

## Clinical Communications: Adults

### “FORGETTABLE” SEX: A CASE OF TRANSIENT GLOBAL AMNESIA PRESENTING TO THE EMERGENCY DEPARTMENT

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**Abstract—Background:** Transient global amnesia is characterized by the sudden development of dense anterograde amnesia, without alteration in level of consciousness and in the absence of focal neurologic deficits or seizure activity. Various precipitating causes have been reported in the medical literature. **Objective:** To present a literature-guided approach to the diagnosis and management of transient global amnesia in the Emergency Department (ED). **Case Report:** We report the case of a 54-year-old woman who presented to the ED with an episode of acute memory loss. **Conclusions:** Although rare, transient global amnesia may present in a dramatic fashion. The occurrence of a distinct precipitating event and repetitive questioning seem to be key features in making the diagnosis. Important differential considerations include transient ischemic attack, seizure, and subarachnoid hemorrhage. Brain imaging and specialty consultation are reserved primarily for patients with unclear circumstances, altered level of consciousness, focal neurologic findings, and persistent (or very brief) amnesic symptoms. Brain imaging may, however, relieve anxiety about more dangerous causes of the event. © 2011 Elsevier Inc.

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#### INTRODUCTION

Transient global amnesia is characterized by the sudden development of dense anterograde amnesia, without alteration in level of consciousness and in the absence of

focal neurologic deficits or seizure activity (1). Various precipitating causes have been reported in the medical literature. We report the case of a 54-year-old woman who presented to the Emergency Department (ED) with an episode of transient global amnesia precipitated by sexual climax.

#### CASE REPORT

A 54-year-old post-menopausal woman presented to the ED complaining of “memory loss” after sexual intercourse. The patient was in her usual state of health until approximately 1 h before ED arrival, when she experienced the acute onset of short-term memory loss coinciding with sexual climax. Both the patient and her husband stated that after climax she was unable to recall events of the preceding 24 h. In addition, she had difficulty incorporating new memories (forgetfulness). Her husband stated that she remained cognizant of herself and was able to recognize him throughout the episode. Profound amnesic symptoms lasted approximately 20 min, with a gradual resolution over the following 40 min. At the time of ED presentation, her symptoms were near-totally resolved. There was no alteration in level of consciousness reported at any time during the episode. There was no suggestion of other loss of specific neurologic function. She denied headache. She denied any form of recent head trauma. The past medical history was

unremarkable, including past history of transient ischemic attack (TIA), migraine headache, other neurologic conditions, psychiatric conditions, or known seizure disorder (personal or family history). Importantly, she denied prior episodes of similar symptoms. The patient admitted to occasional alcohol use, but denied ingestion on the day of presentation. She also denied over-the-counter, prescription, or illicit drug use (including sedative use).

The patient was alert, oriented, and conversant, with vital signs as follows: blood pressure 132/76 mm Hg, pulse rate 67 beats/min, respirations 20 breaths/min, and temperature 37°C. The oxygen saturation was 100% on room air. Initial serum glucose was 107 mg/dL. Comprehensive physical and neurologic examinations were entirely normal, including mental status, cranial nerve, motor, sensory, and cerebellar testing. She did not exhibit deficits in long-term memory, judgment, attention, language, or emotional lability.

Initial laboratory testing was normal (complete blood count, serum chemistry panel, coagulation profile). An electrocardiogram was normal. A non-contrast head computed tomography (CT) scan was normal.

The patient's symptoms resolved completely shortly after ED arrival. Emergent Neurology consultation was requested for evaluation of possible TIA. After a comprehensive evaluation, a presumptive diagnosis of transient global amnesia (TGA) was suggested by the Neurology service. The Neurology service made arrangements for outpatient magnetic resonance imaging (MRI), including MR angiography, and recommended empiric treatment with aspirin (81 mg daily) pending imaging results. The patient was discharged home from the ED with her husband after consultation. A follow-up MRI study of the head obtained as an outpatient was normal.

## DISCUSSION

Transient global amnesia is characterized by the sudden development of dense anterograde amnesia, without alteration in level of consciousness and in the absence of focal neurologic deficits or seizure activity (1). It is a benign, self-limited condition. Numerous precipitating causes have been identified, including strenuous physical activity, severe pain, and physiological or psychological stress (2–4). It has an overall annual incidence of 3.4–5.2 per 100,000 individuals, with increased frequency in those over 50 years of age (reported incidence of 23.5 per 100,000) (5). Theories regarding the pathophysiology of TGA have focused on migraine, seizure discharge, arterial ischemia, and venous congestion of the brain leading to ischemia

(resulting from a Valsalva response), although the precise mechanisms remain unclear (6). The hallmark of TGA is anterograde amnesia, although TGA may also present with retrograde amnesia, with most recently acquired memories at greatest risk (5). Long-term (distant) memories, the meaning of words and objects, as well as an awareness of what one *should* know, are typically preserved (5). Repetitive questioning is frequently reported during an episode of TGA, likely secondary to the inability to learn new information coupled with retrograde memory loss (7). In a case series of TGA, the majority (90%) of patients experienced repetitive questioning (4). Patients suffering from TGA typically return to baseline within a few hours, except for a dense, residual amnesic gap for events occurring during the attack (8).

Helpful in making the diagnosis of TGA is the common occurrence of a precipitating event. Up to 90% of TGA episodes have an identifiable physical or psychological precipitating factor (3). Sexual intercourse (including orgasm) has been reported as a precipitant of TGA (9–11). In sex-induced TGA, the precipitating activity may not be remembered, including only hazy recollections of foreplay in some cases (12). Fisher and Adams in 1964 reported a case of a man whose episode began during orgasm when he suddenly exclaimed “Where am I? What’s happened?” (13). Recurrent episodes of TGA induced by sexual activity have also been reported (12). However, the overall recurrence rate is very low and in most patients, TGA strikes only once (3).

Hodges and Warlow in 1990 devised clinical criteria for the diagnosis of isolated TGA (Figure 1) (1). The utility of these diagnostic criteria to the acute care provider has been questioned. For example, requiring resolution within 24 h to fulfill the diagnostic criteria of TGA is impractical in the ED setting (5). Indeed, these diagnostic criteria are more helpful in hindsight, after complete resolution of the episode. Furthermore, several emergent conditions requiring prompt action may be confused with TGA (as discussed below), hampering the utility of the “wait and see” approach (14).

The differential of acute total amnesia includes TGA, TIA, subarachnoid hemorrhage (SAH), complex partial seizures, transient epileptic amnesia, psychogenic amnesia, and drug-related confusional states, among others (5,14–17). SAH is a particularly important consideration, as headache is frequently reported in TGA patients (up to 40% in one series) (3). In one small case series of sex-induced acute amnesia presenting to an ED, SAH was identified in one of 10 cases, with TGA comprising the remaining cases. However, in contrast to TGA, the patient with SAH demonstrated clear focal (cerebellar) findings on neurological examination (14). It is important to note that the headache associated with SAH may be

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