[CASE REPORT]

Complex Hallucinations and Panic Attacks in a 13–Year-Old with Migraines: The Alice in Wonderland Syndrome

by DIMPLE GEORGE, MBBS, DPM, MRCPSYCH; and PAUL BERNARD, MA, MB BCHIR, MRCPSYCH

Dr. George is Specialty Registrar in Child and Adolescent Psychiatry; and Dr. Bernard is a Consultant Child and Adolescent Psychiatrist at the Mulberry Center, Darlington Child and Adolescent Mental Health Service, Tees, Esk and Wear Valleys NHS Foundation Trust, Darlington, County Durham, United Kingdom.

Innov Clin Neurosci. 2013;10(1):30–32

FUNDING: There was no funding for the development and writing of this article.

FINANCIAL DISCLOSURES: The authors have no conflict of interest relevant to the content of this article.

ADDRESS CORRESPONDENCE TO: Dr. Dimple George, Specialty Registrar, Child and Adolescent Mental Health Service, Darlington, DL 3 6 HX United Kingdom; Phone: 0044 1325 743177; E-mail: Dimple.george@nhs.net

KEYWORDS: Alice in wonderland syndrome, complex hallucinations, panic attacks, migraine

ABSTRACT

This case report describes a 13-year-old girl whose family requested a referral from the pediatrician for Child and Adolescent Mental Health Services in order to understand her recent onset of bizarre behavior. On assessment, she was found to have episodes of complex audiovisual hallucinations and panic attacks with intervals of complete recovery associated with episodes of migraine headaches. The "Alice in Wonderland Syndrome," which is intimately associated with migraine and epilepsy, as well as a number of other neurological conditions, could explain her episodic neurobehavioral disturbance.

INTRODUCTION

The Alice in Wonderland Syndrome (AIWS) was described in 1955 by Todd as "a singular group of symptoms intimately associated with migraine and epilepsy."[1] These symptoms include bizarre disturbance of body image and bizarre perceptual distortions of form, size, movement, and color. Podoll et al[2] suggest that the syndrome denotes a variety of self-experienced paroxysmal body schema disturbances ("obligatory" core symptoms of the AIWS), which may co-occur with depersonalization, derealization, visual illusions, and disorders of time perception ("facultative" symptoms of the AIWS).

Lippman[3] drew attention to seven patients he had seen with migraines accompanied by unusual distortions of body image. Often these appeared as migraine equivalents in which the hallucination constituted the entire attack. In addition to migraine, AIWS has also been reported in association with epilepsy, Epstein Barr virus, and other infections of the central nervous system, intoxication with hallucinogenic drugs, hyperpyrexia, hypnagogic states, and schizophrenia.[4] This neurological syndrome derives its name from the book, *Alice's Adventures in Wonderland*, published in England in 1865 by Charles Lutwidge Dodgson under the pseudonym of

Lewis Carroll where from the opening scene, Alice jumps down a rabbit hole and has a series of fantastical experiences.[5] Follow-up studies have shown that AIWS is typically a benign, self-terminating childhood condition that requires no further diagnostic action nor specific treatment and no long-term medical monitoring.[6,7]

CASE PRESENTATION

A 13-year-old girl was referred to the Child and Adolescent Mental Health Team by a local pediatrician for the assessment and management of anxiety and stress. She had been diagnosed with migraines three months prior to her referral and had her first attack of migraine headaches nearly a year ago. Over the last 12-month period, she had experienced at least three episodes of frontal headaches with photophobia of moderate intensity lasting for several hours. The first attack settled with nonsteroidal anti-inflammatory medication, but the second attack resulted in a trip to the Accident and Emergency Department and subsequent admission to the Pediatric Ward. Neurological examination and all investigations, including an electroencephalogram and magnetic resonance imaging scan were normal.

During her migraine attacks, she described hearing hissing noises inside her head, which were initially muffled, but after several repetitions, these noises clearly sounded like the word "death" to her. They made her extremely frightened and panicky as they triggered thoughts about a dead relative. She also reported seeing a black image with red eyes coming toward her, which terrified her; she tried to kick it. Her family was upset and confused seeing their daughter's distress.

She described how during the attacks she thought that "time had slowed down," and that "getting out of bed was impossible" because she felt very dizzy looking at the floor, which seemed a long way down and she felt "she would fall." She was not able to dress herself because she had "no strength in her hands." She denied any body image disturbances or lingering sense of touch or sound that have been described in cases of AIWS.[8]

In between attacks, she experienced a sense of anticipatory anxiety. She was afraid to go to school fearing a migraine attack. She was worried about becoming an object of ridicule among friends. She also worried how her behavior during attacks impacted other members of her family, especially since her attacks were mostly at night and therefore, disturbed their sleep. All these symptoms settled spontaneously when the headache passed.

There is a family history of migraine in her paternal grandmother and migraine, stroke, and epilepsy in a paternal aunt. Her mother suffers from depression and her father has problems with substance abuse. Her developmental history was unremarkable. She had no previous contact with mental health services. There is also no significant past medical history. She is not known to use any illicit substances.

She was taking 1mg daily of pizotifen as well as 2.5mg of zolmitriptan for use during migraine attacks.

Hallucinations are a known side effect of pizotifen, but the patient described having these symptoms before taking the medication.[9] Since starting on pizotifen, she has had a reduction in the frequency and intensity of headaches as well as reduced perceptual disturbance and anxiety.

