



“Perceiving myself small: a body volume regression to age three” - a new manifestation of Alice in Wonderland Syndrome in a migraine patient

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Introduction

Alice in Wonderland Syndrome (AIWS) is a rare neurological condition often associated with migraine and characterized by perceptual distortions of body image, time, and space.

Case report

We report the case of a 33-year-old woman with a history of migraine since adolescence who presented with recurrent episodes of AIWS. During severe migraine attacks, she experienced microsomatognosia, perceiving her body as shrinking to the size of a child when her eyes were closed, along with temporal distortion described as “slow motion” of her surroundings. Neurological examination, brain MRI, and angiography were unremarkable. Despite prophylactic therapy with topiramate and acute treatment with triptans, disabling attacks persisted.

Conclusion

This case highlights the complex relationship between migraine and AIWS, reinforcing the need for clinicians to recognize AIWS manifestations as part of the migraine spectrum. Early identification can prevent misdiagnosis and improve patient management.

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Introduction

Alice in Wonderland Syndrome (AIWS) is a rare neurological disorder characterized by perceptual disturbances involving vision, somatosensory processing, and temporal perception (1–5). Patients may experience distortions in the size or shape of objects (macropsia, micropsia, dysmorphopsia) (6,7), altered perception of their own body (macrosomatognosia and microsomatognosia) (8), temporal distortions (a sense of acceleration or deceleration of time) (2,3,6,7), as well as phenomena of derealization and depersonalization (8,9). These manifestations are usually transient, occurring in the prodromal phase or during the headache attack itself, and may have a significant impact on functional and emotional well-being during episodes (9).

Alice in Wonderland Syndrome has been associated with various clinical conditions. Reported causes include viral infections (10,11), cerebrovascular diseases (5), epilepsy (11,12), brain lesions, the use of medications (13,14), and psychiatric disorders (4). In adults, however, the most frequently documented associations are with migraine and epilepsy, with migraine considered the main triggering factor (15–18). Recent studies reinforce a close link between AIWS and migraine with aura, demonstrating a higher prevalence of AIWS symptoms in patients with aura compared to those without (1,5,17,19–21). Furthermore, AIWS episodes tend to occur within the first hour of migraine onset and are more common in individuals who present visual or somatosensory symptoms as part of their aura.

The pathophysiology of AIWS remains incompletely understood. Functional neuroimaging evidence suggests that both AIWS and migraine with aura share alterations in thalamic connectivity, although these changes appear to be more diffuse and profound in AIWS (19,20). MRI studies have shown increased connectivity between the visual cortex (area V3) and the posterior superior temporal sulcus, a region involved in multisensory integration, suggesting that AIWS may represent a complex form of migraine aura involving associative and multisensory brain areas (19,20). This finding supports the hypothesis that cortical spreading depression, the neural correlate of migraine aura, may extend to regions responsible for the characteristic manifestations of the syndrome.

In the International Classification of Headache Disorders, 3rd edition (ICHD-3), AIWS is not included among the main aura chapters but is listed in the appendix as a rare entity with diagnostic criteria that have not yet been definitively established (22). Nevertheless, its strong association with migraine suggests that it represents a spectrum of perceptual manifestations that, in specific contexts, may be considered variants of aura.

The objective of this article is to report one case of an adult patient with migraine who presented episodes consistent

with AIWS, and to discuss their clinical manifestations and diagnostic implications.

Case Report

Woman, 33 years old, with a history of migraine since the age of 15, with no personal history of epilepsy or other relevant neurological conditions. Approximately four weeks ago, she presented severe headache attacks (8/10 on the numerical pain scale), accompanied by perceptual distortions compatible with Alice in Wonderland Syndrome. During the episodes, the patient reported somesthetic changes in the form of microsomatognosia, with a sensation that her body decreased in size when she closed her eyes, describing the perception of herself “like a child” (Perceiving myself small: A body volume regression to age three). When she opened his eyes, she reported that the body returned to its usual size, but perceived the surrounding environment in “slow motion”, with slowing down of people’s movements and distorted sounds. The symptoms occurred concomitantly with the painful phase of the migraine and persisted throughout the period of the attack.

Due to severe migraine pain, she has a history of going to the emergency room on two occasions and was hospitalized due to intense migraine attacks. Neurological examination revealed no abnormalities. Brain magnetic resonance imaging (MRI) and MRI angiography showed normal findings, with no correlation between the visual phenomena described and any abnormal cortical alteration.

The patient was already on continuous use of topiramate 100 mg/day, bupropion 150 mg/day, and quetiapine 25 mg/day. For acute crises, she used anti-inflammatory drugs and triptans on demand, with medication overused headache. Despite the ongoing prophylactic treatment, she reported persistence of disabling migraine episodes accompanied by the perceptual distortions described.

Discussion

The present case highlights an unusual manifestation of AIWS in a patient with migraine: the perception of being “small like a child” occurring exclusively when she closed her eyes, while her body size perception normalized upon eye opening. To our knowledge, microsomatognosia limited to the eyes-closed condition has not been previously reported in the literature, making this a novel variant of AIWS. This phenomenon suggests a possible disruption in multisensory integration between somesthetic processing and visual feedback, which has been linked to



the temporoparietal junction, secondary somatosensory cortex, and posterior insula in functional imaging studies. The case also underscores the importance of considering pharmacological influences. Topiramate, a common prophylactic for migraine, has been associated in case reports with perceptual disturbances including palinopsia and AIWS-like symptoms, with resolution after dose reduction or drug withdrawal (23,24). In our patient, the chronic use of high-dose topiramate raises the possibility that the drug acted as a modulatory factor, although migraine remains the primary predisposing condition. The challenge for clinicians is to determine whether these perceptual phenomena reflect the underlying disorder or an adverse drug reaction.

Differential diagnosis must always be taken into account. Although neuroimaging in this patient was normal, AIWS has occasionally been reported as the initial manifestation of structural lesions, including temporo-occipital glioblastoma (5). Such cases emphasize the importance of neuroimaging in atypical or refractory presentations to exclude serious secondary causes.

Finally, the overlap between migraine aura and AIWS remains debated. In many patients, episodes occur within the same temporal window as migraine attacks and share visual and somatosensory features. The unique eyes-closed microsomatognosia in this patient may represent a rare and complex variant of aura, extending beyond the traditional visual symptoms and involving higher-order multisensory networks.

Taken together, this case illustrates the clinical heterogeneity of AIWS in migraine and highlights a previously undescribed presentation, in which body image distortion emerged only when visual input was absent. It also reinforces the need to consider drug effects such as topiramate, and to remain vigilant for red flags that warrant a broader diagnostic evaluation.

Conclusion

This case highlights a novel presentation of AIWS in migraine, characterized by eyes-closed microsomatognosia, which has not been previously reported in the literature. The phenomenon suggests a disruption in multisensory integration between somatosensory and visual processing, possibly involving associative cortical regions. Clinicians should remain alert to such atypical manifestations, consider drug-related influences such as topiramate, and rule out secondary causes through neuroimaging. This report expands the clinical spectrum of AIWS and reinforces its close link with migraine, illustrating how perceptual distortions may occur beyond traditional aura symptoms. Recognizing these rare presentations is essential for accurate diagnosis, patient reassurance, and appropriate management strategies.

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