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Alice in Wonderland Syndrome - A Literature Review

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Abstract

Introduction:
Alice in Wonderland syndrome (AIWS), also known as Todd's syndrome, is a rare perceptual disorder characterized by disturbances in visual perception, body schema, and time experience. First described in 1955, it reflects phenomena similar to those depicted in *Alice's Adventures in Wonderland*. Common symptoms include micropsia, macropsia, and altered perception of one's own body, often accompanied by derealization and temporal distortion. AIWS is most frequently associated with migraine, infections, and epilepsy, and appears more common in children. Despite its characteristic clinical presentation, it remains underdiagnosed and lacks standardized diagnostic criteria.

Aim:
The aim of this narrative review is to summarize current knowledge on AIWS based on literature published between 2015 and 2026, with reference to earlier key studies on diagnostic criteria. The paper focuses on clinical features, pathophysiology, and associated conditions, while identifying gaps for future research.

Materials and Methods:
A literature search was conducted using PubMed, Scopus, Web of Science, ResearchGate, and Google Scholar. Keywords included "AIWS," "micropsia," "macropsia," "migraine," and "AIWS neuroimaging." Studies from 2015–2026 were prioritized, along with a key 2013 study on diagnostic criteria. Both clinical and neurobiological data were analyzed.

Conclusion:
AIWS is a heterogeneous condition likely resulting from dysfunction of distributed brain networks responsible for multisensory integration rather than focal lesions. Various etiologies, including migraine, infections, and epilepsy, appear to converge on common neural pathways. Treatment should target the underlying disorder, often leading to symptom improvement. However, lack of clear diagnostic criteria contributes to underrecognition. Further research, particularly using advanced neuroimaging, is needed to better understand its mechanisms and improve diagnosis.

Key Words: "Alice in Wonderland Syndrome," "AIWS," "micropsia," "macropsia," "metamorphopsia," "migraine," "migraine and perceptual distortion," "AIWS neuroimaging," "AIWS virus," "AIWS COVID-19."

Introduction

Alice in Wonderland syndrome (AIWS), also known as Todd's syndrome, is a perceptual disorder characterized by distorted visual perception (metamorphopsias), abnormalities of body schema, and altered experience of time. The term was adopted from *Alice's Adventures in Wonderland* by Lewis Carroll, in which the protagonist experiences alterations in the size and shape of her body and surrounding objects (Hossain, 2020). These literary descriptions closely resemble the perceptual disturbances reported by patients with AIWS (O'Toole & Modestino, 2017; Farooq & Fine, 2017).

The expression "Alice in Wonderland syndrome" was introduced in 1955 by the British psychiatrist John Todd to describe a group of symptoms "intimately associated with migraine and epilepsy, although not confined to these disorders". As conceptualized by Todd, the syndrome comprised derealization, depersonalization, hyperschematia, hyposchematia, somatopsychic duality, illusory changes in the size, distance, or position of stationary objects,

feelings of levitation, and alterations in the sense of time. However, earlier descriptions of similar phenomena were provided by Caro Lippman in 1952, who documented migraine patients experiencing sensations of becoming short and wide during attacks, and by Coleman, both of whom drew comparisons with Lewis Carroll's narrative before the eponym was formally established (Blom, 2016; Farooq & Fine, 2017).

Clinically, AIWS encompasses a broad spectrum of perceptual distortions. Frequently reported symptoms include micropsia (objects appearing smaller than they are), macropsia (objects appearing larger), teleopsia (objects appearing farther away), and pelopsia (objects appearing closer). Patients may also experience disturbances of their own body image, such as microsomatognosia and macrosomatognosia, as well as hallucinations or illusions involving expansion, reduction, or distortion of body parts. Additional features may include auditory hallucinations (distorted voices, strange music, or noises without psychosis), nausea, dizziness, agitation, and a distorted perception of time that may seem to pass unusually slowly or rapidly. (O'Toole & Modestino, 2017; Farooq & Fine, 2017).