Sessions were arranged to address the anxiety through psychoeducation, relaxation training, and cognitive behavior therapy. When seen two weeks later, she had experienced only one additional attack, which was much milder in severity with less intense voice hearing and visual experiences lasting about 45 minutes. She remained well during her next two follow-up appointments with no further headaches, anxiety, or re-emergence of the perceptual disturbance.

DISCUSSION

It has been suggested that the clinical features seen in AIWS may be caused by abnormal amounts of electrical activity causing altered blood flow in parts of the brain that process visual perception and texture.[3] A long-term outcome study of a case series of 12 patients showed that symptoms of AIWS ceased within 1 week to 4 months. Patients experienced benign and self-terminating metamorphic hallucinations during childhood, with occasional recurrence and good prognosis. The AIWS showed hereditary predisposition with an increased prevalence in those with a positive family history of migraine.[7]

Acute migraine attacks occur in the context of an individual's inherent level of vulnerability. Attacks may be initiated when internal or environmental triggers are of sufficient intensity to activate a series of events, which culminate in the generation of a migraine headache, often preceded by a prodrome of vague vegetative or affective symptoms. This prodrome should be distinguished from the aura, which consists of focal neurological symptoms that may be either visual, sensory, language, or localized brainstem disturbance.[10]

Migraine aura can be spectacular and sometimes frightening. Studies of cerebral blood flow during the aura have shown that the classic slow progression of symptoms experienced corresponds to the rate of spreading bilateral oligemia, which forms the basis of the spreading depression theory, making it unlikely that vasodilatation is a cause of pain.[11,12]

Functional magnetic resonance imaging (MRI) studies have shown that migraine aura is not evoked by ischemia, but instead by aberrant firing of neurons and related cellular elements, which result in activation of the trigeminal/cervical nociceptive

neurons forming the basis for the central neural hypothesis of migraine.[13,14]

Molecular genetic studies have shown that migraine with aura, anxiety disorders, and major depression are associated with NcoI C allelic variations within the DRD_2 (dopamine D_2 receptor) gene.[15]

Reversible palinopsia and AIWS have been reported when topiramate has been used for migraine prophylaxis, with direct increase in symptoms on increased dosage and decrease on reduction or stopping the medication.[16,17] There have been no similar reports with pizotifen. In this case, the symptoms were present even before she started the medication and with increased dosage, it was associated with remission of her symptoms of migraine as well as remission of the AIWS. In other reports, the intensity and frequency of symptoms were said to diminish with effective antimigraine drug treatment and time.

CONCLUSION

This case demonstrates that clinicians working in child and adolescent mental health need to remain vigilant for the psychiatric manifestations of neurological conditions and their management. If not, this presentation might easily have been understood in purely psychological terms.

ACKNOWLEDGMENTS

We would like to thank the young person and her mother for permitting us to publish the findings as a case report.

CONSENT

We can confirm that we received the consent of the young person and her parents.

REFERENCES

1. Todd J. The syndrome of Alice in Wonderland. *Can Med Assoc*. 1955;73:701–704.
2. Podoll K, Ebel H, Robinson D, Nicola U.Obligatory and facultative symptoms of the Alice in Wonderland syndrome. *Minerva Med*. 2002;93:287–293.
3. Lippman CW. Certain hallucinations peculiar to migraine. *J Nerv Ment Dis.* 1952;116:346–351.
4. Kitchner N. Alice in Wonderland-syndrome. *J Neurol.* 2009;256: 340–354.
5. Evans RW, Rolak LA. The Alice in Wonderland syndrome. *Headache.* 2004;44:624–625.
6. Weidenfeld A, Borusiak P. Alice-in-Wonderland syndrome—a case-based update and long-term outcome in nine children. *Childs Nerv Syst.* 2011;27:893–896.
7. Jenke ACW, Borusiak P, Wagner A. Long-term outcome of Alice-in-Wonderland-syndrome: results from a case series of 12 patients. *Neuropediatrics*. 2010;41:174–304.
8. Kew J, Wright A, Halligan PW. Somaesthetic aura: the experience of "Alice in Wonderland." *Lancet*. 1998;351:1934–1935.
9. EMC. http//:www.medicines.org.uk/emc/medicine/24179/SPC/Pizotifen%20Tablets. Accessed January 16, 2013.
10. Blau JN. Migraine: theories of pathogenesis. *Lancet*. 1992;339: 1202–1207.
11. Lauritzen M. Pathophysiology of the migraine aura: the spreading depression theory. *Brain.* 1994;117:199–210.
12. Woods RP, Iacoboni M, Mazziotta C. Brief report: bilateral spreading cerebral hypoperfusion during spontaneous migraine headaches. *N Engl J Med.* 1994; 331:1689–1692.
13. Hadjikhani N, Sanchez M, Wu O. Mechanisms of migraine revealed by functional MRI in the human visual cortex. *Proc Natl Acad Sci USA*. 2001;98:4687–4692.
14. Goadsby PJ, Zagami AS, Lambert GA. Neural processing in cranio-vascular pain: a synthesis of the central structures involved in migraine. *Headache*. 1991;31: 365–371.
15. Peroutka SJ, Price SC, Wilhoit TL, Jones KW. Comorbid migraine with aura, anxiety and depression is associated with the dopamine D_2 receptor (DRD2) NcoI alleles, *MolMed.* 1998;4:14–21.
16. Evans RW. Reversible palinopsia and the Alice in Wonderland syndrome associated with topiramate use in migraineurs, *Headache.* 2006;46:815–818.
17. Jurgens TP, Ihle K, Stork JH. May A. "Alice in Wonderland syndrome" associated with topiramate for migraine prevention. *J Neurol Neurosurg Psychiatry*. 2011;82:228–229.