Case Report

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A Rare Case of Complicated Migraine: A Case Study

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ABSTRACT

This is a case study on 23year old female having severe unilateral headache with tinnitus, dysguesia and hemiplegia, who was initially misdiagnosed as dissociative disorder. Detailed history and evaluation reported it to be a case of complicated migraine with neurological manifestations. Start of migraine prophylaxis showed significant improvement in symptoms.

Abbreviations: CSD: Cortical Spreading Depression; ICHD2: International Classification of Headache Disorders 2; HM: Hemiplegic Migraine

Introduction

As per Global Burden of Disease 2015, Headache disorders were collectively the third leading cause of disability in people under 50 years of age. Even though, migrane is second most common type of headache following tension- type heachadche, the burden of migraine is much higher affecting over a billion of people worldwide. (Stovner [1]) The word "migraine" is derived from the Greek word hemikrania, which was later converted into Latin as hemigrane. Migraine is a cyclic disorder which has different phases, including a premonitory phase, transient neurological symptoms (i.e., migraine aura), intense headache attack and postdrome phase. Migraine can be classified into two types- migraine with aura and migraine without aura. (Jatin Gupta, et al. [2]). International Classification of Headache Disorders 2 [ICHD2], 2004, the classified aura into four types:

1. Visual aura, the most common;
2. Sensory aura;
3. Language aura, which occur less commonly;
4. Motor aura, the least common. (Foroozan, et al. [3])

Case Report

A 23year old female studying abroad came back to India with complaints of severe headache, she reported she had difficulty in concentrating in studies, would not come out of the room, keep the curtains closed, away from any light and sounds. It was also accompanied by presence of nausea and floaters. These episodes lasted for almost 3 days. She had taken acetaminophen without any specialist consultation, reported mild relieve in symptoms. After almost 20

days, she first developed difficulty in breathing and chest pain lasting for almost 1 hour, it was followed by paresthesia throughout body and hemiparesis of left side for almost 30 minutes. Pulsating headache on right side including orbital region continued throughout the day. Next day morning, along with the symptoms of previous day she had tinnitus and dysgeusia, which was followed by weakness of one side of body resulting difficulty bearing weight, developed ptosis and swelling on one side of face. Patient was first admitted in the tertiary hospital where the history of the patient revealed stressors and she was diagnosed with dissociative disorder, was started on antidepressant and anxiolytics. She reported no improvement in the symptoms, her conditions continued to worsen over next 5 days. Following this, she was brought to our hospital, where detailed examination was done.

Past history revealed history of difficulty breathing, followed by episodes of loss of consciousness. This was followed by unilateral pulsating headache associated with nausea and vomiting. She was started on Prothiaden 25mg and Flunarizine 10mg. she took medication for almost 3-4 months. And was asymptomatic for 1 year. After 1 year, again she developed similar complaints. General physical examination revealed reduced power, nystagmus and ptosis. Complete hemogram was normal. Electrocardiography was normal. Electroencephalography showed lower alpha and beta activity. MRI brain showed no significant changes. Case discussion with panel of consultants was done and she was diagnosed with complicated migraine with neurological manifestations. She was started on intravenous valproate 250mg twice daily and flunarizine 10mg at night. Combination of Magnesium and riboflavin was also started. Patient started to report improvement within 48 hours. Sumatriptan nasal spray was prescribed, on need basis in case headache develops. Patient was completely asymptomatic within a week. But she continued to have dysguesia for almost 1 month. She is coming for follow up for past three months, with no further episode.

Discussion

The International Headache Society identifies motor aura as a hallmark feature of hemiplegic migraine (HM), a specific migraine subtype characterized by reversible motor weakness. While motor aura is usually temporary, rare cases exhibit prolonged or even permanent symptoms, leading to lasting neurological impairments, progressive cognitive decline, and significant brain MRI changes following HM episodes. These changes may include a progressive pattern of brain atrophy. (Arnold [4]) The activation of the trigeminovascular system is considered the most common neurobiological mechanism underlying migraine. This activation may result from cortical spreading depression (CSD) and dysfunction in brainstem nuclei involved in central pain regulation. Genetics also play a crucial role, particularly gain-of-function mutations in the SCN1A gene, which contribute to neuronal hyperexcitability and prolonged depolarization linked to CSD. (Cest`ele, et al. [5]) The sustained depolarization of inhibitory neurons leads to chloride ion depletion and extracellular potassium accumulation, disrupting neuronal equilibrium. This imbalance may trigger widespread neuronal depolarization, excessive glutamate release, and the onset of CSD. Prolonged aura and CSD can increase metabolic demand, potentially causing cortical swelling, contrast enhancement, and blood–brain barrier dysfunction. (Charles, et al. [6]) (Pietrobon, et al. [7])

The transient cortical enhancement observed on MRI in these cases may be indicative of a disrupted blood–brain barrier—an uncommon finding in hemiplegic migraine (HM) that could be linked to the severity of the attack. The irreversible changes in such patients may be attributed to cytotoxic edema, representing an extreme manifestation of CSD, ultimately leading to cell death. No evidence of any specific medication for acute or preventive therapies is available. Only past case reports have shown the benefits of using nasal ketamine, intravenous furosemide, magnesium, and verapamil for acute headache and aura treatment. (Di Stefano, et al. [8]) A case study has shown potential benefits of valproate in such cases. Our patient also benefited from the use of valproic acid and flunarizine. (Jokubaitis, et al. [9])

Conclusion

Motor aura being very uncommon can be misdiagnosed, but before reaching the diagnosis of motor aura all the other possible causes of hemiplegia should be ruled out. Timely management would result in prevention of permanent damage in brain.

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