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# ALICE IN WONDERLAND: THE RARE DISEASE IN CLINICAL RESEARCH

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**ABSTRACT**

This review article provides an overview of the current understanding of Alice in Wonderland syndrome (AIWS), including its epidemiology, clinical manifestation, and diagnostic criteria. AIWS is a rare neurological disorder that affects the perception of sensory stimuli, particularly visual, auditory, and tactile stimuli. The condition is characterized by a range of symptoms, including hallucinations, illusions, and distortions of time, space, and body image. AIWS is often associated with other medical conditions such as migraine, epilepsy, and viral infections and has been reported to occur as a result of the use of certain psychoactive drugs. This article aims to provide a comprehensive understanding of AIWS and to highlight the need for further research into its pathophysiology, diagnosis, and treatment. By improving our understanding of this intriguing disorder, we hope to better support those living with AIWS and their families and ultimately improve their quality of life.

**KEYWORDS :** Rare disease, AIWS, diagnosis, treatment, clinical research, MRI.

## Introduction

There are several delineations of "rare" or "orphan" conditions, and these delineations may differ among countries. Common to all delineations is the low frequency of a complaint and the perception that treatments and exploration related to a specific complaint are scarce. In 1983, the United States (U.S.) Congress passed the "Orphan Drug Act" [1]. Using this description, it's estimated that over 7000 rare conditions affect an estimated 25 – 30 million people with a rare complaint in the U.S. (8 – 12% of the population). The Orphan Drug Act also designates conditions as "rare" if they affect more than 200,000 persons in the U.S. There's no reasonable anticipation that the cost of developing and making available in the U.S. a medicine for a similar complaint or condition will be recovered from sales in the U.S. of similar medicine [1].

The clinical donation, natural history, pathophysiology, and frequently mysterious nature of rare conditions have fascinated physicians for centuries. Rare conditions give openings to study mortal physiology and biomedical wisdom from unique perspectives. Major scientific improvements performing from disquisition of rare conditions have frequently handed sapience into more common diseases. The satisfaction of diagnosing a case with a rare complaint successfully is frequently fleetly combated by the consummation that the capability to understand and treat the case's condition is limited by ignorance and the difficulties of studying the complaint. Also, for the "intriguing" case with a rare complaint, being a "fascinoma" to physicians may consolidate suffering. Cases may feel that their physicians are in league with the "intriguing" complaint. Likewise, for cases with a rare complaint, the complaint is no longer rare — it is a constant part of their lives and the lives of their families [1].

Alice in Wonderland syndrome (AIWS) is a rare neurological complaint characterized by deformations of visual perception (metamorphopsias), the body image, and the experience of time, along with derealization and depersonalization. You may also find that the room you're in — or the surrounding furniture — seems to shift and feel further away or nearer than it really is. They're caused by changes in how your brain perceives the terrain you're in and how your body looks. AIWS primarily affects children and young adults [18].

## Natural history of Alice in Wonderland

The term Alice in Wonderland pattern was introduced in 1955 by the British psychiatrist John Todd (1914 – 1987) to cover a group of symptoms privately associated with migraine and epilepsy, although not confined to these diseases". As envisaged by Todd, the group comprised derealization, depersonalization, hyperschematia, hyposchematia, and somatopsychic duality, as well as illusory changes in the size, distance, or position of stationary objects in the visual field; illusory passions of levitation; and illusory differences in the sense of the passage of time. Apropos, Todd was well apprehensive that he wasn't the first to describe those individual symptoms. numerous of them had appeared before in the literature on fever, on general neurology, and on dogfaces with occipital injuries after World Wars I and II. also, in 1933 and 1952, Coleman and Lippman, independently, had formerly drawn comparisons between those symptoms and the gests of Alice in Wonderland, albeit without turning the name into an eponym. Lippman was also the first to suggest that the fleshly changes endured by Alice might well be inspired by body schema visions Lewis Carroll had endured himself. Carroll (alias of the British mathematician Charles Lutwidge Dodgson, 1832 – 1898) had migraines, and his journals indicate that his attacks were occasionally anteceded by audial marvels. still, chroniclers consider Lippman's thesis inconclusive, as the journals fail to demonstrate that Dodgson endured any audial marvels before he wrote his book. An indispensable thesis is that Dodgson had knowledge of — or maybe had experimented with — the hallucinogenic mushroom Amanita muscaria. Whatever the exact course of events may have been, with Alice in Wonderland, Dodgson created a character that appealed as important to croakers as it did to the book's intended followership. And Todd, by espousing the name, chose a memorable moniker for a group of symptoms heretofore described in insulation of each other