AIWS has been reported in association with various conditions, including migraine, epilepsy, intoxicants, infectious states, febrile delirium, and brain lesions such as tumors (Farooq & Fine, 2017). The syndrome appears to be more common in children than adults, with an average age of approximately six years (O'Toole & Modestino, 2017). Although epidemiological data in the general population are lacking, clinical studies suggest that up to 15% of patients with migraine may experience AIWS symptoms. Moreover, individual symptoms such as micropsia and macropsia may not be rare in the general population; a cross-sectional study of 1,480 adolescents reported lifetime prevalence rates of 5.6% in males and 6.2% in females (Blom, 2016).

Despite its distinctive phenomenology and historical background, AIWS remains a complex and heterogeneous syndrome. Previous reviews have summarized its clinical features and associations. However, several new case reports have been published after 2020, contributing additional insights into its clinical presentation and associated conditions.

The aim of the present narrative review is to provide a concise synthesis of the current state of knowledge on Alice in Wonderland syndrome based on scientific publications published between 2015 – 2026. An exception is the study published in 2013 by J. R. Lanska and D. J. Lanska, as it was the only one to comprehensively present proposed diagnostic criteria for Alice in Wonderland Syndrome. The paper focuses on the analysis of clinical presentation. An additional objective is to identify existing research gaps and to indicate directions for future studies that may contribute to a better understanding of the pathophysiology of this phenomenon, while also incorporating newly reported post-2020 cases to update earlier reviews.

Methodology

The literature search was performed using the ResearchGate, PubMed, Scopus, Web of Science, and Google Scholar databases. The following keywords were used: "Alice in Wonderland Syndrome," "AIWS," "micropsia," "macropsia," "metamorphopsia," "migraine," "migraine and perceptual distortion," "AIWS neuroimaging," "AIWS virus," and "AIWS COVID-19."

The applied methodology enabled a current and comprehensive overview of AIWS-related research, incorporating both clinical data and advances in neurobiological research, while maintaining a critical analysis of the limitations of the available literature.

Pathophysiology

Alice in Wonderland Syndrome (AIWS) may stem from disturbances in multisensory integration within the posterior-to-parasagittal network, which includes occipito-temporal regions. These disruptions can alter body representation, perceived size, and the sense of time, even in the absence of permanent structural brain abnormalities (Friedrich et al., 2024; Chirchiglia et al., 2019).

In migraine with aura, AIWS may reflect abnormal connectivity between area V3 (a key site for the propagation of cortical spreading depression) and the superior temporal sulcus (STS), a region involved in multisensory processing. It is suggested that increased interictal connectivity facilitates the spread of depolarization to multisensory integration areas, resulting in perceptual distortions and altered time experience (Mastria et al., 2023; Friedrich et al., 2024).

Lesion-network mapping studies indicate that different lesion sites can belong to a single, functionally coherent network, involving regions such as the right extrastriate body area (EBA) and the inferior parietal lobule (IPL). This supports the view of AIWS as a disorder of distributed multimodal networks rather than a consequence of a single focal lesion (Friedrich et al., 2024).

In the context of infections—particularly those caused by Epstein-Barr virus (EBV)—and other etiologies, AIWS may represent transient and dynamic disruptions in networks responsible for visual and proprioceptive processing. These disturbances can lead to phenomena such as micropsia, macropsia, kinaesthetic distortions, and altered temporal perception across ictal, interictal, and post-ictal phases (Frattale et al., 2023; Linhares et al., 2024; Friedrich et al., 2024).

Functional neuroimaging findings suggest that AIWS arises from dysregulation at the level of large-scale brain networks. Although its causes may vary, they converge on a shared network responsible for multisensory integration and body representation within the broader framework of perception (Naarden et al., 2019; Mastria et al., 2023; Ceriani, 2024; Friedrich et al., 2024).

