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REVIEW ARTICLE

THE NEUROLOGICAL PHENOMENON OF ALICE IN WONDERLAND SYNDROME

NEUROLOGICZNE ZJAWISKO ZESPOŁU ALICJI W KRAINIE CZARÓW

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ABSTRACT

Introduction

Alice in Wonderland Syndrome (AIWS) is a rare neurological disorder characterized by distortions in the perception of size, time, and spatial relationships. Named after Lewis Carroll's novel, the condition has been known since the 1950s, yet its underlying mechanisms remain incompletely understood. AIWS is frequently associated with migraine, epilepsy, and viral infections.

Aim

The aim of this paper was to provide a comprehensive review of AIWS, including its pathophysiology, clinical manifestations, potential causes, diagnostic approaches, and treatment strategies.

Material and methods

This review is based on scientific literature, including 17 epidemiological studies and case reports. It also incorporates neuroimaging data and symptom assessment scales.

Results

The most common symptoms of AIWS include micropsia and macropsia, altered time perception, derealization, and depersonalization. Micropsia was reported in 73% of cases. The condition is more commonly diagnosed in children, with a mean onset age of 8.3 years. Migraines were present in 90% of patients. Treatment involves pharmacotherapy (e.g., triptans, antiepileptics, SSRIs) and non-pharmacological methods, such as cognitive-behavioral therapy.

Conclusions

AIWS is a complex disorder that requires further research to better understand its pathophysiology and optimize treatment strategies. Neuroimaging studies suggest the involvement of occipital and temporal lobes and dysregulation of neurotransmitter systems, including serotonin, dopamine, and GABA. Due to the variability and transient nature of symptoms, AIWS is often underdiagnosed. Effective management should address the underlying condition – most commonly migraine – and include psychological support when psychiatric comorbidities are present.

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STRESZCZENIE

Wstęp

Zespół Alicji w Krainie Czarów (AIWS) to rzadkie zaburzenie neurologiczne objawiające się zniekształceniem percepcji wielkości, czasu i przestrzeni. Nazwa pochodzi od powieści Lewisa Carrolla. Choć zaburzenie to znane jest od lat 50., mechanizmy jego powstawania nadal nie są w pełni poznane. Zaburzenie często współwystępuje z migreną, padaczką oraz infekcjami wirusowymi.

Cel

Celem pracy było kompleksowe omówienie AIWS – jego patofizjologii, objawów klinicznych, możliwych przyczyn, diagnostyki oraz strategii leczenia.

Materiał i metody

W pracy dokonano przeglądu literatury naukowej, analizując wyniki 17 badań epidemiologicznych, studiów przypadków i artykułów. Uwzględniono także dane neuroobrazowe i wyniki skali oceny objawów.

Wyniki

Najczęstszymi objawami AIWS są mikropsje i makropsje (zaburzenia percepcji wielkości), zniekształcenia czasu, derealizacja i depersonalizacja. U 73% pacjentów stwierdzono mikropsję. AIWS częściej występuje u dzieci (średni wiek zachorowania: 8,3 lata). Najczęstszym czynnikiem etiologicznym są migreny (90% przypadków). W leczeniu stosuje się farmakoterapię (np. tryptany, leki przeciwpadaczkowe, SSRI) oraz podejścia niefarmakologiczne, jak terapia poznawczo-behawioralna.

Wnioski

AIWS to złożone zaburzenie wymagające dalszych badań w celu pełniejszego zrozumienia mechanizmów jego powstawania i opracowania skuteczniejszych metod terapii. Badania neuroobrazowe sugerują udział płatów potylicznych i skroniowych oraz zaburzenia w układach neuroprzekaźników, takich jak serotonina, dopamina czy GABA. Ze względu na różnorodność objawów i ich przejściowy charakter, AIWS bywa trudne do rozpoznania i często pozostaje niezdiagnozowane. Skuteczne leczenie wymaga identyfikacji i terapii choroby podstawowej – najczęściej migreny – oraz podejść psychoterapeutycznych w przypadku współistniejących zaburzeń lękowych lub depresyjnych.

Słowa kluczowe: migrena, neuropsychologia, zespół Alicji w Krainie Czarów, zaburzenia percepcji, mikropsja

Background

Alice in Wonderland Syndrome (AIWS) is a neurological condition characterized by distorted perception of size, time, and spatial relationships. Named after Lewis Carroll's novel, the syndrome can manifest in various ways, leading to significant impairment in daily functioning. This paper provides an extensive overview of AIWS, including its pathophysiology, clinical manifestations, potential etiologies, and management strategies. Quantitative data from various studies will be incorporated to highlight the syndrome's prevalence and demographic variations.

