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DISTORTED PERCEPTION - INSIGHTS INTO ALICE IN WONDERLAND SYNDROME

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ABSTRACT

Research objectives: Alice in Wonderland syndrome (AIWS) is a perceptual disorder characterized by distortions of visual perception (metamorphopsias), the body schema and the experience of time. The aim of this study is to summarise the current state of knowledge about this disease.
Methods: The PubMed database was searched using the term: ”Alice in Wonderland Syndrome”. After analysis, 34 papers were included in the review.
Key findings: Affected individuals can experience visual illusions, including dysmetropsia, namely micropsia (objects appear small), macropsia (objects appear large), teleopsia (objects appear further away than they are). In addition to these alterations in perception, patients might have distorted experience of time, disorders of consciousness such as feelings of derealization, depersonalization. There are numerous disorders, intoxications and other conditions that have been described in the context of AIWS. In childhood, AIWS is most commonly linked with migraine and encephalitis caused by Epstein–Barr virus. Migraines are frequently thought to be the main cause in adults. Other possible underlying conditions of the AIWS include infections, epilepsy and intoxications.Treatment must be directed toward the presumed underlying disease whenever it is deemed necessary and beneficial.
Conclusions: Alice in Wonderland syndrome is still poorly known condition and probably misdiagnosed for the lack of clear and universally accepted diagnostic criteria. It can occur at any age, but appears to be more common during childhood and adolescence. The most common symptoms include: distortions of the size, mass or shape of the patient’s own body.

KEYWORDS

Alice in Wonderland Syndrome, Migraine, Visual Distortions

CITATION

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Introduction:

In 1955, the Alice-in-Wonderland syndrome (AIWS) was defined by English psychiatrist John Todd (1914–1987) as self-experienced paroxysmal body-image illusions involving distortions of the size, mass or shape of the patient’s own body or its position in space. [1] Alterations in perception of time, derealization, depersonalization, and somatopsychic duality (sense of being divided into two) are possible additional symptoms. [3] Individuals experiencing AIWS remain aware of the illusory nature of the perception. [4]

Todd named the syndrome for the perceptual disorder of altered body image experienced by the protagonist in ‘Alice’s Adventures in Wonderland’ (1865) [2] - a novel by Lewis Carroll, the pseudonym of the English writer Charles Lutwidge Dodgson (1832–1898).[1]

In the novel, Alice dreamt that she followed a white rabbit down a large hole and fell down into a strange world. Her body subsequently shrank in response to drinking a bottle marked “DRINK ME”. Her body also grew in response to eating a cake labeled “EAT ME”. After consuming the cake she felt that her body was expanding like a telescope until her head, now 9 feet above the floor, struck the roof. Her body size and relation to her surroundings changed rapidly. [2]

A description of symptoms associated with Alice in Wonderland syndrome was first published in medical literature by Caro W. Lippman in 1952, in which he described two patients who said they experienced a sensation of becoming short and wide during attacks of migraine headache. [5] One patient referred to this sensation as her “Tweedledum or Tweedledee feeling”, referring to the short, barreled-shaped characters depicted in Lewis Carroll’s 1871 sequel to ‘Alice’s Adventures in Wonderland’, entitled ‘Through the Looking Glass’ and What Alice Found There’. Lippmann believed that Lewis Carroll suffered from migraine headaches, and was inspired by his migrainous attacks in creating Alice and her magical wonderland.

AIWS has been discovered to be linked with infections caused by pathogens like Epstein Barr virus, migraines, depression, epilepsy and delirium. [3,5]

Material and methods:

The PubMed database was searched using the term: ”Alice in Wonderland Syndrome”. After a preliminary analysis, case reports, reviews and research articles in English language were included.

