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AIWS (Alice in Wonderland Syndrome) Correlation with Migraine

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Abstract: ***Alice in wonderland syndrome is an uncommon and intriguing neuropsychiatric condition characterized by unusual visual hallucinations. The term "Alice in Wonderland syndrome (AIWS) " was derived from Lewis Carroll's world-famous novel" Alice's Adventures in Wonderland, "in which the protagonist "Alice" felt that the size and shape of her body varied in various settings. The pathophysiology of AIWS is still uncertain but there are several causes for this syndrome; however, EBV infection is the most prevalent cause in children, whereas migraine affects adults more frequently [1]. Many studies have found a strong link between migraine and AIWS. Patients with migraine who have AIWS exhibit aberrant perfusion in the medial temporal, hippocampus, temporo-occipital, or temporo-parieto-occipital areas. This review aims to find the correlation between migraine/aura and development of AIWS.***

Keywords: AWIS, Migraine, Aura, Metamorphopsia, temporo-parieto-occipital

1. Introduction

From the Fictional to the Real World: The Art-Disease Relationship [2].

In 1955, English psychiatrist John Todd (1914-1987) defined the Alice-in-Wonderland syndrome (AIWS) as self-experienced paroxysmal body-image illusion [3] Illness and art have always been interconnected, especially when neuropsychiatric diseases are involved [2]

Alice in Wonderland syndrome (AIWS), also known as Todd's syndrome, is a cognitive disorder, often characterized by impaired visual perception or metamorphosis, abnormal body schema, and distorted experience of time [1] This syndrome has been observed in patients with migraines, epilepsy, cerebral cortex lesions, and hallucinogen intoxicated states like LSD, delusional states, and schizophrenia [1].

Several well-known artists experienced migraine (e. g., Picasso, Lewis Carroll, and De Chirico) and some researchers think that their works could have been impelled by migraine features [3].

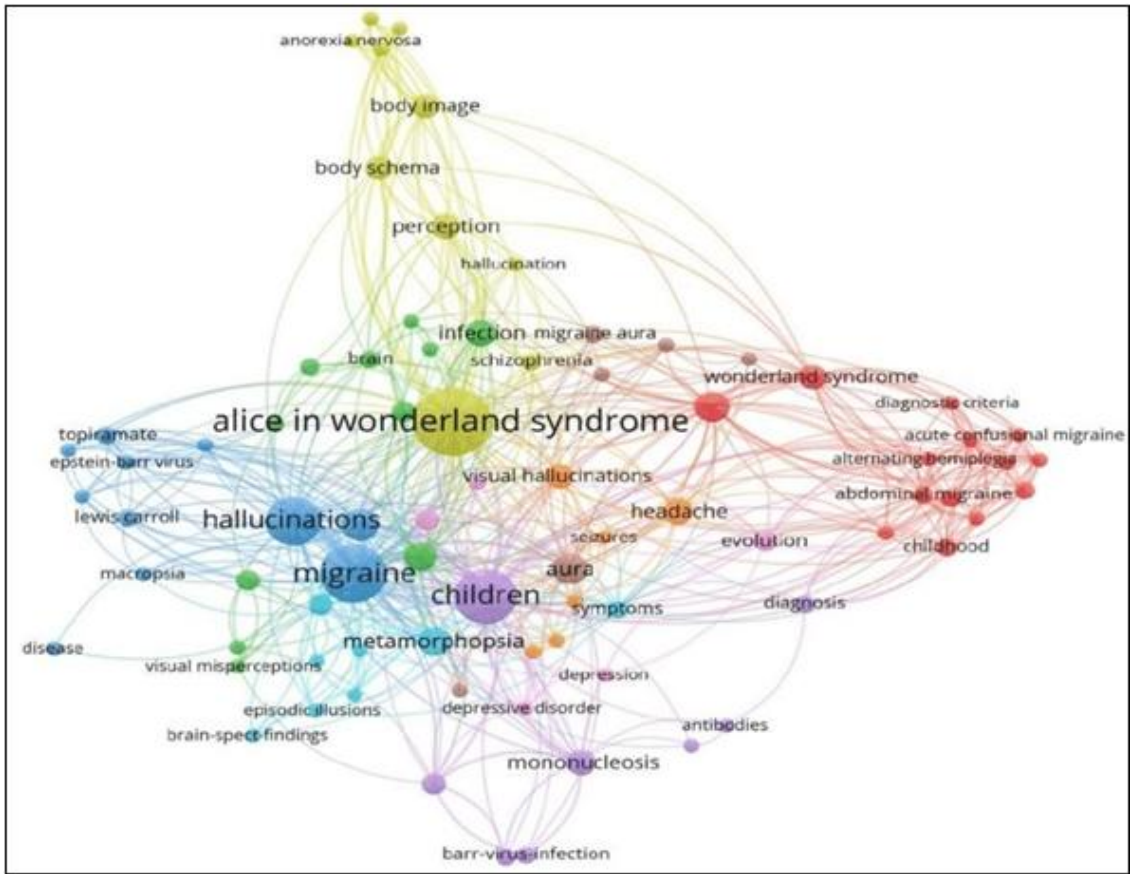

Figure 1: Mapping keywords associated with Alice in Wonderland syndrome (AIWS)