These observations are further supported by similarities in network alterations seen in AIWS, migraine, and infection-related cases, highlighting the primary role of network-level mechanisms over location-specific differences in the pathophysiology of this syndrome (Mastria et al., 2023; Friedrich et al., 2024).

Diagnostic Criteria

Alice in Wonderland Syndrome (AIWS) remains a clinically intriguing but diagnostically challenging condition, as universally accepted and standardized diagnostic criteria have not yet been established. Nevertheless, several attempts have been made to systematize its symptomatology and improve clinical recognition.

A significant contribution to the classification of AIWS was made by J. R. Lanska and D. J. Lanska, who reviewed 81 documented cases and proposed a symptom-based categorization. They distinguished three principal types of AIWS according to the predominant perceptual disturbances reported by patients. Type A involves somesthetic (bodily) perceptual distortions, Type B encompasses visual perceptual disturbances, and Type C represents a combination of both somesthetic and visual alterations Somesthetic disturbances (Type A) refer to distortions in the perception of one's own body or its relation to the surrounding environment. Patients may experience phenomena such as micro- or macrosomatognosia, in which body parts are perceived as abnormally small or large. These alterations affect bodily awareness rather than visual input alone.

In contrast, visual perceptual disturbances (Type B) involve distortions in the size, distance, or spatial relationships of external objects. Reported symptoms include micropsia (objects appearing smaller than they are), macropsia (objects appearing larger), pelopsia (objects appearing closer), and telopsia (objects appearing farther away). According to Lanska and Lanska Type B was the most common presentation, accounting for approximately 75% of cases. This subtype was observed predominantly in pediatric patients, frequently in association with viral infections.

Type C, characterized by the coexistence of both somesthetic and visual distortions, represented about 16% of the analyzed cases. This presentation was more commonly described in older individuals, particularly those with a history of migraine. The remaining cases displayed overlapping or atypical features.

Expanding upon this framework, Giulia Mastria and colleagues further refined the conceptualization of AIWS symptoms. In their clinical and pathophysiological review, they proposed that the core characteristics described in Types A, B, and C should be considered obligatory symptoms for the diagnosis of AIWS. In other words, perceptual distortions affecting bodily schema, visual perception, or both constitute the essential clinical foundation of the syndrome.

In addition to these primary features, Mastria identified several ancillary (facultative) symptoms that may accompany AIWS episodes but are not required for diagnosis. These include derealization (a sense that the external world feels unreal), depersonalization (a feeling of detachment from oneself), somatopsychic duality (a disturbed integration between body and self-experience), and altered time perception. Although these experiences can significantly influence the clinical picture, they are considered supportive rather than defining criteria.

In summary, while no universally endorsed diagnostic guidelines currently exist for AIWS, the classification proposed by Lanska and Lanska, later elaborated by Mastria, provides a structured approach to understanding its clinical manifestations. Core diagnostic consideration should focus on perceptual distortions—somesthetic, visual, or combined—while additional dissociative and temporal symptoms may complement the overall presentation. (Lanska & Lanska, 2013; Mastria et al. (2016).

Migraine

Alice in Wonderland Syndrome (AIWS) has been reported in association with various neurological and systemic conditions, including infections, epilepsy, cerebrovascular diseases,

structural brain abnormalities, medication use, and psychiatric disorders; however, in adults, migraine represents the most frequently identified and clinically relevant underlying condition. Functional neuroimaging data demonstrating altered connectivity patterns in migraineurs with AIWS further support the hypothesis of a migraine-related network dysfunction (Piervincenzi et al., 2023). Large cohort data indicate that AIWS symptoms occur in approximately one out of six adult migraine patients, with a higher prevalence among individuals with migraine with aura, although nearly half of affected patients do not experience aura, suggesting that AIWS is not exclusively an aura-dependent phenomenon (Fitzek et al., 2024).

