Acute Amnestic Disorder After Gym-Related Physical Exertion

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Abstract

Transient global amnesia (TGA) is a clinical syndrome characterized by the acute onset of isolated anterograde amnesia without other cognitive deficits or focal neurological signs. It is commonly encountered in clinical practice, with patients frequently presenting to outpatient clinics or emergency departments, necessitating prompt evaluation to determine the cause of acute memory loss. Several evidence from structural brain imaging studies has demonstrated focal lesions within the temporal lobe, particularly involving the hippocampus. Lesions affecting the CA1 neuronal region-an essential component of memory circuits-are strongly associated with memory impairment in patients with TGA when compared with healthy controls. In this article, we report an interesting case of first episode acute memory loss after physical exercise, highlighting characteristic MRI findings.

Keywords: Transient global amnesia, CA1, Memory impairment

Introduction:

Transient global amnesia (TGA) is a distinct clinical entity characterized by the sudden onset of episodic memory impairment, predominantly presenting as anterograde amnesia that typically resolves within 24 hours[1-3]. During the acute attack, patients exhibit isolated anterograde memory loss lasting from minutes to several hours, while other cognitive domains-including language, praxis, calculation, and executive functions-remain preserved[4]. There is no alteration in the level of consciousness or disturbance of personal identity. Clinically, patients frequently present with repetitive

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questioning, such as repeatedly asking about their location or the reason for their current circumstances and may appear confused about the ongoing situation. A variety of precipitating factors have been reported, suggesting that TGA may be triggered by multiple physiological or environmental stressors.

Neurological examination and psychological testing primarily reveal impairment of anterograde memory, with relative preservation of retrograde memory and no focal neurological deficits. Mild accompanying somatic symptoms, including headache, dizziness, or nausea, may also be observed[3,4]. Structural brain magnetic resonance imaging (MRI) studies have demonstrated focal lesions affecting the hippocampal CA1 neuronal region, with characteristic signal abnormalities in the mesiotemporal area, implicating disruption of memory-related neural

circuit[5,6]. Despite these neuroimaging findings, the pathogenesis of TGA remains incompletely understood, and several mechanisms-including migraine-related phenomena, focal ischemia, venous flow abnormalities, and epileptic activity-have been proposed[1,4,7].

In this report, we present an illustrative case of a patient experiencing a first episode of acute-onset memory loss following intense physical exertion. In addition, we provide a concise review of the neuroscientific knowledge of memory systems and the mechanisms underlying TGA, including clinical presentation, diagnostic criteria, proposed pathophysiology, diagnostic evaluation, differential diagnosis, and current approaches to management and prognosis, derived from a literature review conducted by preclinical medical students and discussed collaboratively with academic staff.

Case presentation:

A 51-year-old Thai man, a university lecturer with no significant past medical history, presented to the neurology outpatient clinic with an episode of acute memory loss that had occurred one day prior to presentation. One day before admission, he engaged in strenuous physical activity at a fitness center, consisting of resistance training and cardiovascular exercise for nearly two hours, followed by a warm shower. While walking toward his locker to change clothes, he suddenly developed acute confusion and was unable to recognize his surroundings or recall his intended actions. Closed-circuit television footage reviewed by fitness center staff showed the patient standing in a confused state and pacing near the lockers. When approached, he was able to state his name but repeatedly asked the same questions, such as *"why I was there"*. He also complained of a mild headache, after which staff assisted him to rest.

Approximately one hour later, he awoke spontaneously without any focal neurological symptoms. He subsequently drove home and reported the episode to his wife. The following day, he presented to the hospital for further evaluation. He was unable to clearly recall the entire amnestic period but estimated that the episode-from entering the shower to returning home-lasted approximately three hours. All symptoms had completely resolved after returning home. He reported that this was the first such episode in his life and sought medical attention due to concern. He denied any chronic medical conditions, regular medication use, smoking, or illicit drug use. He reported occasional alcohol consumption and no history of severe traumatic brain injury. There was no family history

of cerebrovascular or cardiovascular disease.

