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Transient global amnesia occurring as migraine aura

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Abstract We describe a case of transient global amnesia (TGA) occurring as migraine aura. TGA prevalence is higher among migraineurs and has been ascribed to spreading depression in the hippocampus. Our patient, a 38-year-old physician, developed migraine without aura in early adolescence and, from age 20 years, had experienced rare attacks of migraine with aura. At ages 36 and 38 years, he suffered two attacks of sudden-onset anterograde amnesia, which lasted 5 hours and were immediately followed by

unilateral right-sided pounding headaches. Ictal neurological examination was normal except for fixation amnesia. Interictal brain magnetic resonance imaging (MRI) and neurological examination were normal. Thus, in our patient affected with migraine with and without aura, TGA occurred as the aura phase of two migraine attacks. Our case report suggests that TGA shares common mechanisms with migraine.

Key words Transient global amnesia • Migraine • Aura

Introduction

Transient global amnesia (TGA) is characterized by the abrupt onset of anterograde amnesia, accompanied by repetitive questioning and resolving within 24 hours [1]. Pure TGA is unassociated with other focal neurological abnormalities or epileptic activity. The pathophysiology of TGA remains unclear and several explanatory hypotheses have been put forward. TGA is associated with migraine, and the prevalence rate of migraine in TGA is 298 per 1000 [2]. Migrainous phenomena may accompany the TGA attack in selected cases [3, 4]. Similar changes in cerebral blood flow, namely hypoperfusion of the temporal lobe, have been reported for migraine and TGA [5, 6].

In 1986 Olesen and Jørgensen hypothesized that TGA could occur as a consequence of spreading depression of Leão in the hippocampus [7]. Spreading depression has also been hypothesized to underlie the migraine aura [8]. We report here a case of TGA occurring as the aura

phase of a migraine attack, thus confirming Olesen and Jørgensen's hypothesis.

Case report

A 38-year-old male general physician had a family history for amnesia episodes (in his father at a young age), but no history of migraine. Stammering was present in his son. No further details were available.

The patient had been stammering since childhood. Since adolescence, he had suffered unilateral right-sided or bilateral headache attacks, once or twice per month. The headaches lasted a few hours, sometimes were associated with vomiting, were often triggered by mental fatigue and lack of sleep, and were ameliorated by rest and acetaminophen. At age 20 years he had the first of 3 attacks of

migraine with aura; the last attack occurred at age 28 years. Each was characterized by visual aura with positive scotoma in both eyes (“like a shining sun”), and was soon followed by a unilateral headache for a few days. On three additional occasions, auditory hallucinations (like a whistle prolonged for several seconds) were followed by right-sided throbbing headaches, lasting a few hours.

The patient had no cardiovascular (heart disease, diabetes, hypertension, coagulation abnormalities) risk factors. At age 36 years, he had the first of 2 episodes of TGA. According to evidence given by a pharmacist and several friends, the patient, after leaving a drug store in the afternoon, suddenly became unable to recollect what to do or where to go. He behaved nervously. Although apparently unable to recognize the function, shape and coding colours of the car keys, he managed to drive home, where he could not recognize his son (who had just had a haircut) except when he was speaking. The patient did not remember things which had happened previously, nor the day’s appointments with patients. He was, however, well aware of his disorder. At 9.00 p.m. he was seen by a neurologist, who confirmed the absence of any other neurological deficit except fixation amnesia. After 4–5 h from onset, the amnesia slowly began to improve. However, a right-sided nuchal-frontotemporal pounding headache, quite similar to his usual migraine attacks, began; the headache lasted 3 hours and was resolved by acetaminophen. The day after, all symptoms had resolved. The patient had been in a severe stress situation for several weeks before the attack, sleeping less than 2–3 h per night. Brain magnetic resonance imaging (MRI) and electroencephalography (EEG) promptly performed were completely normal.

At age 38 years the second episode of TGA occurred, witnessed by his wife. During a prolonged stressful period with sleep deprivation (his second son had just been born), he woke up in the morning unable to remember what to do or where to go (he had previously woken in the night without any symptoms). He was unable to recollect things that had happened the day before, was anxious and continued to question his wife about his surroundings and objects in the house. There were no motor or language problems, and he was fully oriented as to himself and his family members. This lasted for 4–5 h and, while the amnesia was beginning to improve, a right-sided, throbbing, frontotemporal headache began, which responded to acetaminophen. He felt apathetic and depressed for a few days after the episode, but had no vigilance alterations and could return to his practitioner’s job.

The patient is currently experiencing 1–2 attacks per month of unilateral, often right-sided, mildly throbbing headaches, without any focal neurological symptoms.

Interictal neurological examination at 38 years of age was again normal.

Discussion

Our patient had migraine without aura, and additional migraine with aura according to the IHS classification [9]. Moreover, he suffered 2 episodes which conform to the definition of TGA by Hodges [1]: pure loss of fixation memory, no impairment of alertness, self-identity or other focal neurological involvement, resolving within 12 hours and associated with repetitive questioning and anxiety. Our patient’s attacks were witnessed by friends and a neurologist, and by his wife on the second occasion, as required by Hodges’ criteria. The patient had no cardiovascular risk factors, and brain MRI and EEG performed a few hours after the first episode gave completely normal results. Both attacks were followed by throbbing, unilateral, mainly frontotemporal headaches with clear migrainous characteristics. Therefore, we conclude that TGA occurred in our patient twice as the aura of a migraine attack, and was probably triggered by a period of stress and sleep deprivation.

Our case report, although isolated, confirms the links between pure TGA and migraine which were suggested by several epidemiological surveys and case reports [2–4]. It also lends credit to Olesen and Jørgensen’s hypothesis [7] that TGA may result from spreading depression (SD) of Leão in the hippocampus. SD of Leão has also been hypothesized to underlie the aura phase of migraine [8]. Although the relation between TGA and migraine is still debated, SD could be hypothesized to represent the common pathogenic mechanism underlying these two clinical situations. In this regard, it is relevant that recent diffusion-weighted MRI studies demonstrated decreased interstitial space and cell oedema in the temporal lobe during TGA, compatible with SD [10]. Our patient did not complain of classic aura symptoms in conjunction with the TGA attacks, and the pathogenic relations between migraine and TGA remain, therefore, undefined.

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