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ALICE IN WONDERLAND SYNDROME IN THE LIGHT OF THE LATEST REPORTS – A LITERATURE REVIEW

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ABSTRACT

Alice in Wonderland Syndrome (AIWS) was first described in 1955 by British psychiatrist John Todd, hence it is also known as Todd's syndrome. The name of the disorder refers to Lewis Carroll's novel Alice in Wonderland, whose author, who probably suffered from migraines with aura, may have been inspired by his own experiences with perceptual disturbances. AIWS is a rare, complex sensory perception disorder that lies at the intersection of neurology and psychiatry. The syndrome is characterized by transient, reversible distortions in the perception of space, time, movement, and one's own body image. These episodes are usually short-lived, lasting from a few minutes to several hours, and in most cases are completely reversible. The aim of this literature review was to present the current state of knowledge on AIWS, with particular emphasis on its etiology, symptomatology, pathophysiological mechanisms, diagnostic methods, and therapeutic options. The analysis also included studies comparing AIWS with other conditions with similar clinical presentations, such as migraine with aura, temporal lobe epilepsy, and schizophrenia. Contemporary research emphasizes the importance of transient disturbances in brain bioelectrical activity and abnormalities in blood flow in the occipital and temporal lobes in the pathogenesis of AIWS. Treatment is causal and focuses on the underlying disease. Alice in Wonderland syndrome, despite its rarity, is a fascinating example of the interaction between human perceptual, cognitive, and neurological processes. The increase in the number of scientific publications in recent years indicates a growing interest in this syndrome, but the limited number of studies on its mechanisms highlights the need for further interdisciplinary research collaboration.

KEYWORDS

Alice in Wonderland Syndrome, AIWS, Perceptual Distortion, Migraine Aura, Neurology

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1. Introduction

Alice in Wonderland Syndrome (AIWS) was first described by British psychiatrist John Todd in 1955 in the Canadian Medical Association Journal. Todd observed characteristic perceptual disturbances in his patients—distortions in the size, shape, and distance of objects or body parts that resembled the fantastical experiences of the heroine in Lewis Carroll's novel Alice in Wonderland (1865). It is from this book that the syndrome gets its name. [1] Todd's interest was piqued by the fact that the author of the novel himself suffered from severe migraine attacks with aura, during which he may have experienced similar perceptual disturbances. Todd linked AIWS to migraine, suggesting that its mechanisms may be the key to understanding this phenomenon. There are speculations that some of the surreal episodes in his book—such as Alice's sudden shrinking and enlarging—were inspired by his personal experiences. [2] After Todd's publication, Alice in Wonderland Syndrome began to be recognized as a rare but distinctive neurological disorder, often associated with migraine, temporal lobe epilepsy, viral infections (especially Epstein-Barr virus), as well as metabolic disorders or brain injuries. In the following decades, research on AIWS has led to a better understanding of its relationship to the functioning of the parietal and occipital lobes and the complex processes of visual-spatial stimulus integration. [3]

2. Literature review methodology

A literature review was conducted to collect and analyze current data on AIWS. Scientific publications published since 2016 were included. The literature search was conducted in the PubMed® database using a combination of keywords: “Alice in Wonderland syndrome,” “AIWS,” “perceptual distortion,” “migraine aura,” and “neurology.” The analysis included review publications, articles describing clinical cases, as well as studies comparing AIWS with other diseases with similar clinical presentations. Articles discussing new reports on the pathophysiological mechanisms, etiology, diagnosis, and therapeutic options for the syndrome were also included.

3. Symptomatology

AIWS symptoms include a variety of perceptual disturbances that may occur in isolation or in various combinations. [5] The most commonly reported symptoms include:

- **Macropsia and micropsia** – altered perception of the size of objects (e.g., objects appear much larger or smaller than they actually are). [6]
- **Dysmetropia** – misjudgment of distance, e.g., objects appear closer or farther away than they actually are. [7]
- **Changes in body perception** – a feeling of disproportion or changes in the size of body parts. [8]
- **Time perception disorders** – subjective feeling of acceleration or deceleration of time. [9]
- **Sensory distortions** – altered perception of sounds, colors, or tactile sensations (less commonly reported).