On physical examination, vital signs were stable (BP 125/80 mmHg, T 37°C, HR 75 beats/min with regular rhythm, RR 18 breaths/min), and no carotid bruit was detected. Neurological examination revealed an alert and cooperative patient with intact cranial nerve function. Motor power was grading V/V in all extremities, sensory examination was normal, deep tendon reflexes were 2+ symmetrically, and no long tract signs were observed.

Neuropsychological testing revealed a Thai Mental State Examination (TMSE) score of 30/30 and a Montreal Cognitive Assessment (MoCA) score of 24/30, with deficits confined to the delayed recall domain, while attention, executive function, language, visuospatial ability, and orientation were preserved. Laboratory investigations, including complete blood count, serum glucose, electrolytes, thyroid function, liver function, and lipid profile, were within normal limits. A 12-lead electrocardiogram and chest radiograph showed no abnormalities. Magnetic resonance angiography and bilateral carotid Doppler ultrasonography were unremarkable.

Brain MRI demonstrated a single punctate focus of restricted diffusion weighted imaging (DWI) in the left hippocampal region with a corresponding low signal on the apparent diffusion coefficient (ADC) map, consistent with an acute infarction (Fig.1). No corresponding signal abnormalities were observed on T1-weighted or gradient-echo sequences to suggest acute or chronic hemorrhage. A minimal increase in signal intensity on fluid-attenuated inversion recovery (FLAIR) imaging was noted, compatible with mild underlying edema but could not find an increased rate of microangiopathic white matter hyperintensities.

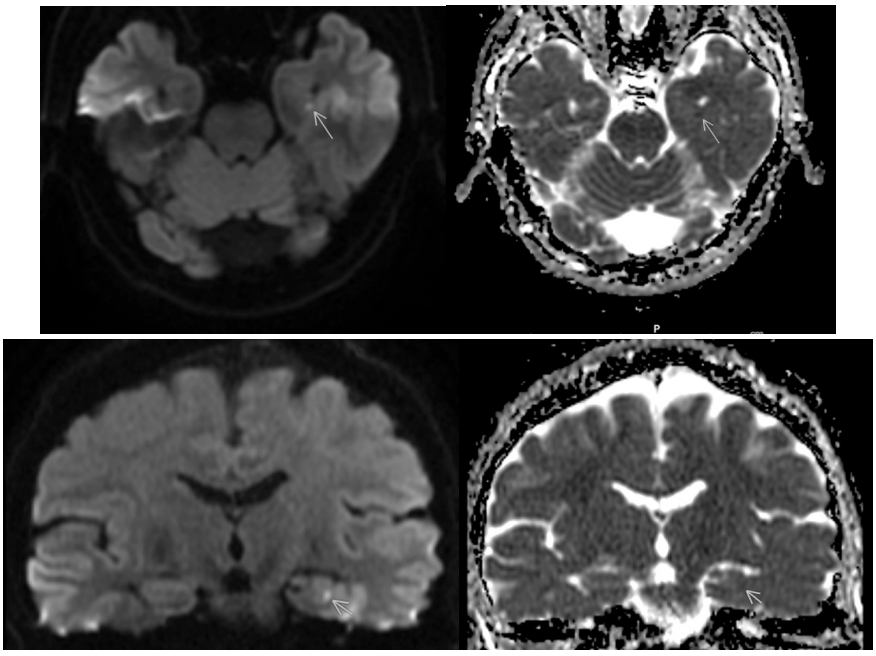

Figure 1: Representative 1.5T MRI of a patient shown a single punctuate lesion on diffusion weighted imaging with a corresponding low signal on the apparent diffusion coefficient map (green arrow) in the axial and coronal planes. (Front cover page)

At the 3-month follow-up visit, he reported no recurrence of symptoms. His general condition and mood were normal, and he had returned to regular exercise with reduced intensity. Follow-up physical and neuropsychological examinations revealed no residual abnormalities.

