

Postgraduate Medicine

ISSN: 0032-5481 (Print) 1941-9260 (Online) Journal homepage: <http://www.tandfonline.com/loi/ipgm20>

Transient global amnesia

Gilbert J. Toffol DO & Michael Swiontoniowski MD

To cite this article: Gilbert J. Toffol DO & Michael Swiontoniowski MD (1990) Transient global amnesia, Postgraduate Medicine, 88:2, 217-219, DOI: [10.1080/00325481.1990.11704711](https://doi.org/10.1080/00325481.1990.11704711)

To link to this article: <http://dx.doi.org/10.1080/00325481.1990.11704711>



Published online: 17 May 2016.



Submit your article to this journal



View related articles

Full Terms & Conditions of access and use can be found at
<http://www.tandfonline.com/action/journalInformation?journalCode=ipgm20>

Transient global amnesia

Gilbert J. Toffol, DO Michael Swiontoniowski, MD

Preview

Although transient global amnesia tends to occur only once in a patient and is usually benign, such an episode can be alarming. The authors describe a typical case and discuss the characteristics, possible causes, prognosis, and treatment of transient global amnesia.

A 63-year-old woman was admitted to Mesa General Hospital, Mesa, Arizona, because of transient confusion that had been noted by her husband. She had suddenly become disoriented after having an orgasm during sexual intercourse. She had a mild, throbbing headache that had begun at onset of her confusion, and she continually repeated questions as to her whereabouts and the time. An emergency department nurse noted that "the patient doesn't remember what you talked about a few minutes earlier."

On physical examination, the patient was oriented only to her name. Recent and remote memory for the past several months was absent. She was unable to retain new information for more than 30 seconds. The patient was normotensive, and the remainder of the physical examination was unremarkable. Results of noninvasive carotid studies, electrocardiogram, echocardiogram, electroencephalogram, magnetic resonance imaging, and thyroid function studies were normal. Within 4 hours the patient's memory deficits resolved, except for permanent amnesia of the events that occurred between onset of the transient global amnesia and recovery.

Discussion

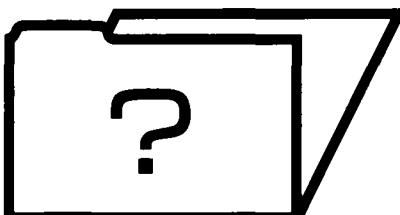
Amnesic syndromes (table 1) are disorders of cognitive function in which memory is impaired out of proportion to other components of mentation and behavior.¹ Transient global amnesia is characterized by sudden onset of retrograde amnesia (impaired ability to recall previously well-established information), an-

sudden onset of profound amnesia without loss of consciousness. Retrograde amnesia can extend for several hours to years but recedes spontaneously as the attack subsides. The patient is unable to recall events that occur during the attack, which usually lasts 4 to 8 hours but can last up to 24 hours before slowly resolving. The exact cause and incidence of transient global amnesia in the general population are uncertain.

A unique characteristic of transient global amnesia is the repetition of questions during an attack. In one study,⁴ 64 of 71 patients repeated a series of questions or statements. Although this may indicate confusion, neuropsychological testing during an attack has shown only impairment of memory with no other cognitive dysfunctions.⁵ Patients are able to perform complex motor skills, such as dressing, preparing food, typing, or driving. They indicate an awareness of their predicament by repeating questions such as "Where am I?" or "Why am I here?" and stating that something is wrong.

Transient global amnesia occurs mainly in the middle-aged and elderly. In Rollinson's review of 213 cases from the literature,⁶ the age range was 34 to 92 years, with a mean age of 55 years. Attacks tended to be singular, with only 34 (16%) of the 213 patients having one or more recurrences. There was a 4:3 male-female ratio. Precipitating factors included coitus, bathing in cold water, pain, migraine, and emotional experiences. One report of transient

continued



terograde amnesia (inability to form new memories), and repetitive queries. No other neurologic signs and symptoms are present, and personal identity is preserved.