Introduction

Alice in Wonderland Syndrome (AIWS) is a complex neurological disorder characterized by transient perceptual distortions affecting sensory integration, body schema, and reality interpretation. First systematically described and named by John Todd in 1955 (Todd, 1955), the syndrome draws inspiration from Lewis Carroll's *Alice's Adventures in Wonderland*, whose protagonist experiences bodily and environmental size distortions mirroring patient reports. Carroll himself suffered from migraines with aura, suggesting potential personal insight into these phenomena (Podoll & Robinson, 1999; Blom, 2016). Earlier clinical observations of similar distortions, particularly in migraineurs, were documented by Lippman (1952).

Clinically, AIWS manifests through distinct symptom clusters: **Type A (Somaesthetic distortions)** including micro/macrosomatognosia (altered body part size), depersonalization, and altered time perception; **Type B (Visual distortions)** such as micro/macropsia (objects shrinking/enlarging), teleopsia/pelopsia (altered depth perception), and metamorphopsia (shape distortion); and **Type C** involving combined A and B symptoms (Lanska & Lanska, 2013; Mastria *et al.,* 2016). These episodes typically last minutes to hours, with a significant proportion (65.1%) reporting a temporal association with headaches (Liu *et al.,* 2014). While migraines are a frequent association, AIWS also arises from infections (notably Epstein-Barr virus, implicated in 68.4% of pediatric cases) (Abe *et al.,* 2018), epilepsy, strokes, or psychoactive substances (Blom, 2016). Neuroanatomically, dysfunction at the temporo-parieto-occipital junction (TPO-C), a critical hub for multisensory integration, is strongly implicated in symptom generation (Lanska & Lanska, 2013; Mastria *et al.,* 2016). Incidence remains difficult to ascertain due to diagnostic ambiguities and underreporting, but studies suggest a non-trivial prevalence: lifetime AIWS symptoms occur in 16.5% of migraine patients (Fitzek *et al.,* 2024) micropsia/macropsia specifically affect 6.5% of male and 7.3% of female adolescents (Weidenfeld & Borusiak, 2011), and only approximately 200 severe cases requiring medical intervention were documented between 1955 and 2016 (Blom, 2016).

Clinical manifestations

Symptomatology

The symptoms of AIWS can vary significantly among individuals, but commonly reported features include micropsia and macropsia – distortions in the perceived size of objects, leading to feelings of objects being smaller (micropsia) or larger (macropsia) than they actually are. A study by Fitzek *et al.,* (2024) found that 73% of patients reported micropsia as a primary symptom. Also, temporal distortion, which is altered perception of time, where time may feel accelerated or decelerated. This symptom is frequently reported in conjunction with visual distortions. The AIWS is characterised by derealization and depersonalization which often describe feelings of detachment from their environment or their own bodies, which can be distressing and contribute to anxiety. It is also worth mentioning other sensory distortions – individuals may experience altered perception of sound and touch, although these symptoms are less frequently reported.

The Mastria *et al.* (2016) classification system categorizes AIWS into three subtypes: Type A (somaesthetic distortions: 9% of cases), Type B (visual distortions: 75%), and Type C (mixed symptoms: 16%). This framework clarifies that visual distortions alone (Type B) represent the most common presentation, necessitating broader diagnostic criteria beyond somaesthetic symptoms. The numerical data they used came from previous studies (Lanska & Lanska, 2013).

Prevalence and demographics

There are no definitive prevalence estimates for Alice in Wonderland Syndrome (AIWS) in the general population reported in the current literature. However, one method of

approximation involves extrapolating from migraine data, as AIWS symptoms – particularly visual distortions such as micropsia and macropsia – are frequently associated with migraine, especially with aura. Migraine affects roughly 12% of the global population (Dong *et al.,* 2024). A recent large-scale study by Fitzek *et al.* (2024) reports a 16.5% lifetime prevalence of AIWS in adult migraine patients, with significantly higher rates in migraine-with-aura (19.5%) versus without aura (14.1%). Micropsia/telopsia (72.9%) and macrosomatognosia (49.6%) dominate symptom profiles, typically lasting ~30 minutes. Extrapolating from Fitzek *et al.* (2024), AIWS prevalence in migraineurs is 16.5%. Applied to the global migraine population (12%), this suggests a general population prevalence of ~2%, though true rates may differ significantly when other extrapolating approach is used. Considering that some publications report only 27% of AIWS cases involving migraines prior to diagnosis (Blom, 2016) (rather than 90% as in Mastria *et al.,* 2020), the higher estimate of the AIWS rates would be considered more likely. The condition is more commonly observed in children, particularly between the ages of 5 and 12 years. AIWS is likely underdiagnosed because patients (especially children) may fear stigma or lack vocabulary to describe bizarre symptoms. Clinicians might misinterpret symptoms as psychiatric (e.g., psychosis) without careful history-taking. This stresses the need for direct questioning about perceptual distortions (size, time, body image) in patients with migraines (especially with aura), epilepsy, or viral illnesses especially if accounting for Blom's finding that only 27% had prior migraine diagnosis.