Key findings:

Symptoms:

Alice in Wonderland syndrome (AIWS) is a perceptual disorder characterized by distortions of sensory perception rather than hallucinations or illusions. [3] Affected individuals can experience visual illusions, including dysmetropsia, namely: micropsia (objects appear small), macropsia (objects appear large), teleopsia (objects appear further away than they are) and pelopsia (objects appear closer than they are). [2,7] Strikingly, micropsia and macropsia have been described most frequently in the literature, which might indicate that they are the most prevalent types of distortion but also that they are the best known and therefore studied most frequently. [3] People with this syndrome can aslo experience image distortions such as for instance having a horn growing on their forehead as a unicorn. [6] In addition to the metamorphopsias of micropsia, macropsia, telopsia and pelopsia, another distinct hallucinations includes animals. These hallucinations can include swarms of little animals (e.g.insects, mice, etc.) or confined bunches of bigger creatures for exmaple elephants, mutts and are referred to as zoopsia. [2,7]

Depth perception can be modified too, where distances look incorrect; for example a hallway may appear to be exceptionally long or the sky may appear too close or far away. Moreover, individuals can also experience sound distortion, such as soft sounds appearing amplified or even a misinterpretation of common sounds. [2] Other symptoms involve disorders of consciousness, as feelings of derealization, depersonalization. [3] Patients with Alice in Wonderland Syndrome might have distorted experience of time such as retrospective time-judgment errors, inability to correctly assess the passage of on-going time, inability to sense the passage of on-going time, quick-motion and slow-motion phenomena, bizarre alterations in the perception of time. [8,9] Other less commonly experienced symptoms may include loss of limb control and discoordination, memory loss, lingering touch and sound sensations, emotional instability, somatopsychic duality (i.e., the idea to be split in two, more often vertically in the middle), nausea, dizziness and agitation. [2,7,10]

Despite the lack of rigorous, standardized diagnostic criteria, attempts have been made to further categorize AIWS. Based on the symptoms that were recorded, Lanska and Lanska divided 81 cases of AIWS into three categories: somesthetic- (Type A), visual- (Type B) and somesthetic and visual alterations in perception (Type C). Somesthetic alteration in perception involve distortions in perceiving one’s body or environment, like partial or total macrosomatognosia or microsomatognosia; paraschematia. Visual perception alterations include symptoms such as micropsia, macropsia, pelopsia, and telopsia. [11]

Building on this classification, in 2015, Mastria designated Type A, B and C features as obligatory symptoms, while symptoms of derealization, depersonalization, somatopsychotic duality, and change in time perception were defined as facultative. [7]

Epidemiology:

The precise predominance of the AIWS is obscure, for a few reasons. To begin with, no epidemiologic studies on large scale are available. Second, the lack of univocally accepted diagnostic criteria for AIWS casts a shadow on reported data that ought to be considered carefully. Although it is generally assumed that the syndrome is rare, some clinical studies among patients with migraine indicate that the prevalence rate in this group may be around 15%. In addition, some studies indicate that individual symptoms of AIWS are not rare in the general population. [3,7] One study showed that ad hoc questions targeting AIWS symptoms at the first visit can reveal a prevalence of around 19% in a tertiary referral headache unit. [12] Children and young adults appear to be more susceptible to this syndrome. [13]

Etiology:

There are numerous disorders, intoxications, and other conditions that have been described in the context of AIWS. In childhood, AIWS is most commonly linked with encephalitis caused by the Epstein–Barr virus and migraine. Migraines are frequently thought to be the main cause in adults. Other possible underlying conditions of the AIWS include various viral infections, epilepsy, intoxications and fever. Complete remission is usual, however AIWS can last for years. In the active stages of chronic conditions like migraine, symptoms may reappear. [11]

Migraine

Aside from epidemiology, several other questions concerning AIWS remain unanswered. Todd considered migraine to be a major cause of AIWS and noted that many affected individuals have a personal or family history of migraine, but did not establish a temporal association of body-image illusions with migraine episodes in his cases. [1] To date, the temporal association of AIWS episodes with migraine attacks has not yet been investigated. On the one hand, the relationship between AIWS and migraine might be explained in the context of a general susceptibility of migraineurs to experience complex sensory and neuropsychological symptoms. This seems to be the case in children, where no temporal association between the AIWS episodes and migraine attacks have been clearly shown. On the other hand, this relationship might be more specific in adult migraineurs. [12]