Clinically, AIWS in migraine is characterized predominantly by visual distortions, particularly alterations in perceived size and distance (e.g., micropsia and telopsia), which represent the most common core symptoms. In tertiary headache cohorts, visual-only presentations predominate, whereas combined visual–somatosensory forms are less frequent and isolated somatosensory symptoms are rarely observed. Many patients report more than one perceptual distortion over their lifetime, indicating that AIWS often manifests as a multidimensional perceptual disturbance rather than a single isolated symptom. Additional features include altered perception of time, disturbances of body schema, and feelings of environmental unfamiliarity, reflecting impairment of higher-order integrative processing. Dissociative experiences—particularly depersonalization and derealization—are significantly more frequent in migraineurs with AIWS than in those without the syndrome (Fitzek et al., 2024;Mastria et al., 2021).

Episodes are transient, typically lasting around 30–40 minutes, and most commonly occur in close temporal relationship with migraine attacks, often within the hour preceding headache onset or during the headache phase itself (Mastria et al., 2021; Valença et al., 2025). Notably, symptom onset frequently occurs in childhood or adolescence and may precede the development of migraine by several years, supporting the notion of a shared neurobiological substrate (Mastria et al., 2021; Fitzek et al., 2024). In patients with vestibular migraine, visual distortions and extrapersonal misperceptions are particularly prominent, and preventive migraine therapy often leads to partial or complete symptom improvement (Beh et al., 2018).

Infection

Multiple infectious agents have been implicated in the development of Alice in Wonderland syndrome (AIWS), particularly those associated with encephalopathy. Reported pathogens include Epstein–Barr virus (EBV), influenza viruses, herpes simplex virus, enteroviruses, and varicella, highlighting the broad infectious background of this condition. Among these, EBV-related infectious mononucleosis is the most frequently described infectious etiology of AIWS (Farooq & Fine, 2017).

Zika virus (ZIKV) infection represents a notable emerging cause of AIWS, with patients developing perceptual disturbances such as metamorphopsia, micropsia, macropsia, and altered spatial perception following acute infection. These symptoms may occur after the resolution of systemic manifestations, suggesting a post-infectious neurological process. Mechanistically, ZIKV may induce dysfunction within temporo-occipital cortical regions involved in sensory integration, either through direct neuronal injury or immune-mediated pathways (Paniz-Mondolfi et al., 2018).

SARS-CoV-2 and Neurological Manifestations

SARS-CoV-2 infection has been increasingly associated with AIWS presentations in both adult and pediatric populations In adults, AIWS has been described as a manifestation of non-convulsive status epilepticus in the context of cerebral venous sinus thrombosis related to COVID-19, suggesting a potential role of vascular and epileptic mechanisms. These findings indicate that AIWS may arise from dysfunction in distributed cortical networks under inflammatory or vascular stress.

In children, SARS-CoV-2 infection has been linked to transient perceptual disturbances, including micropsia, macropsia, teleopsia, and hallucinations, often occurring without structural abnormalities or epileptiform activity. Proposed mechanisms include neuroinflammation, disruption of the blood–brain barrier, autoimmune responses, and direct viral neurotropism. Additionally, prolonged or recurrent symptoms in some cases suggest that post-viral inflammatory processes may contribute to the persistence of AIWS manifestations. (Biggi et al., 2023; Staccioli et al., 2025).

Influenza-Associated AIWS

Influenza virus infection is another recognized trigger of AIWS, particularly in pediatric patients. Clinical cases have demonstrated the onset of visual distortions such as micropsia and macropsia shortly after resolution of influenza symptoms, with normal neurological and imaging findings. These episodes are typically transient and resolve spontaneously over time, supporting the hypothesis of reversible cortical dysfunction following viral infection. EBV remains one of the most frequently reported infectious causes in comparison to influenza and other viruses (Kubota et al., 2020).

Epstein–Barr Virus and Post-Infectious AIWS

EBV-associated AIWS is well documented, particularly in pediatric populations presenting with transient disturbances in perception, body schema, and time processing following mild viral illness. These symptoms typically occur in fully conscious individuals and are not associated with epileptic activity or structural brain abnormalities. The temporal relationship between infection and symptom onset supports the hypothesis of a reversible, post-infectious cortical dysfunction (Ansari et al., 2025).