Etiologies

Migraine-related AIWS

Migraine is the most common association with AIWS, with approximately 90% of patients reporting a history of migraine attacks (Mastria *et al.,* 2020). The mechanism underlying this association may involve shared neurobiological pathways, including cortical excitability and the modulation of neurotransmitter systems.

Other neurological conditions

AIWS can also occur in conjunction with other neurological disorders, for example epilepsy, which is shown in mentioned study that temporal lobe epilepsy has been linked to episodes of AIWS, particularly during ictal or postictal states (Matsudaira *et al.,* 2020).

Although primarily a neurological phenomenon, AIWS can coexist with psychiatric conditions, particularly anxiety and depression.

Stroke is an emerging etiology, with a 2025 case study linking macropsia to right parietal and left occipital chronic infarcts. Symptoms resolved with low-dose quetiapine, implicating vascular dysfunction in TPO-C pathways (Mbizvo *et al.,* 2025).

When it comes to neurodegenerative disorders, the first case of AIWS in dementia with Lewy bodies (DLB) (2025) reported object-size agnosia (micropsia) linked to right extrastriate cortex dysfunction, highlighting the role of visual associative areas in perception distortions (Demas, 2025).

Infections

Infectious diseases may also have an influence, because Conditions such as Epstein-Barr virus infections have been reported in some older cases of AIWS, suggesting a potential viral etiology (Ho *et al.,* 1992). More recently other infectious triggers were discovered. These include Zika virus and COVID-19, particularly in paediatric populations. These associations suggest viral-induced neuroinflammation disrupts TPO-C sensory integration (Brooks *et al.,* 2019; Hossain, 2020).

Implications for diagnostics

It is highly important to rule out acute neurological emergencies (e.g., stroke, encephalitis, brain tumours) and psychiatric conditions (e.g., schizophrenia, depersonalization-derealization disorder) using appropriate investigations (neuroimaging, EEG, labs) to avoid misdiagnosis. Certain features warrant urgent investigation.

These include: new-onset in adults, persistent/progressive symptoms, associated focal neurological deficits, altered consciousness or fever.

Pathophysiology

Neurological mechanisms

The pathophysiology of AIWS remains poorly elucidated, but several hypotheses have been proposed. Neuroimaging studies indicate that altered activity in the occipital and temporal lobes may contribute to the syndrome's symptoms. The visual processing areas, particularly the primary visual cortex (V1) and surrounding regions, are thought to be involved in the distorted perception of size and shape (Blom, 2016; Landais & Michelin, 2019).

Recent advances in neuroimaging (2020–2025) identify the temporoparietal-occipital carrefour (TPO-C) as a critical neural hub for AIWS. Hypoperfusion in this region – observed via SPECT and fMRI – disrupts multisensory integration, explaining the co-occurrence of dysmetropsia and body schema distortions (Mastria *et al.,* 2023; Mbizvo *et al.,* 2025). Cortical spreading depression (CSD), the mechanism underlying migraine aura, is directly linked to AIWS through biparietal hypoperfusion patterns during episodes, reinforcing shared pathophysiological pathways with migraine (Viganò *et al.,* 2020; Mastria *et al.,* 2023).

Cortical dysregulation

Alterations in cortical excitability, particularly in response to migraine-induced changes, have been observed. In individuals with migraines, there is evidence of increased cortical spreading depression, which may lead to transient disruptions in visual perception (Charles & Baca, 2013). The involvement of the temporal lobe is also significant, as this region is crucial for integrating sensory information.

Neurotransmitter systems

Several neurotransmitter systems may play a role in the manifestation of AIWS symptoms. Dopaminergic dysregulation has been implicated in perceptual disturbances, while serotonergic pathways may contribute to alterations in mood and cognition (Beh *et al.,* 2018). Additionally, disturbances in the gamma-aminobutyric acid (GABA) system may influence sensory processing and perception. Critically, dopaminergic/serotonergic dysregulation may modulate TPO-C excitability, potentiating CSD-induced perceptual disruptions (Beh *et al.,* 2018; Sprenger *et al.,* 2018).