The intriguing parallels between AIWS and migraine aura have led to discussions about shared pathophysiological principles, with some authors proposing AIWS as a distinctive form of migraine aura. One hypothesis suggests alterations in functional connectivity, especially in the visual cortex, underlying both conditions. Changes in the functional connectivity especially within visual networks are well documented for migraine patients with aura. While the pathophysiology of AIWS remains poorly understood, lesion-based and functional MRI (fMRI) studies indicate the involvement of various cerebral regions, also including visual networks like the extrastriate visual cortex, as well as the temporal-parietal-occipital Carrefour, a meeting point of temporooccipital, parietooccipital and temporoparietal junctions, relevant for integrating visual and somatosensory information. A recent resting-state fMRI study identified similarly reduced functional connectivity in the lateral and medial visual networks in AIWS patients and migraine patients with aura compared to healthy controls. These changes also affected areas like the lingual gyrus and the superior lateral occipital cortices known as "aura generator", suggesting shared underlying pathophysiological processes. Individuals with AIWS however, showed more extensive and noticeable changes in functional connectivity compared to those with migraine aura. In migraine patients, the extent of change in functional connectivity is linked with the complexity of migraine aura. Therefore, the more pronounced changes in AIWS patients might be attributed to the more complex symptomatology of AIWS. The observed relationship between AIWS and migraine with aura, coupled with the temporally linked onset of headaches and a similar time course raises the possibility of shared underlying processes. [11]

Epilepsy

The symptoms of Alice in Wonderland syndrome can be brought on by the severe localized electrical changes that take place in the brain during a seizure. These have been described to occur in association with seizures originating in the frontal, occipital and parietal lobes as well as temporal lobe epilepsy. AIWS symptoms might manifest as a component of the seizure itself or as part of the aura that precedes seizures. [2] Matsudaira T. et al described a case of an elderly patient with focal onset epilepsy whose seizures were documented by video EEG to investigate AIWS pathophysiology. The patient's focal awareness seizures consisted of turning both eyes to the left followed by macropsia, psychological time acceleration, derealization and fear. During macropsia, objects such as one's face appeared bigger on the left side. Video-EEG revealed multiple focal impaired awareness seizures accompanied by sudden motion arrest and subtle body movements. Ictal EEG changes arose over the right occipital region with small periodic positive discharges with evolution towards the right centro-parietal regions. They eventually ended with slow rhythmic waves in the right temporal region. Their patient demonstrated a relationship between epileptic seizures and AIWS, implying a common psychopathological basis. Ictal EEG changes suggested that her macropsia started from the right occipital region and propagated to the parietal and temporal regions, eventually manifesting symptoms such as psychological time acceleration and fear. [14]

Psychiatric disorders

Alice in Wonderland Syndrome can also be linked with psychiatric disorders. Mudgal V et al. reported a case of AIWS in a patient with schizophrenia. Mrs K, a 30-year-old female, was brought to the psychiatry OPD with complaints of suspiciousness, irritable behavior, decreased sleep and decline in household work. During mental status examination, it was revealed that she used to see her son in a miniature form, she tried to catch hold of him and keep it under protection, else some insect or rodent may harm him. The image was in external space, vivid and involuntary. Delusion of infidelity for her husband was observed. A diagnosis of paranoid schizophrenia was formulated and patient was treated with olanzapine. According to some researchers AIWS can be explained through a functional disconnection between primary and association visual cortices as a result of schizophrenic neurobiology and circuits. [15] Other reported causes of AIWS also include anxiety and depressive disorder. [1,3,16]

Infections:

Viruses

The Alice in Wonderland syndrome can be linked to a number of infections that result in encephalopathy. Epstein-Barr virus (EBV), influenza-A, H1N1 influenza, Coxsackie B1 virus, Lyme disease, herpes simplex, enterovirus, Zikavirus and varicella are among the documented causes. Of all cases due to infection, EBV is the most common infection type. [2,5]

Well-described efects of EBV infections in the CNS that could be responsible for symptoms in AIWS include: parenchymal edema, acute encephalitis and focal reductions of cerebral blood flow. Evidence supporting alteration in cerebral perfusion as a etiologic mechanism for AIWS include a study of 4 patients with acute symptoms, including two with documented EBV infection, all of whom were found to have decreased cerebral blood flow to the visual tracts, visual cortex and portions of the temporal lobe.