The symptoms are transient, usually lasting from a few minutes to a few hours, and are reversible in most cases. [5]

Table 1. The most common visual symptoms in AIWS:

Symptom	Prevalence (%)
Micropsia	58.6
Macropsia	45.0
Teleopsia	23.1
Dysmorphopsia	20.1
Kinetopsia	8.9
Pelopsia	6.5
Achromatopsia	5.3
Hyperchromatopsia	2.4
Visual achallesthesia	2.4
“Zoom vision”	2.4

4. Etiology and pathophysiological mechanisms

Alice in Wonderland syndrome is a multifactorial disorder. Due to its low

diagnosis rate and limited epidemiological data, it is difficult to determine its main causes. To date, eight main groups of disorders associated with its occurrence have been identified:

1. Migraines

● The most common cause of AIWS, especially migraines with aura. Pathophysiological mechanisms include changes in blood flow in the occipital lobes and cortical dysfunction. [8]

2. Viral and bacterial infections

● AIWS can occur in the course of viral infections such as infectious mononucleosis

(Epstein-Barr Virus), chickenpox, or influenza. Lyme disease is also mentioned as a potential etiological factor. [4]

3. Epilepsy

● AIWS symptoms may be the result of epileptic seizures, particularly in the temporal and parietal lobes. [10]

4. Mental disorders

● Schizophrenia, depression, and psychotic episodes may accompany AIWS symptoms. [11]

5. Neurological and neuroinflammatory diseases

● Encephalitis (e.g., autoimmune) and neurodegenerative diseases such as multiple sclerosis are potential causes of AIWS. [10]

6. Metabolic and toxic disorders

● Transient metabolic disorders, e.g., hypoglycemia, and exposure to toxic substances (e.g., drugs) may trigger AIWS episodes. [3]

7. Sleep disorders

● Hypnagogia and narcolepsy have been associated with AIWS episodes, indicating the involvement of sleep disorders in the pathogenesis. [5]

8. Developmental disorders in children

● AIWS is more common in children, which may be related to the immaturity of the central nervous system. [12]

Pathophysiological mechanisms include transient dysfunctions in the occipital, temporal, and parietal lobes, which are responsible for the integration of sensory stimuli. Most cases are transient, suggesting that AIWS may be the result of dynamic changes in brain function, such as transient ischemia or bioelectrical instability in specific areas of the brain (e.g., the occipital lobes). Vascular disorders (migraines) and infections that can cause nervous system dysfunction appear to be of particular importance. [13]

5. Diagnostics

No uniform diagnostic criteria for AIWS have been developed. Diagnosis is based on

a detailed clinical history and the exclusion of other neurological causes, such as brain tumors, strokes, or encephalopathies. [5] Additional tests, such as MRI, EEG, and serological tests, can help determine the etiology. Often, the diagnosis can be presumed after other causes have been ruled out. A person suffering from Alice in Wonderland syndrome may be reluctant to describe their symptoms for fear of being considered mentally ill. [14]

6. Results

In order to facilitate differentiation between various neuropsychological and neurological conditions, a comparative table has been provided for AIWS, schizophrenia, hallucinogenic persisting perception disorder (HPPD), temporal lobe epilepsy, and migraine.

This table takes into account the characteristics of symptoms as well as causes, duration, patient awareness, cognitive impairment, diagnosis, and treatment: This table takes into account the characteristics of symptoms, causes, duration, patient awareness, cognitive impairment, diagnosis, and treatment:

Table 2. Comparison of diseases:

Criterion	Epilepsy	Schizophrenia	Temporal Lobe Epilepsy	Migraine
Definition	A neurological disorder characterized by recurrent seizures resulting from abnormal electrical activity in the brain.	A mental disorder characterized by distortions in thinking, perception, emotions, language, sense of self, and behavior.	A form of epilepsy involving the temporal lobe, often associated with complex partial seizures and altered states of consciousness.	A neurological disorder characterized by recurrent headaches, often one-sided and pulsating, sometimes accompanied by aura.
Symptoms	Seizures (convulsions), loss of consciousness, muscle spasms, sometimes sensory disturbances or automatisms.	Hallucinations (mostly auditory), delusions, disorganized speech or behavior, negative symptoms such as emotional flatness.	Hallucinations, altered consciousness, automatisms, possible memory gaps during seizures.	Headache, nausea, vomiting, photophobia, phonophobia, sometimes visual or sensory aura.
Causes	Most commonly idiopathic, genetic, or post-traumatic; may result from brain injury, infection, or stroke.	Changes in neurotransmitter balance (dopamine, glutamate); genetic and environmental factors.	Lesions in the temporal lobe, head trauma, infections, or idiopathic causes.	Changes in vascular tone and neurochemical balance; triggers include stress, hormonal changes, sleep deprivation, certain foods.
Duration of symptoms	Episodic, lasting from seconds to minutes; may recur irregularly or periodically.	Persistent, symptoms may last months or years, with remissions and relapses.	Episodic, seizures lasting seconds to minutes; may occur repeatedly.	Episodic, attacks lasting from hours to days, usually preceded by aura and followed by fatigue.
Patient awareness	The patient may lose consciousness during the seizure and usually does not remember the episode.	Lack of insight — the patient often does not realize the pathological nature of their experiences.	The patient may be conscious during partial seizures but not recall them afterward.	The patient is usually conscious, though sometimes impaired during aura or severe pain.
Cognitive disturbances	Temporary disorientation after a seizure; long-term cognitive deficits in chronic cases.	Disorganized thinking, cognitive decline, disturbances in abstract reasoning and attention.	Possible memory and emotional disturbances after long-term illness.	Sometimes cognitive slowing, concentration problems during or after an attack.
Diagnostics	Interview, EEG, neuroimaging (CT, MRI), laboratory tests to exclude metabolic causes.	Psychiatric and psychological evaluation, observation of behavior and thought processes.	EEG, neurological examination, neuroimaging of the temporal lobe region.	Interview, neurological exam, exclusion of other causes (CT, MRI, EEG if necessary).
Treatment	Antiepileptic drugs (e.g., valproate, carbamazepine, lamotrigine); surgery in resistant cases.	Antipsychotic medication, psychotherapy, psychoeducation, social support.	Antiepileptic medication, sometimes surgical treatment of the temporal lobe.	Analgesics, triptans, prophylactic treatment (beta-blockers, antidepressants, antiepileptics).

Key differences:

- AIWS is a transient perceptual disorder, most commonly occurring in migraines, epilepsy, or after viral infections. The patient is usually aware of the unnatural nature of the perceptual distortions. [5]
- Schizophrenia is a chronic mental disorder characterized by hallucinations and delusions. The patient is usually unaware of the illness, and symptoms persist for long periods of time. [15]
- HPPD is a persistent visual effect that persists after taking hallucinogens, often in the form of recurring visual hallucinations that can last for months or years. [16]
- Temporal lobe epilepsy is associated with seizures that may manifest as hallucinations and changes in perception during their duration. These seizures are short-lived but can lead to confusion and memory difficulties. [17]
- Migraine is a disorder mainly associated with headaches and aura, in which perceptual distortions (e.g., visual disturbances) may occur. These symptoms are usually transient and associated with disturbances in blood flow in the brain. [18]

7. Treatment

The lack of specific therapies for AIWS means that treatment focuses on the underlying condition:

- Migraine – antimigraine drugs (e.g., triptans, beta-blockers).
- Epilepsy – antiepileptic drugs (e.g., carbamazepine). [21]
- Mental disorders – pharmacotherapy and psychotherapy. [19] [20]