Quantitative analysis

Epidemiological studies

Based on 169 case descriptions Blom, J. D. (2016) revealed that approximately 27.1% of patients had a history of migraines. The mean age of symptom onset was found to be 15.5 years, with a male-to-female ratio of approximately 1.25 : 1. In terms of symptom frequency, 58.6% of patients experienced micropsia and 45.0% experienced macropsia, indicating that these were the most commonly reported perceptual distortions. Epidemiological surveys in non-clinical populations also suggested that up to 30.3% reported at least one AIWS-like symptom during their lifetime, with some studies documenting 6-month prevalence rates for specific symptoms ranging from 1.3% to 3.9% among adolescents. Most recently, Fitzek *et al.* (2024) identified micropsia/telopsia as the most prevalent visual distortion (72.9%), followed by macrosomatognosia (49.6%), with episodes averaging 30 minutes – suggesting transient but clinically impactful disruptions.

Correlations with migraines

Research suggests a strong association between Alice in Wonderland Syndrome (AIWS) and migraines. A study by Liu *et al.* (2014) found that paediatric patients with AIWS often experienced more frequent migraines, with some reporting perceptual distortions before or during attacks. Similarly, Lanska & Lanska (2013) noted that AIWS symptoms frequently co-occurred with migraines, supporting the idea of shared pathophysiological mechanisms. One proposed explanation involves Cortical Spreading Depression (CSD), a wave of neuronal depolarization linked to migraine

aura (Hadjikhani *et al.,* 2001). CSD may trigger transient perceptual disturbances resembling AIWS, while dysregulation in serotonin and dopamine pathways – known to influence migraines – could further contribute to these phenomena.

Diagnostic criteria

Clinical assessment

The diagnosis of AIWS is primarily clinical, relying on detailed patient history and symptomatology. Neurological examinations, including neuroimaging and electrophysiological studies, are often utilized to rule out other conditions.

Diagnostic tools

Standardized scales for measuring the severity and frequency of symptoms, such as the Migraine Disability Assessment Scale (MIDAS) (Stewart *et al.,* 2001) and the Hamilton Anxiety Rating Scale (HAM-A), can aid in assessing the impact of AIWS on daily functioning. For providing an example, the MIDAS is in in the form of a questionnaire – people are asked to fill out the paper and then their answers are summed up as follows:

Table 1. MIDAS scale (Stewart *et al.*, 2001).

MIDAS grade	Level of disability	MIDAS score
First	Little or no disability	0–5
Second	Mild disability	6–10
Third	Moderate disability	11–20
Fourth	Severe disability	21+

If the MIDAS score is higher than 6, the person should visit a doctor.

Management strategies

Management is primarily targeted at the underlying cause (migraine prophylaxis, epilepsy control, treating infection). Success therein often resolves AIWS.

Tiered approach

Suggested is a practical framework to follow in the management of the condition:

1. Rule out serious pathology.
2. Identify and treat primary trigger (Through: migraine, epilepsy, infection and psychiatric causes).
3. Add symptom-specific support: Pharmacological (if linked to primary trigger and severe) and Non-pharmacological (CBT/mindfulness for anxiety/coping, patient/family education) as first-line for distress.

Pharmacological interventions

Pharmacological management of AIWS primarily focuses on treating underlying conditions, such as migraines. There are some common medications involved in AIWS treatment. Triptans are first-line treatment for migraines and cluster headaches (Lew & Punnapuzha, 2023). They are effective for acute migraine relief, these medications can reduce the frequency of AIWS episodes in migraine sufferers. People use antidepressants as well, such as selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants which may alleviate anxiety and mood disturbances associated with AIWS and antiepileptics meaning medications such as lamotrigine and topiramate have shown promise in managing both epilepsy and associated perceptual disturbances (Sprenger *et al.,* 2018). Lacosamide shows efficacy in AIWS triggered by cortical venous thrombosis, while low-dose quetiapine resolves macropsia in post-stroke cases (Mbizvo *et al.,* 2025).