Few reports link AIWS to VZV infection and no clear pathophysiological etiology has been established for the development of VZV-related AIWS. Possible mechanisms include direct parenchymal infection, subacute vascular dysfunction and immune-mediated processes as described for other VZV-associated neurological disorders, including encephalitis and meningitis in both children and adults, whether during primary infection or during virus reactivation. [17]

There were five cases of AIWS associated with influenza virus infection. Onset of AIWS in four cases occurred after recovery from infectious symptoms. Omata et al. suggest that AIWS is induced by an autoimmune mechanism because AIWS-associated symptoms in their patient started after mycoplasma infection was ameliorated. A similar autoimmune mechanism might be also associated with onset of AIWS in cases decribed by Kubota K. et al., but the detailed etiology has not yet been elucidated. Although most symptoms of AIWS associated with influenza virus infection were isolated visual perceptual disorders such as micropsia and macropsia, other symptoms were also seen. Various central nervous system (CNS) manifestations can occur during influenza infection The susceptibility of the CNS owing to influenza may therefore be associated with the onset of AIWS. [18]

Bacteria

Single cases of AIWS have been reported in association with Lyme neuroborreliosis, Mycoplasma pneumoniae infection and Streptococcus pyogenes pharyngotonsillitis, all in children. As for AIWS caused by bacterial infections, proposed pathogenic mechanisms include direct neuronal infection followed by production of infammatory mediators or infltration by infammatory or immune cells, indirect neuronal harm due to development of autoimmune responses that include the release of autoantibodies and/or immune complexes, and hypoperfusion. [17] Omata T. et al. described a case of a patient with AIWS induced by mycoplasma infection. A 7-year and 11-month-old girl arrived with a fever (temperature, 40°C) and a cough. Even though the fever subsided after about 10 days, she remarked that her mother's face unexpectedly seemed smaller to her. Subsequently, she complained that objects intermittently appeared smaller than normal. Particle agglutination test indicated elevated serum antibodies against Mycoplasma pneumoniae. The patient was therefore diagnosed with AIWS secondary to mycoplasma infection. Reduced blood flow to the occipital and parietal lobes, which causes ischemia in these regions, including the visual cortex, as visualized on single-photon emission computed tomography (SPECT), may potentially explain AIWS in this case. [19]

Prions

Pandey S. et al. described a rare presentation of sporadic Creutzfeldt-Jakob disease (CJD) with Alice in Wonderland syndrome as one of the early manifestations. Although visual symptoms, such as diminution of vision, hemianopia, macropsia, and micropsia, have been reported in Heidenhain variant of sporadic CJD, the occurrence of Alice in Wonderland syndrome was an unusual feature as in this case. Magnetic resonance imaging (MRI) of the brain showed DWI restriction along fronto-parieto-occipital cortex, characteristic of cortical ribboning and electroencephalogram demonstrated sharp wave discharges at irregular intervals with background slowing. The presentation of AIWS in CJD isn't well known. Only a few other case reports are available. The AIWS observed here is persistent and non-fleeting as evidenced from the case reports. [20]

Only one report of AIWS caused by protozoan infection exists. [17]

Intoxicants and medications

The strong hallucinogenic effects of lysergic acid diethylamide (LSD) can cause a brief episode of Alice in Wonderland. After intoxication with LSD, individuals may experience distortions of their vision, false progression of their own body or external objects through space and reoccurring hallucinations of geometric patterns. Palinopsia, the illusion of persistence of a visual image, after the object has left the field of vision, can follow LSD ingestion. Repeated users of LSD have reported experiencing hallucinations that look like