Discussion:

The patient presented with an acute amnestic disorder characterized by sudden-onset memory impairment in the absence of deficits in other cognitive domains. This clinical presentation necessitates consideration of several important differential diagnoses[8]. (i) Delirium is a common cause of acute confusion; however, it is typically associated with fluctuating levels of consciousness, inattention, and an identifiable precipitating factor, none of which were observed in this patient. (ii) Transient epileptic amnesia should also be considered, but it usually manifests as recurrent episodes, often involves more than one cognitive domain, and is commonly associated with epileptiform abnormalities on electroencephalography. (iii) Ischemia involving the posterior cerebral circulation may present with acute memory disturbance; however, it is frequently accompanied by additional focal neurological deficits or visual symptoms. (iv) Metabolic disturbances, such as hypoglycemia, can also cause acute amnesia but were excluded in this case based on normal laboratory findings.

Neurological examination and neuropsychological testing confirmed isolated impairment of anterograde memory, with preservation of semantic and procedural memory. No focal neurological deficits were identified, and laboratory investigations revealed no metabolic abnormalities. These findings strongly support a diagnosis of TGA[8]. Furthermore, structural brain MRI demonstrated a focal lesion in the mesial temporal lobe, specifically involving the hippocampus, a key component of the limbic system and memory circuitry. The presence of a punctate lesion within this region is consistent with previously reported neuroimaging findings in patients with TGA and further supports the diagnosis. The MRI findings suggest that a transient perturbation of hippocampal function is the correlation of TGA, as focal lesion can be reliably detected in the CA1 field by use of DWI.

The present case demonstrates classic clinical features of TGA and highlights intense physical exertion as a potential precipitating factor. Strenuous exercise has been frequently reported as a trigger for TGA. Activities involving intense physical effort may provoke Valsalva-like maneuvers, leading to transient alterations in intrathoracic and intracranial pressures. This physiological response can impair cerebral venous outflow, particularly in the hippocampal region, which is highly vulnerable to metabolic stress. Venous congestion and transient ischemia within the hippocampal CA1 neuronal region have therefore been proposed as key mechanisms underlying exercise-induced TGA. In addition, prolonged or intense physical activity may result in transient hypoperfusion or metabolic imbalance within selectively vulnerable neuronal populations[10]. The CA1 neurons of the hippocampus are known to be particularly susceptible to ischemic and metabolic stress, which may explain the characteristic punctate diffusion-restricted lesions observed on diffusion-weighted MRI in patients with TGA. The delayed appearance of these lesions further supports a transient, rather than permanent, ischemic process.

The absence of focal neurological deficits preserved level of consciousness, and isolated impairment of episodic memory in this patient argue against alternative diagnoses such as posterior circulation stroke or seizure-related amnesia. Furthermore, the lack of recurrent episodes and the spontaneous resolution of symptoms are inconsistent with transient epileptic amnesia. The temporal association between intense exercise, subsequent warm showering, and the onset of symptoms suggests that combined physiological stressors may have contributed to the development of TGA. Similar precipitating events-including physical exertion, emotional stress, sudden temperature changes, and sexual activity-have been well documented in the literature.

Overall, this case supports the hypothesis that exercise-induced, Valsalva-related mechanisms play an important role in the pathophysiology of TGA (Fig.2), particularly through transient dysfunction of hippocampal memory circuits[12]. Recognition of these triggers is essential for accurate diagnosis, patient reassurance, and avoidance of unnecessary diagnostic or therapeutic interventions.