In some cases, EBV-related neurological involvement may result in prolonged or chronic AIWS symptoms. Persistent perceptual disturbances have been described following EBV encephalitis, suggesting long-term dysfunction of temporo-parieto-occipital cortical regions responsible for multisensory integration. This indicates that while AIWS is often self-limiting, more severe infectious involvement may lead to lasting neurological sequelae (Hosokawa et al., 2024).

Pathophysiological Mechanisms of Infection-Related AIWS

The pathophysiology of infection-associated AIWS is thought to involve disruption of cortical regions responsible for integrating visual and somatosensory information, particularly within the temporo-occipital and parietal lobes. Multiple mechanisms have been proposed, including direct viral neurotropism, immune-mediated neuronal injury, molecular mimicry, and neuroinflammation (Paniz-Mondolfi et al., 2018; Staccioli et al., 2025). Additionally, vascular

factors and altered cerebral perfusion may contribute to symptom development in certain cases (Biggi et al., 2023). The interplay of these mechanisms likely explains the variability in clinical presentation and duration of AIWS across different infectious contexts (Staccioli et al., 2025).

Epilepsy

Alice in Wonderland syndrome (AIWS) may occur in the course of focal epilepsy and is associated with disturbances of visual perception, such as macropsia or micropsia, without necessarily impairing consciousness. In the case of an 82-year-old patient, AIWS symptoms—including macropsia, derealization, and altered time perception—were closely linked to focal seizures originating in the right occipital region, as confirmed by video-EEG; this suggests that these disturbances may result from the spread of epileptic activity to parietal and temporal regions responsible for sensory integration (Matsudaira et al., 2020). Similarly, in an 11-year-old boy with a history of frontal lobe epilepsy, episodes of visual perceptual changes characteristic of AIWS were ultimately associated with an epileptogenic focus in the occipital lobe, despite the absence of clear abnormalities on standard EEG; further investigation revealed epileptiform activity in the left occipital region, and treatment with lacosamide led to a significant reduction in symptoms (Harris et al., 2024). Furthermore, a case of a 34-year-old pregnant woman with a right temporo-parietal cavernoma demonstrated that AIWS symptoms—including complex visual distortions (e.g., objects changing size and shape, people appearing distorted) and auditory hallucinations—may be secondary to epileptiform activity associated with structural brain lesions; these symptoms improved with antiepileptic treatment such as levetiracetam, despite initially fluctuating clinical and EEG findings, supporting an epileptic mechanism underlying AIWS manifestations (Philip et al., 2015).

Conclusions

From a clinical perspective, management of AIWS should primarily focus on treating the underlying condition—most commonly migraine, infections (e.g., those associated with Epstein-Barr virus), epilepsy, or other neurological disorders. In many cases, effective treatment of the primary disorder leads to partial or complete resolution of AIWS symptoms (Beh et al., 2018; Kubota et al., 2020; Ansari et al., 2025).

Current evidence indicates that the pathophysiology of AIWS is predominantly related to network-level dysfunction rather than focal structural brain lesions (Friedrich et al., 2024; Mastria et al., 2023). However, despite advances in research, the precise neurobiological mechanisms remain insufficiently understood. This highlights the need for further advanced neuroimaging studies to better characterize the dynamics and specificity of brain network alterations in AIWS (Naarden et al., 2019; Ceriani, 2024).

Importantly, due to the lack of standardized diagnostic guidelines and the broad, often variable spectrum of symptoms, AIWS may be easily overlooked or misdiagnosed in clinical practice. This further emphasizes the need for increased awareness and more structured diagnostic approaches.

Overall, AIWS represents a highly intriguing yet still underexplored syndrome with considerable clinical diversity. Its unique phenomenology and potential relevance for

understanding brain network function underscore the importance of broader and more in-depth research in both neurology and cognitive science.