stereoscopic camera images. [2] Dugauquier A. and Bidgoli S. described the case of a child affected by typical symptoms of Alice in Wonderland syndrome which was related to the methylphenidate treatment he was taking for an attention deficit hyperactivity disorder (ADHD). A 12-year-old boy was brough to ophthalmologic office due to his complaints of experiencing micropsia and macropsia. His mother was concerned about his mental well-being because he was already suffering from a disruptive ADHD, treated by methylphenidate for 2 years. The symptoms began 2 months earlier, in the context of a presumed bacterial bronchitis, treated by an amoxicillin/clavulanic acid combination and a codeine cough syrup for 1 week. This episode coincided with a modification in his ADHD treatment regimen: he transitioned from taking methylphenidate 10mg twice daily to using modified release methylphenidate 20mg once each morning. At that moment, the parents stopped the treatment and the symptoms vanished. [21] There is also a described case of a 67-year-old woman, with a relevant history of depressive disorder, nontoxic goiter and dyslipidemia that presented short episodes of altered body perception associated with altered external space perception for a period of one week. Her physical examination was unremarkable and her cerebral computerized axial tomography (cerebral CAT) and blood tests revealed no changes. She had been medicated with sertraline till the previous year and had restarted it in the previous month. She reported similar episodes of body image distortions in the first weeks of initiating sertraline for the first time. The recent episode continued for two weeks and during a one-year follow-up, she indicated no recurrence. The patient was diagnosed with AIWS probably induced by sertraline, being the first reported case of the kind. [22] Additionally, there are other medications, such as: cough syrup (containing dihydrocodeine and DL-methylephedrine), montelukast, oseltamivir, risperidone, topiramate, aripiprazole that may cause AIWS. [3,23]

Other

Alice in Wonderland syndrome has also been described in individual cases of other disorders. It can be the initial and sole manifestation of ischaemic stroke. or it can present itself following an ischemic stroke.[24,25] It was reported to manifest as aura accompanying cluster headache**.** [26] There is aslo known case of AIWS in a woman with a right temporo-parietal cavernoma. [27] Entezami P. et al. reported a patient who developed symptoms of AWS postoperatively. A 48-year-old man presented in shunt failure, attributed to a proximal catheter occlusion. Operative revision with replacement of the proximal catheter was performed without incident. After the surgery, he reported experiencing visual disturbances, such as the perception that people had small heads on full-sized bodies. Symptoms resolved postoperatively. The patient's symptoms were diagnosed as a transient episode of AWS. This was attributed to manipulation of the parieto-occipital cortex during the revision. The local inflammatory response from manipulation of that area is thought to have caused patient's symptoms. [28] Kobayashi Y. et al. reported two cases of AIWS with isolated cortical venous thrombosis. Both patients had lesions in the right occipital lobe and exhibited visual distortions. [29] AIWS was also decribed in patinets with dementia with Lewy bodies, [30] temporal-occipital glioblastoma, [31] PRES and multiple myeloma. [32]

Treatment:

The majority of AIWS cases, both clinical and nonclinical, are regarded as benign since complete symptom remission is frequently achieved, sometimes on its own and in other situations following appropriate therapy. However, symptoms tend to reoccur in accordance with active periods of the disease in clinical cases with an underlying chronic condition. Treatment must be directed toward the presumed underlying disease whenever it is deemed necessary and beneficial. In clinical practice, this typically entails prescribing antibiotics, antiviral medications, migraine preventatives, or antiepileptics. [3,33] Every case of AIWS requires systematic, multidisciplinary approach. [34]

Conclusion:

First described in 1955, Alice in Wonderland syndrome (AIWS) is a perceptual disorder characterized by distortions of visual perception (metamorphopsias), the body schema and the experience of time. In addition to these alterations in perception, patients also experience hallucinations or illusions of expansion, reduction or distortion of their own body image. A wide range of illnesses, intoxications, and other diseases have been associated with AIWS. In childhood, AIWS is most commonly linked with encephalitis caused by the Epstein–Barr virus and migraine. Migraines are frequently thought to be the main cause in adults. Alice in Wonderland syndrome can occur at any age, but appears to be more common during childhood and adolescence. The outcome is usually benign, particularly in children. Most often, patients outgrow these episodes. The long term prognosis typically depends on the etiology of the condition and the underlying condition must be evaluated. Treatment must be directed at the underlying condition. It is still poorly known condition and probably misdiagnosed for the lack of clear and universally accepted diagnostic criteria.

Disclosure:

Conceptualization: Natalia Bębenek, Gabriela Barszcz, Monika Błądek, Karolina Drygała, Justyna Bogdan, Izabela Harpula, Patryk Brzezicki

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