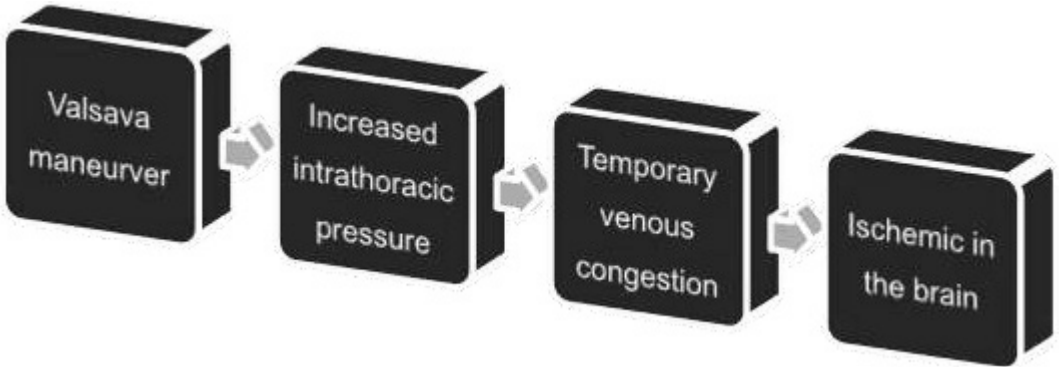

Figure 2: Pathophysiology of TGA due to Valsalva-related mechanisms

Review of TGA:

The incidence of TGA varies across studies, with reported rates ranging from approximately 3 to 10 cases per 100,000 population per year[2-3]. TGA most commonly affects individuals between 50 and 70 years of age and shows no significant sex predilection[2]. Despite the dramatic clinical presentation, the condition is associated with a favorable prognosis. Morbidity and mortality rates are low, with an annual recurrence rate of less than 5%, and there have been no reports of long-term cognitive morbidity or increased mortality attributable to TGA[3].

The diagnosis of TGA is primarily clinical and is based on established criteria proposed by Caplan and Hodges[8]. These include the presence of anterograde amnesia witnessed by an observer, absence of clouding of consciousness or loss of personal identity, cognitive impairment limited to amnesia, absence of focal neurological or epileptic signs, no recent history of head trauma or seizures, and complete resolution of symptoms within 24 hours[2,4,9]. A wide range of precipitating factors have been reported in association with TGA, including emotional stress, strenuous physical exercise, sudden temperature changes, severe acute pain,

and sexual intercourse. In addition, Valsalva-associated maneuvers resulting in abrupt increases in intrathoracic pressure have been implicated. These triggers are thought to induce transient alterations in cerebral venous outflow or hippocampal perfusion, thereby contributing to the development of transient amnesia[10,11].

Early hippocampal dysfunction, particularly involving the CA1 neuronal region, leads to selective impairment of memory processes within the Papez circuit of short-term memory. Clinically, this manifests as loss of environmental familiarity, impaired spatial orientation, and deficits in episodic memory[9,12]. In contrast, semantic memory, procedural learning, and language-related functions-including reading, writing, and speech—are typically preserved. Long-term outcome in patient with TGA is generally considered a benign condition[12]. The patients who have experienced TGA regarding the risk of recurrence and the incidence of cognitive decline, stroke, and seizures over time.

Medical student's Learning points:

- **Recognize** the characteristic clinical presentation of TGA, including acute-onset isolated anterograde amnesia with preserved consciousness, personal identity, and other cognitive functions.
- **Differentiate** TGA from other causes of acute amnestic syndromes, such as delirium, transient epileptic amnesia, posterior circulation ischemia, and metabolic disturbances.
- **Identify** common precipitating factors of TGA, including strenuous physical exertion, Valsalva-like maneuvers, emotional stress, and sudden temperature changes.
- **Interpret** neuropsychological and neuroimaging findings in TGA, particularly isolated impairment in delayed recall and characteristic hippocampal CA1 lesions on diffusion-weighted MRI.
- **Understand** the generally benign, self-limited nature and favorable prognosis of TGA to guide appropriate management, patient reassurance, and avoidance of unnecessary investigations.

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