Transient Global Amnesia in a Patient with Postherpetic Neuralgia

J.TWMU 2: 37-39, 2018

Case Report

Transient Global Amnesia in a Patient with Postherpetic Neuralgia

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(Accepted February 13, 2018)
(Advance Publication by J-STAGE May 11, 2018)

Transient global amnesia (TGA) is a clinical syndrome of reversible anterograde amnesia that occurs in the middle-aged and elderly individuals. The etiology is obscure, and many underlying factors have been reported. We describe an 83-year-old man with postherpetic neuralgia (PHN) for which he received pregabalin, fentanyl, and a serotonin-norepinephrine reuptake inhibitor (SNRI). Pain intensity on a numeral rating scale (NRS) was 8/10. He had no history of vascular disease or epilepsy. On November 28th 20XX he visited our hospital and underwent intercostal nerve block with phototherapy as usual. He was slightly feverish and had a body temperature of 37.8°C with no other symptoms. After 15 minutes rest, he left our hospital and tried to return to his home, but he was unsure which train to take. About 4 hours later, he suddenly knew where he was and got back to his home uneventfully. He had been amnetic for several hours. Two weeks later, he was referred to a neurologist, and the episode was diagnosed as TGA. Brain magnetic resonance imaging (MRI) showed non-specific cerebral atrophy with signs of aging at that point. Differential diagnoses include ischemia, migraine, epileptic seizures, and thromboembolic cerebrovascular disease. The cause of TGA remains controversial. However, drugs such as GABAergic drugs, SNRIs, and opioids are known to have effects on cognitive function. Polypharmacy can result in substantial drug-drug interactions, especially in elderly patients. The trigger of this episode in our patient is unclear, but multiple analgesics might have been a causal factor of his symptoms of TGA.

Key Words: global amnesia, postherpetic neuralgia, polypharmacy

Introduction

Transient global amnesia (TGA) is a clinical syndrome that was proposed by Hodges and Warlow in 1990[1)].TGA is characterized by the sudden onset of anterograde amnesia lasting up to 24 hours without compromise of other neurologic functions in middle-aged and elderly individuals. We report a case of TGA associated with postherpetic neuralgia (PHN).

Case Presentation

An 83-year-old man was given a diagnosis of herpes zoster in the Th8-9 region in June 20XX. He was referred to our hospital to receive pain management in August 20XX. He complained of continuous and shooting pain (intensity 7 of 10 on a numerical rating scale[NRS]) in his chest and back, associated with allodynia. He had no history of vascular or cerebral diseases, epilepsy, diabetes

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doi: 10.24488/twmuj.2017004

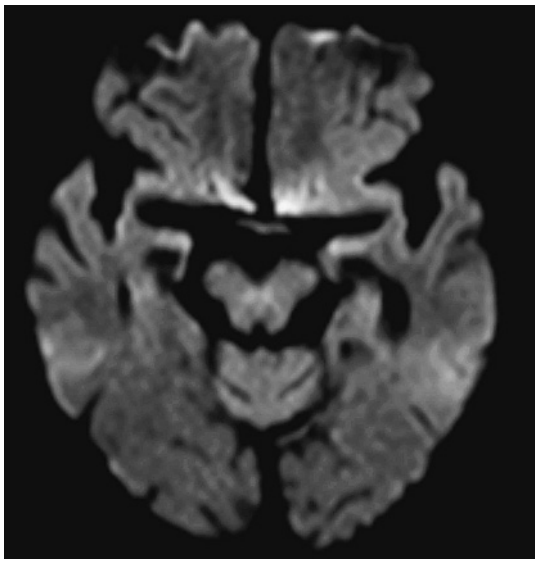

Fig. 1 Magnetic resonance imaging (MRI) of the patient.
Axial diffusion-weighted imaging (DWI) taken 2 weeks after episode revealed only slight ischemic change.

mellitus, myocardial infarction,mental diseases, or recent head injury. He also had no histry of temporary memory loss. No other medication was administered by another medical department. After thoracic epidural brocks and intercostal nerve brocks were performed 10 times, the pain score decreased to 3 of 10 on the NRS. We gave the patient pregabalin 300 mg/day, celecoxib 200 mg/day, and vitamine B12 1,500 μg/day.

In November, we changed the prescription to amitriptyline 20 mg/day and pregabaline 300 mg/day because he claimed to have compressive pain. In October 20XX + 1 he had increasing pain (NRS7/10) associated with the decreasing atmospheric temperature in the winter season. We replaced amitriptyline with duloxetine 20 mg/day, and decreased the dose of pregabaline to 225 mg/day because he was staggering. On October 20XX + 2, the patient claimed to have increasing pain (NRS7/10) in the winter season again, and we therefore prescribed an opioid analgesic (fentanyl patch 1 mg/day), pregabaline 150 mg/day, and duloxetine 20 mg/day. No side effect developed. In July 20XX + 3, fentanyl patch 2 mg/day, pregabaline 125 mg/day, and duloxetine10 mg/day decreased the pain intensity to NRS2/10, but he had mild sleepiness. In October the pain increased to NRS8/10 again. On November 28th he visited our hospital and received intercostal nerve block with 0.5 % bupivacaine 5 ml with phototherapy as usual. Before the treatment he was slightly feverish (37.8°C) with no other symptoms. There was no abnormal behavior or conversation before or during our examination.