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References:

Ansari, A. Z., Smith, S. L., Anderson, A. S., Siddiqi, A. A., & Hafeez, S. (2025). Alice in Wonderland syndrome in a child following Epstein-Barr virus infection: A case report. *Cureus, 17*(6), e86532. https://doi.org/10.7759/cureus.86532

Beh, S. C., Masrour, S., Smith, S. V., & Friedman, D. I. (2018). Clinical characteristics of Alice in Wonderland syndrome in a cohort with vestibular migraine. *Neurology: Clinical Practice, 8*(5), 389–396. https://doi.org/10.1212/CPJ.0000000000000518

Biggi, M., Contento, M., Magliani, M., Giovannelli, G., Barilaro, A., Bessi, V., Lombardo, I., Massacesi, L., & Rosati, E. (2023). Alice in Wonderland syndrome "through the looking-glass" in a rare presentation of non-convulsive status epilepticus in cerebral venous sinus thrombosis and COVID-19. *Cortex, 167*, 218–222. https://doi.org/10.1016/j.cortex.2023.06.020

Blom, J. D. (2016). Alice in Wonderland syndrome: A systematic review. *Neurology: Clinical Practice, 6*(3), 259–270. https://doi.org/10.1212/CPJ.0000000000000251

Ceriani, C. E. J. (2024). Beyond vertigo: Vestibular, aural, and perceptual symptoms in vestibular migraine. *Current Pain and Headache Reports, 28*(7), 633–639. https://doi.org/10.1007/s11916-024-01245-3

Chirchiglia, D., Chirchiglia, P., & Marotta, R. (2019). New-onset Alice in Wonderland syndrome following brucellosis. *Romanian Journal of Neurology, 18*(3), 137–139. https://doi.org/10.37897/RJN.2019.3.6

Farooq, O., & Fine, E. J. (2017). Alice in Wonderland syndrome: A historical and medical review. *Pediatric Neurology, 77*, 5–11. https://doi.org/10.1016/j.pediatrneurol.2017.08.008

Fitzek, M. P., Mecklenburg, J., Overeem, L. H., Lange, K. S., Siebert, A., Triller, P., Neeb, L., Dreier, J. P., Kondziella, D., Reuter, U., & Raffaelli, B. (2024). Alice in Wonderland syndrome (AIWS): Prevalence and characteristics in adults with migraine. *Journal of Neurology, 271*(8), 5146–5155. https://doi.org/10.1007/s00415-024-12471-5

Frattale, I., Papetti, L., Ursitti, F., Sforza, G., Monte, G., Voci, A., Proietti Checchi, M., Mazzone, L., & Valeriani, M. (2023). Visual disturbances spectrum in pediatric migraine. *Journal of Clinical Medicine, 12*(8), 2780. https://doi.org/10.3390/jcm12082780

Harris, A., et al. (2024). Occipital lobe epilepsy presenting with Alice in Wonderland syndrome. *Neurology*. Advance online publication. https://doi.org/10.1212/WNL.0000000000206126

Hosokawa, K., Oi, K., Hitomi, T., Mitsueda, T., Nakagawa, T., & Ikeda, A. (2024). *Clinical Neurology, 64*(2), 99–104. https://doi.org/10.5692/clinicalneurol.cn-001906

Hossain, M. M. (2020). Alice in Wonderland syndrome (AIWS): A research overview. *AIMS Neuroscience, 7*(4), 389–400. https://doi.org/10.3934/Neuroscience.2020024

Kubota, K., Shikano, H., Fujii, H., Nakashima, Y., & Ohnishi, H. (2020). Alice in Wonderland syndrome associated with influenza virus infection. *Pediatrics International, 62*(12), 1391–1393. https://doi.org/10.1111/ped.14341