8. Discussion

Alice in Wonderland Syndrome (AIWS) remains one of the most enigmatic and poorly understood perceptual disorders. Although seventy years have passed since John Todd first described it in 1955, the etiology and pathophysiology of this syndrome have still not been clearly defined. A review of the literature confirms that AIWS is multifactorial in nature and its occurrence is associated with neurological diseases, infections, and mental disorders. Most often, the syndrome co-occurs with migraine with aura, suggesting the involvement of transient blood flow disturbances in areas responsible for the integration of visual and somatosensory stimuli, especially in the occipital and temporal lobes. Numerous cases of AIWS have been described in the literature in the course of Epstein-Barr virus, chickenpox, or influenza infections, indicating a possible neuroinflammatory mechanism. Clinical observations in patients with temporal lobe epilepsy suggest that transient disturbances in brain bioelectrical activity may underlie the symptoms. Nevertheless, the available data are largely fragmentary, based mainly on individual case reports and small clinical series, which limits the possibility of generalizing the results. There is still a lack of randomized clinical trials on AIWS, and most reports are descriptive or retrospective in nature. The limited number of population studies makes it impossible to accurately estimate the prevalence of the syndrome and its relationship to other disease entities. An additional challenge is the small number of specialists familiar with this disorder, which leads to frequent diagnostic delays or misdiagnosis, especially in contrast to schizophrenia, epilepsy, or psychotic disorders. The direction of research on AIWS is currently moving towards functional neuroimaging (fMRI, SPECT, PET) and analysis of brain bioelectrical activity (EEG, MEG), which allow for the assessment of disorders in the dynamic network of neural connections. The use of modern imaging methods allows for increasingly accurate localization of the areas responsible for perceptual distortions, which may in the future form the basis for the development of objective diagnostic biomarkers. Increasing importance is also being attached to research on the role of the serotonergic and dopaminergic systems in the development of AIWS symptoms, which may indicate common mechanisms with migraine and epilepsy. One of the key problems remains the lack of standardized diagnostic criteria that would enable unambiguous diagnosis of the syndrome in clinical practice. Currently, diagnosis is based mainly on the patient's subjective description, which makes it difficult to compare cases and produce reliable epidemiological analyses. The introduction of standardized criteria, covering both neurological and psychological components, is necessary to improve data quality and conduct future clinical trials. From a therapeutic perspective, the treatment of AIWS remains symptomatic and causal, focusing on the treatment of the underlying disease, such as migraine, epilepsy, or viral infection. However, there are no specific treatments that directly target perceptual disturbances. In the future, combining classic pharmacological interventions with neuromodulatory therapies (e.g., transcranial magnetic stimulation, TMS) may open up new therapeutic possibilities. In summary, Alice in Wonderland Syndrome is a fascinating model for studying the mechanisms of perception, consciousness, and sensory integration. Knowledge in this area is developing rapidly, but still needs to be deepened through well-designed clinical trials, interdisciplinary

collaboration, and standardization of diagnostic criteria and assessment methods. Only such an approach will allow for a more complete understanding of this rare but extremely interesting disorder and the development of more effective therapeutic methods in the future.

9. Conclusions

Alice in Wonderland syndrome is a rare but extremely interesting disorder that reflects the complexity of perceptual processes in the brain. Its links to migraine, infections, and other conditions point to the need for further research into its pathophysiology and the development of better diagnostic tools. The case of AIWS also reminds us how subtle neurological changes can affect our perception of reality. The history of AIWS, including its literature-inspired name, highlights the importance of understanding sensory perception in health and disease. Lewis Carroll, who suffered from migraines, may have reflected his own experiences in his work, adding a unique cultural context to the description of this syndrome.

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REFERENCES

1. Blom JD, Looijestijn J, Goekoop R, et al. Treatment of Alice in Wonderland syndrome and verbal auditory hallucinations using repetitive transcranial magnetic stimulation. A case report with fMRI findings. Psychopathology 2011;44:337–344.
2. Morland D, Wolff V, Dietemann JL, Marescaux C, Namer IJ. Robin Hood caught in Wonderland: brain SPECT findings. Clin Nucl Med. 2013 Dec;38(12):979-81. doi: 10.1097/RLU.0000000000000230. PMID: 24152648.
3. Blom JD. Alice in Wonderland syndrome: A systematic review. Neurol Clin Pract. 2016 Jun;6(3):259-270. doi: 10.1212/CPJ.0000000000000251. PMID: 27347442; PMCID: PMC4909520.
4. Lahat E, Eshel G, Arlazoroff A. "Alice in wonderland" syndrome: a manifestation of infectious mononucleosis in children. Behav Neurol. 1991;4(3):163-6. doi: 10.3233/BEN-1991-4304. PMID: 24487499.
5. Lanska JR, Lanska DJ. Alice in Wonderland Syndrome: somesthetic vs visual perceptual disturbance. Neurology. 2013;80(13):1262-1264. doi:10.1212/WNL.0b013e31828970ae.
6. Farooq O, Fine EJ. Alice in Wonderland Syndrome: A Historical and Medical Review. Pediatr Neurol. 2017 Dec;77:5-11. doi: 10.1016/j.pediatrneurol.2017.08.008. Epub 2017 Aug 24. PMID: 29074056.
7. Mastria G, Mancini V, Viganò A, Di Piero V. Alice in Wonderland Syndrome: A Clinical and Pathophysiological Review. Biomed Res Int. 2016;2016:8243145. doi: 10.1155/2016/8243145. Epub 2016 Dec 27. PMID: 28116304; PMCID: PMC5223006.
8. Fitzek MP, Mecklenburg J, Overeem LH, Lange KS, Siebert A, Triller P, Neeb L, Dreier JP, Kondziella D, Reuter U, Raffaelli B. Alice in Wonderland Syndrome (AIWS): prevalence and characteristics in adults with migraine. J Neurol. 2024 Aug;271(8):5146-5155. doi: 10.1007/s00415-024-12471-5. Epub 2024 May 31. PMID: 38822148; PMCID: PMC11319383.
9. Brooks JBB, Prosdocimi FC, Rosa PBD, Fragoso YD. Alice in Wonderland syndrome: "Who in the world am I?". Arq Neuropsiquiatr. 2019 Sep 23;77(9):672-674. doi: 10.1590/0004-282X20190094. PMID: 31553398.
10. Panpruang P, Wongwandee M, Rattanajaruskul N, Roongsangmanoon W, Wongsoasu A, Angkananard T. Alice in Wonderland Syndrome-Like Seizure and Refractory Supraventricular Tachycardia. Case Rep Neurol. 2021 Nov 2;13(3):716-723. doi: 10.1159/000519509. PMID: 34950010; PMCID: PMC8647104.
11. Kuo YT, Chiu NC, Shen EY, Ho CS, Wu MC. Cerebral perfusion in children with Alice in Wonderland syndrome. Pediatr Neurol. 1998 Aug;19(2):105-8. doi: 10.1016/s0887-8994(98)00037-x. PMID: 9744628.

