentation, prior to magnesium administration, the symptoms were not attributed to hypomagnesemia.

As child had hemiparesis with seizures, further workup was done with MRI with MRA, EEG, haemoglobin electrophoresis and prothrombotic markers, including protein C, protein S, and homocysteine levels, and all were within normal limits. In view of the unremarkable diagnostic workup and the rapid resolution of neurological deficits, genetic testing was performed, which revealed a CACNA1A mutation, consistent with hemiplegic migraine. In

the but mother and maternal grand mother were on treatment for migraine. The child was clinically diagnosed with sporadic hemiplegic migraine (SHM) [Table/Fig-2] [1].

Genetic testing	
Whole genome sequencing	*CACNA1A*
GENE (Transcript)	*CACNA1A*
Location	EXON 41
Variant	c.5975C>T(p.Pro1992Leu)
Zygosity	Heterozygous
Disease (OMIM)	Familial Hemiplegic Migraine (HM) -1
Inheritance	Autosomal dominant
Copy number variants	No significant CNVs

[Table/Fig-2]: Genetic testing reports.

The child was initially started on Flunarizine at 2.5 mg once daily regime as a preventive therapy and paracetamol 15 mg/kg was given for acute attacks. Child improved over the next six months but then had increased episodes of headache; hence, the dose of Flunarizine was increased to 5 mg per day. Sought paediatric neurologist opinion and was advised to add oral Aspirin prophylaxis (75 mg per day) DWS. Aspirin is generally avoided in children due to Reye's syndrome risk. While the text acknowledges this, it does not explicitly state that no active viral illness was present at the time of prescription — which is the primary trigger for Reye's syndrome with aspirin use. Additionally, the prescribing authority (paediatric neurologist) is mentioned but no accepted guideline or reference supporting aspirin prophylaxis for SHM in a 13-year-old is cited. After the aforementioned treatment, no further episodes of hemiplegia were seen until his recent follow-up a month ago. Although, he had mild episodes of headache occasionally which responded to NSAIDs. The child is currently on Flunarizine (5 mg per day) and Aspirin (75 mg per day) as advised by paediatric neurologist.

DISCUSSION

HM is a rare migraine subtype characterised by transient motor weakness (hemiparesis) with or without other aura symptoms (visual, sensory, aphasic, or brainstem) and typical migrainous features. Seizure-like presentation, as in this case, is uncommon. HM may be SHM or Familial Hemiplegic Migraine (FHM); SHM is diagnosed in the absence of a first- or second-degree relative with similar motor attacks, as in this child. However, as some apparently sporadic cases may later reveal affected relatives, the diagnosis may require revision [1].

HM is the only subtype of migraine in which monogenic mutations have been identified in the following genes: *CACNA1A*, *ATP1A2*, and *SCN1A* [1,2]. Among these mutations, variants of the Ca^{2+} channel gene- *CACNA1A* has emerged as a recognised aetiology of other rare neurological phenotypes such as chronic cerebellar dysfunction and variable paroxysmal manifestations which include migraine with hemiplegic aura, episodic ataxia, epilepsy and paroxysmal non-epileptic movement disorder [3]. For individuals with *CACNA1A*-FHM with frequent attacks, prophylactic migraine medications (e.g., tricyclic antidepressants, beta-blockers, CCB, anti-seizure medications) are routinely advised [4].

Despite its typically early onset, paediatric data on SHM remain limited. Informed parental consent was obtained for publication of this case report. Early transient neurological manifestations preceding HM- including febrile aphasia, isolated seizures, transient hemiparesis, and post-traumatic clumsiness- have been described [5-7]. SHM may also mimic acute meningoencephalitis or present with fluctuating focal deficits although these features were absent in the present case [8,9]. This patient presented with unilateral, stroke-like hemiplegia, consistent with prior reports [10-12]. In children, SHM is associated with more prolonged and severe attacks than familial forms, particularly early in the disease course [2]; however, attack frequency, severity, and duration typically decrease over time, as observed in this case [2-7].

The HM has to be considered in children with paroxysmal symptoms of hemiplegia, ataxia, seizures with or without other non-motor aura [6,7]. This can potentially avoid unwarranted investigations and an overtreatment. Despite no family history of hemiparetic episodes, a differential of HM was considered and a genetic study was done and found to have *CACNA1A* mutation confirming the diagnosis. Although previous case reports of adults with HM suggested a trial of acetazolamide [3,4] and Fremanezumab [5], our index child had good response to CCB (Flunarizine) without any further hemiplegic episodes. Parental education and appropriate counselling regarding symptomatology including early identification of aura and compliance to medications are of utmost priority as these episodes can have significant morbidity on child's day to day activities.

CONCLUSION(S)

The SHM should be considered in children presenting with acute hemiparesis and seizure-like episodes, especially when neuroimaging and other investigations are inconclusive. Early recognition and genetic testing can help differentiate it from stroke and avoid unnecessary interventions. Timely initiation of prophylactic therapy and appropriate counselling can significantly reduce morbidity and improve long-term outcomes.

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REFERENCES

[1] Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition. Cephalalgia. 2018;38(1):01-211.
[2] Bonemazzi I, Brunello F, Pin JN, Pecoraro M, Sartori S, Nosadini M, et al. Hemiplegic migraine in children and adolescents. J Clin Med. 2023;12(11):3783. [cited 2023 Nov 27].
[3] Indelicato E, Boesch S. CACNA1A-related channelopathies: Clinical manifestations and treatment options. Handb Exp Pharmacol. 2023;279;227-48.
[4] Villar-Martinez MD, Moreno-Ajona D, Goadsby PJ. Chapter 9 - Familial hemiplegic migraine. Handbook of Clinical Neurology. 2024;203:135-44.
[5] Hotz JF, Kaindl L, Krebs S, Vigl M, Ferrari J, Neumann C, et al. Reduced frequency of prolonged sporadic hemiplegic migraine attacks following Fremanezumab treatment-a case report. Eur J Neurol. 2026;33(2):e70514.
[6] Kana T, Mehjabeen S, Patel N, Kawami A, Shamim Z. Sporadic hemiplegic migraine. Cureus. 2023;15(5):e38930.
[7] Santhan S, Jehangir HM, Mugunthan. Sporadic hemiplegic migraine: A rare presentation in children. International Journal of Medical Research & Health Sciences [Internet]. 2025;3(2):445-47.
[8] Gajam S, Peterson RR, Mathew AA, Thomas A. Sporadic hemiplegic migraine with CACNA1A mutation masquerading as acute meningoencephalitis. Ann Indian Acad Neurol. 2022;25(3):528-29.
[9] Naddaf T, Ghotra G. Fluctuating neurological deficits in a patient with sporadic hemiplegic migraine. Cureus [Internet]. 2025;17(1):e78262. [cited 2025 Feb 22]. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC11872012/.
[10] Maita H, Kobayashi T, Akimoto T, Osawa H, Hanada H. Transient hemiplegia in a patient with migraine: A case of sporadic hemiplegic migraine. SAGE Open Med Case Rep. 2024;12:2050313X241275386.
[11] Kaushal R, Kashyap A, Yogesh S, Agarwal M, Banerjee I. A rare case of sporadic hemiplegic migraine mimicking stroke: A diagnostic challenge solved by comprehensive history taking. Cureus. 2024;16(4):e57790.
[12] Luchia KC, Lajoie J. Unmasking hemiplegic migraine: A diagnostic dilemma in the shadow of stroke. Cureus. 2025;17(10):e95541. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC12657268/.

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