## Epidemiology

The exact frequency of AIWS is unknown for several reasons. First, no large-scale epidemiological studies are available. Second, the lack of universally accepted individual criteria for AIWS casts a shadow on reported data that should also be carefully considered. A study conducted on 3224 Japanese adolescents, aged 13 to 18 years, demonstrated that the occurrence of micropsia and macropsia was 6.5 in boys and 7.3 in girls, suggesting that the visual visions of AIWS may not be so rare in the general population. The female rate seems to vary with age while at a younger age, boys are generally affected with a 2.69-fold higher risk of having AIWS (in a sample aged 5 to 14 years). No significant differences in coitus frequency were recorded in inferior scholars (13 – 15 years old) by Abe et al., while ladies were significantly more current (56.7) among elderly scholars (16 – 18 years) [7].

Out of 166 published cases of AIWS, the most common cause is migraine (27.1%), followed by infections (22.9%), basically EBV (15.7%). In dwindling order, other etiologies are as follows: brain lesions (7.8%), cure (6%) and medicines (6%),

psychiatric diseases (3.6%), epilepsy (3%), complaint of the supplemental nervous system (1.2%), and others (3%). In about 20% of cases, no cause of AIWS was found. In 65% of cases, AIWS passed in children under 18 years of age [7].

**Pathophysiology**

The symptoms of AIWS are attributed to functional and structural rarities of the perceptual system. On the whole, central pathology is considered the most current cause; still, dysmorphopsia, for illustration, is also endured in the environment of retinal ablation and some other types of eye complaint, and plagiopsia (visual cock) is also endured in the environment of complicate complaint. nonetheless, utmost symptoms of AIWS are attributed to centrally located neuron populations and indeed cell columns that respond widely to specific types of sensitive input (for vision, especially cortical areas V1 – V5) Area V4 of the extrastriate visual cortex, for illustration, responds widely to color, whereas area V5 responds to movement. Both areas also respond to shape and depth, but bilateral loss of function of V4 results in achromatopsia (the incapability to see color) and bilateral loss of V5 results in akinetopsia (the incapability to see stir). The incapability to visually perceive perpendicular lines (plagiopsia) or lines under a different angle is attributed to loss of function of exposure columns that are grouped together throughout the vertical layers of visual cortex. also, colorful neuron populations have been linked as being responsible for interceding different types of metamorphopsia, and for other metamorphopsias educated suppositions have been made. Sometimes this involves advanced- order mismatches between larger factors of the visual network, which can vary inter individually. An illustration of the ultimate situation can be set up in complex types of prosopometamorphopsia, in which mortal faces may be perceived constantly as beast faces, and indeed in an supposedly straightforward symptom similar as micropsia, which was set up to be associated with a harmonious pattern of occipital hypoactivation and parietal hyperactivation in an fMRI study.

Mutatis mutandis, the same would feel to hold true for somesthetic deformations, in the sense that functional and/ or structural rarities of specific neuron populations in somatosensory cortical areas are responsible for interceding body schema visions similar as microsomatognosia, palisomesthesia, aschematia, etc. In these cases, a corridor of the network located around the parieto- temporo- occipital junction is responsible, although then to a mismatch between advanced-order factors of the network as a total may be at play, as in ischemia of the distal corridor of the anterior cerebral highways that supply corridor of the perceptual network responsible for integrating compound sensitive data for mindfulness of the body schema [6].

**Diagnosis**

The normal of age to the opinion was9.5 ±3.8 times (range 4-16 times). It appeared in an acute way in 85 and progressive in 15. 90 had micropsias and/ or macropsias, 85 deformation of the form of the objects, 80 relegation of objects, 45 disturbances of body image, 45 acceleration of the time and 30 sensation of vision. 95 of the children had numerous occurrences a day; these occurrences lasted less than 3 twinkles in 90. Electroencephalogram was realized in all the cases, it was abnormal in 11 cases, in one case was set up and epileptic foci (left temporal) and in 10 cases was set up posterior slow swells. The tests of neuroimage were normal in all the cases. The visual elicited capabilities were realized in 7 children; five of these children showed advanced breadth in elicited capabilities and two of these children had normal. The contagious etiology was set up in nine cases (five mates to Epstein- Barr contagion), migraine in eight, poisons in two and epilepsy in one case. 80 didn't have rush [9].