Lanska, J. R., & Lanska, D. J. (2013). Alice in Wonderland syndrome: Somesthetic vs visual perceptual disturbance. *Neurology, 80*(13), 1262–1264. https://doi.org/10.1212/WNL.0b013e31828970ae

Linhares, E., Menezes, A., & Castro, M. (2024). Down the rabbit hole: A literature review based on causes and consequences of the Alice in Wonderland syndrome. *Brazilian Medical Students, 8*(12), 258. https://doi.org/10.53843/bms.v8i12.258

Mastria, G., Mancini, V., Cesare, M. D., Puma, M., Alessiani, M., Petolicchio, B., Viganò, A., & Di Piero, V. (2021). Prevalence and characteristics of Alice in Wonderland syndrome in adult migraineurs. *Cephalalgia, 41*(5), 515–524. https://doi.org/10.1177/0333102420968245

Mastria, G., Mancini, V., Viganò, A., & Di Piero, V. (2016). Alice in Wonderland syndrome: A clinical and pathophysiological review. *BioMed Research International, 2016*, 8243145. https://doi.org/10.1155/2016/8243145

Mastria, G., Mancini, V., Viganò, A., Piervincenzi, C., Petsas, N., Puma, M., Giannì, C., Pantano, P., & Di Piero, V. (2023). Neuroimaging markers of Alice in Wonderland syndrome in patients with migraine with aura. *Frontiers in Neurology, 14*, 1210811. https://doi.org/10.3389/fneur.2023.1210811

Matsudaira, T., Terada, K., & Takahashi, Y. (2020). Alice in Wonderland syndrome in an elderly patient with focal onset epilepsy. *Journal of Clinical Neuroscience, 76*, 243–245. https://doi.org/10.1016/j.jocn.2020.04.010

Naarden, T., ter Meulen, B. C., van der Weele, S. I., & Blom, J. D. (2019). Alice in Wonderland syndrome as a presenting manifestation of Creutzfeldt-Jakob disease. *Frontiers in Neurology, 10*. https://doi.org/10.3389/fneur.2019.00473

O'Toole, P., & Modestino, E. J. (2017). Alice in Wonderland syndrome: A real life version of Lewis Carroll's novel. *Brain & Development, 39*(6), 470–474. https://doi.org/10.1016/j.braindev.2017.01.004

Paniz-Mondolfi, A. E., Giraldo, J., Rodríguez-Morales, A. J., et al. (2018). Alice in Wonderland syndrome: A novel neurological presentation of Zika virus infection. *Journal of NeuroVirology, 24*(5), 660–663. https://doi.org/10.1007/s13365-018-0645-1

Philip, M., Kornitzer, J., Marks, D., et al. (2015). Alice in Wonderland syndrome associated with a temporo-parietal cavernoma. *Brain Imaging and Behavior, 9*(4), 910–912. https://doi.org/10.1007/s11682-015-9355-y

Piervincenzi, C., Petsas, N., Viganò, A., Mancini, V., Mastria, G., Puma, M., Giannì, C., Di Piero, V., & Pantano, P. (2023). Functional connectivity alterations in migraineurs with Alice in Wonderland syndrome. *Neurological Sciences, 44*(1), 305–317. https://doi.org/10.1007/s10072-022-06404-1

Staccioli, S., Mariani, R., Bompard, S., Olivini, N., Fanfoni, C., Mirra, G., Bisozzi, E., Campana, A., & Lettori, D. (2025). Alice in Wonderland syndrome in children with severe acute respiratory syndrome SARS-CoV-2 infection. *Pediatric Neurology, 162*, 28–31. https://doi.org/10.1016/j.pediatrneurol.2024.10.008

Valença, M. M., Reis, R. S. dos, & Andrade, J. R. de. (2025). "Perceiving myself small: A body volume regression to age three"—A new manifestation of Alice in Wonderland syndrome in a migraine patient. *Headache Medicine, 16*(3), 176–179. https://doi.org/10.48208/HeadacheMed.2025.28