On the way back to his home, the patient suddenly forgot how to get home and which train to take. About 4 hours later, he suddenly knew where he was, and he went back to his home in the evening without any injury, palsy or visual or speech disturbances. He had had amnesia for several hours. After returning to his home and on the following days, his physical and mental status was the same as usual. However, he did not remember what had happened to him. There was no witness to his behavior during the amnestic period.

After 2 weeks he consulted a neurologist, and the episode was diagnosed as TGA. The mini-mental state examination (MMSE) score was 21/30, indicating slight cognitive impairment. He had never undergone the MMSE before the event. His families mentioned his slight forgetfulness for several months. However, no other abnormal behavior was reported. Brain magnetic resonance imaging (MRI) showed non-specific slight cerebral atrophy associated with signs of aging (**Fig. 1**) Treatment with duloxetine and pregabaline was discontinued, and there was no deterioration of amnesia for at least 8 months after the episode.

Discussion

The diagnosis of TGA is clinical and one of exclusion. Differential diagnoses include several other transient amnesic disorders with varying epidemiology and disease courses[2]. Transient epileptic amnesia (TEA) is a rare form of temporal epilepsy, often associated with motor automatisms of brief duration, occurrence on awakening, and multiple attacks. Prominent and short durations of retrograde amnesia occur in patients with TEA. Our patient did not have a past history of epilepsy, and anterograde amnesia began during the daytime, persisted for about 4 hours, and presented as a single event of disorientation to place. Transient ischemic attack (TIA) usually lasts for less than 1 hour with some other symptoms, such as palsy, speech disturbances, or perception disturbances. Patients with psychogenic amnesia have isolated retrograde amnesia. Brain MRI with diffusion-weighted imaging (DWI) can demonstrate even minute and tran-

sient hippocampal lesions shortly after events in patient with TGA. In our patient, DWI was not performed during and shortly after the episode, but TGA was diagnosed on the basis of the characteristic episodes. He was slightly feverish on morning of the presentation, but his physical condition did not exacerbate subsequently. Many events have been reported to potentially trigger hippocampal ischemia, including emotional or physical changes, the Valsalva maneuver, postural changes, medical procedures, and changes in body temperature[2]. Fever on the day of onset might have triggered our patient's illness. Pregabarin and duloxetine are popular drugs for the management of PHN. Opioid analgesics are also recommended for intractable PHN[3]. Because our patient had recurrent exacerbations of pain in association with atmospheric temperature changes in the winter season, we needed to increase the doses of analgesics, which led to polypharmacy. Drug-drug interactions induced by polypharmacy are known to cause cognitive disfunction[2].

Some analgesic or hypnotic drugs can also affect cognitive function. Anticholinergic drugs such as tricyclic antidepressants or benzodiazepines may cause cognitive disfunction, but serotonin-noradrenaline reuptake inhibitors are not considered to worsen it.

Opioids and pregabalin have effects on the GABAergic receptor system, thereby affecting cognitive function[4]. Pickering et al. reported cognitive impairment and pain associated with antidepressants in patients with PHN. Chronic pain itself might also impair cognitive function[5].

We consider drug-drug interactions the most likely cause of TGA in our patient.

Conclusion

We must exercise care and carefully administer drugs that affect cognitive function when treating elderly patients with chronic pain.

Consent for publication was obtained from the patient.

Conflicts of Interest: None of the authors of this manuscript have a conflict of interest to declare.

References

1) Hodges JR, Warlow CP: Syndromes of transient amnesia: towards a classification. A study of 153 cases. J Neurol Neurosurg Phschiatry 53: 834−843, 1990
2) Spiegel DR, Mccroskey AL, Deyerle BA: A case of transient global amnesia. Innov Clin Neurosci 13 (3-4): 32−41, 2016
3) The Committee for the Guidelines for the Pharmacologic Management of Neuropathic Pain of JSPC: Guidelines for the Pharmacologic Management of Neuropathic Pain. Shinko Trading, Tokyo (2011)
4) Mizukami K: Drug-induced cognitive inpaiment. Psychiat Neurol Jap 111: 947−953, 2009 (in Japanese)
5) Pickering G, Pereira B, Clère F et al: Cognitive function in older patients with postherpetic neuralgia. Pain Prac 14: E1−E7, 2014