**Differential diagnosis**

Often, these episodes with hallucinations and metamorphopsia are very impressive. Even if Todd and Lippmann described an association with epilepsy and other underlying pathology, we suggest that the term AIW syndrome should only be applied when differential diagnoses like occipital lobe A. epilepsy, encephalitis and psychosis were ruled out. Even if these differential diagnoses are grave, most children do not require invasive diagnostic procedures. The majority of AIW episodes does not last longer than a few minutes, and encephalitis or intoxication as well as psychosis can be ruled out on clinical grounds. We did not find any case report of a tumour as an underlying cause. Thus, there is only a relative indication for cranial imaging like CT or MRI. As metamorphopsia can also be a symptom of (e.g. occipital lobe) epilepsy, an EEG recording is suggested [10].

**Screening Methods**

Case A (fictitious original, for 'Alice') was signed in the environment of an imaging study carried out by the Parnassia Bavo Group (PBG) and the University Medical Centre Utrecht in the Netherlands from among individualities with a schizophrenia diapason complaint and VAHs. The study was designed to gain functional MRI (fMRI) data during occurrences of VAH exertion, and to employ the preceding cerebral activation maps for an experimental treatment with rTMS. On the day of scanning, the PANSS( Positive and Negative Pattern Scale) was used to assess the current symptomatology. Detailed characteristics of the VAHs were assessed with the PSYRATS Auditory Hallucinations Rating Scale, and the Hallucination Isolation List, semi-structured interview developed at the PBG. All clinical conditions were performed by trained canvassers. The study was approved by the Humans Ethics Committee of the University Medical Centre Utrecht. After a complete description of the study to the subjects, written informed concurrence was attained [11]

**Experimental Design and Data Acquisition**

Imaging was carried out on a Philips Achieva 3-T clinical MRI scanner. Eight hundred BOLD fMRI images were acquired with the following parameter settings: 40 (coronal) slices, TR/TE 21.75/32.4 ms, flip angle 10°, FOV 224, 256 ,160, matrix 64,64, 40, and voxel size 4 mm isotropic. This scan sequence achieves full brain coverage within 609 ms by combining a 3DPRESTO pulse sequence with parallel imaging (SENSE) in two directions, using a commercial 8-channel SENSE head coil. After completion of the functional scans, a high-resolution anatomical scan, with parameters TR/TE: 9.86/4.6 ms, voxels, and flip angle 8°, was acquired to improve localization of the functional data. Scanning time for functional imaging was 8 min and 7.2 s, and 8 min for structural scanning. In their right hand, the subjects held an fMRI-compatible button (custom-designed for studying hallucinatory activity) which they were required to press at the onset of VAHs (onset of 'HALLUCINATION' condition), and to release at the termination of each hallucinatory episode (onset of 'REST' condition). The onset times and duration of the button presses were recorded, and employed as the basis for model fitting [11].

**Treatment And Prognosis**

Utmost nonclinical and clinical cases of AIWS are considered benign, in the sense that full absolution of the symptoms can frequently be attained, occasionally spontaneously and in other cases after proper treatment. still, in clinical cases with an underpinning habitual condition (similar as migraine and epilepsy), symptoms tend to reoccur in concordance with active phases of the complaint, and in cases of encephalitis the prognostic may also vary. As a consequence, the need to treat requires careful assessment, proper knowledge of the natural course of the colorful beginning conditions that are possible, and a careful explanation to the case of what to anticipate from which rectifiers under which circumstances. In

numerous cases consolation will serve. Whenever treatment is considered useful and necessary, it needs to be aimed at the suspected underpinning condition. In clinical practice this substantially involves the tradition of antiepileptics, migraine prophylaxes, antiviral agents, or antibiotics. The literature indicates that antipsychotics are infrequently prescribed7 and that in utmost cases their effectiveness is considered borderline. also, when deformations are endured as comorbid symptoms in cases with psychosis, it's important to take into account the possibility that they can occasionally be convinced or exacerbated by antipsychotics because of their implicit to lower the threshold for epileptic exertion [5].

## Clinical Manifestation

Really, it could be said that the so called AIWS is a clear illustration of what used to be defined as cryptogenic — a clinical incarnation of supposedly unconnected pleiotropic symptoms, with a secret background, a neurological etiology lying inside the depths of the neural fabric. Over the once 60 times, a aggregate of around 42 somatosensory symptoms have been associated with the AIWS. generally, the pattern presents during non age, which may be considered a precursor of migraine; children feel to be most susceptible to develop migraines in the future, or during early non age, but this doesn't avert the possibility of adult clinical cases. There's no coitus partiality, except for migraine- associated cases that are current in ladies. In general, the symptoms don't present contemporaneously with migraine, but just before of after the migraine occasion. The pattern is characterized by the presence of flash occurrences of visual visions and distorted perception. The top clinical incarnation is a malformed perception of the body image, where the case is completely apprehensive of what's going on, with no mindfulness revision. Cases are under the print that different body corridor are changing.