Symptoms of 'Alice in Wonderland' syndrome

Metamorphopsia:

- Body Image Distortion
- Dyschronometria

Metamorphopsia, a visual distortion of objects, is the major clinical neurologic symptom of AIWS. This perceptual disturbance may result from disorders of the retina, focal stimulation of visual associations and the parietal cortex, or inflammation of the oculo motor or vestibular systems [5].

It was discovered that the primary deficits in regional cerebral blood flow in individuals with AIWS were located in the temporal lobe, occipital lobe, and the neighboring area of the perisylvian fissure, all of which are adjacent to the visual pathway and the related visual cortex [5].

Previous documented etiologies for Alice in Wonderland syndrome include viral infections (especially Epstein-Barrvirus [EBV]), migraine, epilepsy, and central nervous system abnormalities [6].

AWS was linked with Epstein-Barrvirus (EBV) (48%) and migraine (11%) in a review of 81 cases, although it has also been described in the context of a wide range of other illnesses, including infections, toxicity, depression, and stroke [7]. AIWS is under recognized due to stigma and ignorance of its symptoms, which makes patients fearful of being recognized with mental illnesses [8] and it is a under explored syndrome despite neurological studies dating back over three decades [9].

The treatment is aimed at the specific pathology, and there is presently no recognized treatment for the symptoms associated with AIWS. The symptoms are rarely life-threatening, and the attacks normally pass within a few days to weeks [10].

Figure 2: Metamorphopsia

Neuroanatomical basis

AIWS originated in the parietal lobe (Todd 1955), a previous finding by Bollea (1948) that electrical stimulation of the posterior parietal cortex was able to induce autoscopic hallucinations and disturbances of body image, including illusions of somatic elongation and disappearance of all four limbs. The current consensus refers to a "critical zone" at the junction of the temporal, parietal, and occipital cortices, in the temporoparie to occipital carre four (TPO-C), where all somato sensory and visual information is integrated to construct an inner and exterior image of one's body [11].

AIWS and Migraine

Migraine is a multi faceted disease characterized by throbbing headaches of moderate to severe intensity, lasting four to 72 hours, and connected with photophobia/ phonophobia and/ or digestive symptoms (e. g., nausea, vomiting).

Migraine is the leading cause of AIWS in adults (27.6 percent) and the second leading cause in children (26.8 percent) [2].
Epidemiologic studies in patients with migraine have reported an Alice in Wonderland syndrome prevalence rate of upto 15% among this population [6].

Lippman recorded numerous individuals reporting symptoms of appearing unusually tall or short during migraine attacks in1952 [2]. Migraine has a wide-ranging impact on how the brain interprets sensory information.

Alice in Wonderland Syndrome, a disease frequently linked with migraine, is hypothesized to be caused by an abnormal integration of visual and somato sensory stimuli. However, there's very less information regarding the incidence of this illness among migraineurs, as well as the relationship between Alice in Wonderland Syndrome episodes and migraine attacks [12].

Current studies of migraine pathophysiology emphasize that migraine is a brain condition that affects various systems related to the processing and filtering of sensory inputs.

The neuronal effect of cortical spreading depression (CSD) on brain regions may explain the relationship between migraine and AIWS from a pathological stand point [13].

AIWS is more prevalent in children, and it may be due to anatomical differences between children's and adult brains: according to Flechsig's sequence of corticalmyelination, associative cortical regions are the last to mature. Thus, the immaturity of associative regions may explain why they are more sensitive to spreading depression than adult migraine sufferers. To date, the close relationship between migraine and AIWS supports this theory, but it is still speculative [14].

Migraine is a chronic neurological disorder characterized by intervals of well-being (interictal phase) and painful episodes (ictal phase). The basic pathophysiological changes, however, occur throughout both the interictal and ictal stages. The migrainous brain seems hyper responsive to recurrent sensory inputs (e. g., visual, somato sensory and

auditory) following a migraine episode, with no typical capacity to habituate to decrease neural burden depending on stimulus saliency and metabolic resources. This neurophysiological phenomenon, which has been seen in several studies, is referred to as the deficiency of habituation hypothesis.