Non-pharmacological approaches

Cognitive behavioural therapy (CBT) represents a valuable psychological intervention for managing the distress and perceptual abnormalities associated with Alice in Wonderland Syndrome (AIWS). While AIWS itself is not

life-threatening, the profound perceptual distortions frequently induce significant anxiety, confusion, and distress. CBT, a structured and goal-oriented psychotherapy, equips individuals with AIWS to reframe maladaptive thoughts and behaviours triggered by these distortions. Specifically, cognitive restructuring helps patients challenge catastrophic interpretations of their experiences, while exposure techniques facilitate gradual adaptation to altered perceptions (Blom, 2016; O'Toole & Modestino, 2017). By developing these coping strategies, CBT reduces the emotional impact of symptoms and enhances emotional resilience. Furthermore, CBT effectively addresses common comorbidities like anxiety disorders and migraines, which often exacerbate AIWS symptoms (Hamed, 2010; Losada-Del Pozo *et al.,* 2011). Although CBT cannot eliminate the core perceptual distortions, its capacity to significantly reduce distress and improve quality of life is particularly crucial given the current lack of specific pharmaceutical treatments for AIWS. Treatment typically involves regular sessions, homework for skill practice, and a focus on identifying and modifying dysfunctional cognitions. While further research is needed to establish its precise efficacy for AIWS specifically, evidence supporting CBT for analogous perceptual and anxiety disturbances underscores its promise for managing the psychological sequelae of this syndrome.

Functional impact and quality of life

Alice in Wonderland Syndrome (AIWS) imposes significant functional impairment and reduced quality of life. In paediatric populations, educational performance is frequently disrupted by micropsia (Fitzek *et al.,* 2024) and spatial distortions (Liu *et al.,* 2014), with children avoiding classroom activities due to distress. Adults report occupational disability, including diminished work efficiency and safety risks during driving or machinery operation (Mastria *et al.,* 2020). Psychosocial burden is profound: >80% of patients experience anxiety about symptom misinterpretation as psychiatric illness, leading to social withdrawal (Mastria *et al.,* 2020; Beh *et al.,* 2018). Physical safety is compromised by falls from distorted depth perception (Fitzek *et al.,* 2024) and sleep disruption from nocturnal derealization (Landais & Michelin, 2019). Diagnostic delays exacerbate distress, with patients reporting inadequate validation of experiences (Blom, 2016). Standardized metrics confirm severe disability: migraine-associated AIWS patients score >21 on the MIDAS (indicating "severe disability") (Stewart *et al.,* 2001), exceeding typical migraine disability due to compounded distress from both headache and reality distortion (Fitzek *et al.,* 2024), while comorbid depression (present in 31% of vestibular migraine-related cases) amplifies QoL erosion (Beh *et al.,* 2018). Non-pharmacological interventions like CBT show promise in mitigating anxiety (Curtiss *et al.,* 2021), yet residual functional deficits often persist despite pharmacological management of underlying etiologies (Sprenger *et al.,* 2018).

Conclusion

AIWS represents a critical intersection of neurology and psychiatry, where transient but distressing perceptual distortions significantly impair quality of life despite the syndrome's non-life-threatening nature. The condition demonstrates distinct etiological patterns across age groups, with migraines constituting a primary trigger in adults while infectious processes frequently precipitate paediatric cases (Blom, 2016; Mastria *et al.,* 2016). Neuroanatomically, dysfunction within the temporo-parieto-occipital junction (TPO-C) provides the most compelling explanation for the syndrome's heterogeneous manifestations – from somaesthetic illusions to environmental dysmetropsia – given this region's role in multisensory integration (Lanska & Lanska, 2013; Mastria *et al.,* 2016).

Therapeutic emphasis must prioritize cognitive-behavioural therapy (CBT) as a cornerstone intervention for mitigating AIWS-related distress. By targeting catastrophic misinterpretations of perceptual anomalies

(e.g., "my shrinking hand indicates neurological deterioration") and comorbid anxiety, CBT builds essential emotional resilience in the absence of syndrome-specific pharmacotherapies (O'Toole & Modestino, 2017; Blom, 2016). Exposure techniques promote habituation to distortions, while cognitive restructuring disrupts symptom-triggered panic cycles (O'Toole & Modestino, 2017). Nevertheless, the evidence base remains constrained by limited AIWS-specific CBT studies, underscoring the need for randomized controlled trials. Efficacy data for both pharmacological and non-pharmacological interventions remain disproportionately scarce in paediatric populations, warranting age-stratified clinical trials.

Critical priorities for advancing the field include: 1) **Standardizing diagnostic criteria** to reduce reliance on subjective clinical recognition (Blom, 2016; Mastria *et al.,* 2016); 2) **Validating neuroimaging biomarkers** (e.g., temporal lobe perfusion deficits) to objectivize diagnosis (Mastria *et al.,* 2016); and 3) **Designing integrated protocols** that concurrently address primary pathologies (e.g., migraines) and perceptual distress. As Mastria *et al.* (2016) observed, AIWS epitomizes how "pathologies of sensory integration" necessitate multidisciplinary collaboration – uniting neurology, psychology, and patient education to effectively demystify this complex condition.

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Acknowledgments
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