Also, the sense of time is also affected; time may feel to pass veritably sluggishly or just the contrary, that everything happens in a veritably short time. Some cases witness visual visions, changes in size, distance, or the position of stationary objects with frequent deformation of the circumstances around the event. There may be differences in hail and tactile perception, and occurrences of depersonalization and derealization, somatophycic duality, and felling like levitating. Others witness fear and indeed terror during the event. he occurrences may present several times during the day and last lower than 24 hours, with a launch and end in utmost cases. To a large extent, the donation is episodic and some cases may evolve towards regularity. Some findings indicate that metamorphopsias may seen to last for seconds or twinkles and will be altered after fixing the sight on 1 single object; still, during that perception process, the objects won't be seen as distorted. The explanation may be cerebral asthenopia, interpreted as the fatigability of the perception system (MRI) studies have linked an occipital hypoactivation and a hyperactivation of the parietal lobe when micropsia occurs. [15].

## Case Study Report

A 30-year-old male patient applied to our clinic with complaint of long term head-ache episode. The patient presented with a pulsating headache on left side of his head which had lasted for approximately 24 hours and usually occurred once a week in the form of attack. During his headaches nausea was accompanied with photophobia and phonophobia the patient stated that the object around him appeared to be larger than they actually were,that his fingers appeared to be smaller than they actually were and that he noticed deformation in the objects around him. head ache started after approximately 30 minutes after these symptoms. These auras are not seen in or after every attack. He had seen the visual phenomenon for about one year he told that all these visual distortion would continue for almost half an hour. besides his mother and sister, there were no significant characteristics in the patient background. His psychiatric examination was normal, results from neurological and systemic examination of the patient were normal and his blood test results were at normal limits. No pathologies were detected in MRI and EEG were taken. Two of them were normally. The patient was administered 500mg\day of valporic acid complaints improved, but repeated during follow ups valporic acid were increased to 1000mg/day and the complaints of the patients were completely eliminated [19].

## Conclusion

Since 1955, no further than 169 case descriptions of AIWS have been published. The literature indicates that this may be only the tip of the icicle, with numerous individual symptoms of AIWS being endured (albeit sometimes and only fleetingly) by over to 30 of adolescents in the general population. Although consolation seems to serve in roughly half of the clinical cases, the dubitation of a central origin of the symptoms should prompt supplementary examinations in the form of blood tests, EEG, and brain MRI. Although firm substantiation to justify these supplementary examinations is lacking, I recommend them on clinical grounds because of the diapason of known etiologies and the prospect of bettered outgrowth in a substantial number of cases after acceptable treatment. Treatment, if necessary, needs to be directed at the suspected underpinning cause. Regarding exploration, much larger case sample sizes are demanded to allow for sufficient statistical power of empirical studies of AIWS and its individual symptoms. In addition, epidemiologic checks in the population at large are demanded to establish sound frequence data. As an volition or an adjuvant strategy, one might consider creating an transnational database for cases of AIWS, with special attention paid to phenomenological characteristics, individual findings (including substance abuse), natural course, and treatment results. For such a database to be effective, all new cases of AIWS should be subordinated to a methodical assessment, including proper history- taking, neurologic and other physical examinations, and supplementary examinations. In habitual cases, functional imaging ways may be helpful in establishing specific neurobiological supplements of individual symptoms (although there are frequently colourful practical obstacles to be overcome). AIWS is in need of proper representation in transnational individual groups similar as the ICD (for illustration under the title of "conditions of the Nervous System, Episodic and ferocious diseases" or "Other diseases of the Nervous System") and the DSM (rather under a new title called "Perceptual diseases" which in unborn editions might also include other nonpsychotic perceptual diseases similar as the Charles Bonnet pattern, exploding head pattern, and cenesthesiopathy). Last but not least, our sapience into the nature of AIWS might be enhanced by network analyses of the collective connections of individual symptoms as well as their connections with the perceptual networks underpinning them. In the meantime, still, it's possible to carry out a careful individual procedure to help this incompletely hidden group of cases gain a proper opinion and, if possible, acceptable consolation and, if necessary, applicable treatment.

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