Even when migraineurs are not in the ictal phase, their brains remain caught in a state of "never-ending attack." In truth, habituation deficiency is worse in severe situations and improves with proper therapy.

The absence of habituation is a cerebral corticalepi phenomenon caused by thalamocorticaldysrhythmia, which is an abnormal thalamocortical regulation dysfunction. This flaw has been linked to a variety of neurological and psychiatric diseases [12].

The prevalence of AIWS in migraineurs is around 20% and doubles when considering only migraine with aura (MA), therefore AIWS should not be considered as a rare disorder [12].

Diagnostic Criteria for Migraine-Related AIWS Proposal by Valence et al.

If a person experiences one or more episodes of self-experienced body schema illusion or metamorphopsia.

a) Duration < 30 min
b) Accompanied by headache or a history of migraine
c) RMI, CSF, and EEG all normal (visual evoked potentials maybe abnormal) [12]

AIWS is under estimated due to the unwillingness of patients to refer their symptoms to clinicians and due to the lack of data. [14]

- Migraine preventive therapy might be effective in reducing the number of, and even resolving, AIWS episodes.
- The strong association between AIWS and MA and the responsiveness to migraine preventive therapy suggests a causal role of cortical spreading depression (CSD)

If AIWS and migraine have the same pathophysiology, the drugs used for migraine prevention might influence AIWS [12].

The great variety of hallucinations' which precedes to headache in migraine is little known to the medical profession. Many migraine cases have noted the various hallucinations of taste, smell, hearing, irritability of the patient or its rarer opposite, euphoria, lassitude, which precedes the attack [15]

Aura

Inabout15%toone-third of migraineurs, headache onset is preceded or accompanied by aura [13]

Aura is a completely reversible localized neurological phenomenon characterized by visual, sensory, verbal, and/or motor symptoms that emerge gradually and frequently precede the headache phase.

It is distinguished by a sluggish start, symptoms lasting less than an hour, a combination of positive and negative elements, and total reversibility. Visual aura is the most common type of aura, affecting more than 90% of patients. When there are many aura symptoms, they usually occur in the following order: visual, sensory, aphasic. [13]

A migraine aura is a short neurological condition that often affects the visual fields and comes before to the headache phase. Aura symptoms include flashing lights that arise in the center of the vision and radiate out in unpredictable patterns to the periphery. [16]

Migraine symptoms with typical aura develop over 5-20 minutes and subside after60 minutes. Within 60 minutes of the aura, the headache generally develops. Numbness and tingling in the lips or fingers are possible somato sensory signs. They might also entail a substantial alteration in the way you perceive space and time. [17]

In recent decades, neurophysiological and functional neuro imaging studies have universally recognized cortical spreading depression (CSD) as a mechanism under pinning the aura phenomena.

Migraine with typical aura. The symptoms last little more than an hour, are a combination of good and negative characteristics, and are completely reversible. Migraine with aura, which is differentiated by its gradual onset and duration and consists of visual, sensory, and/or speech/ language symptoms but no motor weakness.

Migraine with typical aura. Migraine with aura, which has a delayed start, a one-hour duration for each symptom, a combination of positive and negative elements, and is completely reversible.

Typical aura without headache. Migraine with typical aura in which aura is neither accompanied nor followed by headache of any sort.

Migraine with brain stem aura. Migraine with aura symptoms clearly originating from the brain stem, but no motor weakness

Hemiplegic migraine. Migraine with aura including motor weakness.

Retinal migraine. Repeated attacks of monocular visual disturbance, including scintillations, scotomata, or blindness, associated with migraine headache

Retinal migraine is a rare cause of transient monocularvision loss. There have been cases of migraine-induced chronic monocular vision loss. Appropriate Investigations are required to rule out other causes of transient monocular blindness. [7]

Some symptoms, such as the Alice in Wonderland syndrome, are not classified as aura in the International Headache Classification, but are almost certainly very unusual aura. [18]

2. Conclusion

Because there are no clear and commonly accepted diagnostic criteria, AIWS is still poorly understood and likely misdiagnosed.

Most often misdiagnosed due to a lack of precise, globally acknowledged diagnostic criteria. AIWS clinical manifestations can be complicated and vary depending on the pathophysiological processes involved. Furthermore, multidisciplinary research at the institutional level should be promoted in order to better understand the neuropsychiatric pathophysiology of AIWS across groups. It is critical to promote and empower researchers and practitioners to use collaborative abilities and develop collective actions to better understand and avoid AIWS.

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