12. Brumm K, Walenski M, Haist F, Robbins SL, Granet DB, Love T. Functional magnetic resonance imaging of a child with Alice in Wonderland syndrome during an episode of micropsia. J AAPOS. 2010 Aug;14(4):317-22. doi: 10.1016/j.jaapos.2010.03.007. Epub 2010 Jul 3. PMID: 20598927; PMCID: PMC2928409.
13. Liu AM, Liu JG, Liu GW, Liu GT. "Alice in wonderland" syndrome: presenting and follow-up characteristics. Pediatr Neurol. 2014 Sep;51(3):317-20. doi: 10.1016/j.pediatrneurol.2014.04.007. Epub 2014 Apr 12. PMID: 25160537.
14. Picchioni MM, Murray RM. Schizophrenia. BMJ. 2007 Jul 14;335(7610):91-5. doi: 10.1136/bmj.39227.616447.BE. PMID: 17626963; PMCID: PMC1914490.
15. Martinotti G, Santacroce R, Pettorruso M, Montemitro C, Spano MC, Lorusso M, di Giannantonio M, Lerner AG. Hallucinogen Persisting Perception Disorder: Etiology, Clinical Features, and Therapeutic Perspectives. Brain Sci. 2018 Mar 16;8(3):47. doi: 10.3390/brainsci8030047. PMID: 29547576; PMCID: PMC5870365.
16. Binding LP, Dasgupta D, Giampiccolo D, Duncan JS, Vos SB. Structure and function of language networks in temporal lobe epilepsy. Epilepsia. 2022 May;63(5):1025-1040. doi: 10.1111/epi.17204. Epub 2022 Mar 4. PMID: 35184291; PMCID: PMC9773900.
17. Aguilar-Shea AL, Membrilla Md JA, Diaz-de-Teran J. Migraine review for general practice. Aten Primaria. 2022 Feb;54(2):102208. doi: 10.1016/j.aprim.2021.102208. Epub 2021 Nov 16. PMID: 34798397; PMCID: PMC8605054.
18. Losada-Del Pozo R, Cantarín-Extremera V, García-Peñas JJ, Duat-Rodríguez A, López-Marín L, Gutiérrez-Solana LG, Ruiz-Falcó ML. Características y evolución de los pacientes con síndrome de Alicia en el País de las Maravillas [Characteristics and evolution of patients with Alice in Wonderland syndrome]. Rev Neurol. 2011 Dec 1;53(11):641-8. Spanish. PMID: 22086425.
19. Bui E, Chatagner A, Schmitt L. Alice in wonderland syndrome in major depressive disorder. J Neuropsychiatry Clin Neurosci. 2010 Summer;22(3):352j.e16-352.e16. doi: 10.1176/jnp.2010.22.3.352.e16. PMID: 20686154.
20. Symonds JD, Zuberi SM, Johnson MR. Advances in epilepsy gene discovery and implications for epilepsy diagnosis and treatment. Curr Opin Neurol. 2017 Apr;30(2):193-199. doi: 10.1097/WCO.0000000000000433. PMID: 28212175.