The term "transient global amnesia" was first used by Fisher and Adams² in 1958. Two years earlier, the condition had been described by Bender³ as a "syndrome of isolated episode of confusion with amnesia." A typical attack is characterized by

Table 1. Disorders characterized by an amnesia syndrome

Transient global amnesia
Thiamine deficiency
Tumors
Trauma
Subarachnoid bleeding
Central nervous system infections
Paraneoplastic syndromes (eg, limbic encephalitis)
Cerebral infarction
Migraine
Seizure (postictal)

global amnesia in four brothers⁷ suggests a familial occurrence.

Croft and associates⁸ found that persons with transient global amnesia formed the largest group (62%) of patients referred to neurologic clinics for evaluation of transient amnesia. Differential diagnostic considerations include the following:

- Epilepsy. Epileptic seizures usually are brief, and automatism and depersonalization may be present.
- Psychogenic disturbance. Patients with a psychogenic disturbance often have a history of stress, anxiety, depression, or histrionic personality

**Gilbert J. Toffol, DO
Michael Swiontoniowski, MD**

Dr Toffol is clinical assistant professor of neurology, College of Osteopathic Medicine of the Pacific, Pomona, California. He is also in the private practice of neurology in Phoenix.

Dr Swiontoniowski is clinical instructor in stroke and attending physician, department of neurology, Michael Reese Hospital and Medical Center, Chicago.

disorder. A psychogenic attack may end abruptly, in contrast to the gradual shortening of retrograde amnesia in transient global amnesia.

- Encephalitis. A prodromal illness, fever, and abnormal cerebrospinal fluid and electroencephalographic findings may be present.
- Trauma. Evidence of head injury may be found on physical examination or on skull films of patients with posttraumatic amnesia.
- Tumors. Cerebral tumors usually manifest with other neurologic findings. Symptoms are progressive, and computed tomographic or magnetic resonance imaging scans are abnormal.
- Migraine. An aura or headache may accompany migraine.
- Alcohol abuse. An alcoholic patient may present with confabulation and loss of insight in addition to the stigmata of alcohol abuse (eg, malnutrition, neuropathy, ophthalmoplegia).
- Hypoglycemia. Hypoglycemic episodes are shorter than attacks of transient global amnesia, and laboratory results are abnormal.

Transient amnesia has also been linked to allergic encephalopathy due to antitetanus serum, cerebral and coronary angiography, pituitary tumor, polycythemia, myxomatous degeneration of the mitral valve, neurosyphilis, and intoxication with digitalis, iodochlorhydroxyquin (clioquinol) (Vioform), or diazepam (Valium, Valrelease).⁶⁻¹⁰

The exact cause of transient glob-

al amnesia is uncertain. An attack is usually solitary, unaccompanied by other signs and symptoms, and followed by complete recovery, all of which make it difficult to explain. Fisher and Adams^{4,11} argue that seizure is the cause, but this theory is contradicted by the age at onset, low recurrence rate, absence of clearly recognizable attacks of epilepsy, and infrequency of electroencephalographic abnormalities.

Another possible explanation is transient ischemia in the posterior cerebral artery. Arguments favoring this theory include the facts that amnesia is seen with occlusion of the posterior cerebral artery,¹² other signs and symptoms of vertebrobasilar insufficiency may be present, and risk factors for cerebrovascular disease are present in some patients with transient global amnesia.¹³ Jensen and de Fine Olivarius¹⁴ argue that there is a clinical spectrum of ischemia: At one end is transient global amnesia with a single episode and no other findings; in the center, transient amnesia with other transient symptoms of insufficient posterior cerebral circulation; and at the other end, more widespread ischemia and infarction.

Caplan and associates¹⁵ agree that a problem in the posterior cerebral circulation explains transient global amnesia, but they argue in favor of migraine because it is a vascular disorder with bias for the posterior cerebral circulation. In addition, electroencephalographic abnormalities have been described in transient

global amnesia,¹⁵⁻¹⁷ and these are also consistent with migraine.¹⁸

Follow-up studies reveal that transient global amnesia is usually benign. In one series of 33 patients,¹⁹ 6 (18%) had recurring episodes and 2 (6%) progressed to stroke. In another report,²⁰ 4 (12.5%) of 32 patients had recurring episodes and 1 (3%) had transient ischemic attacks. Other investigators have found that the outlook is not so benign. Of the 28 patients described by Jensen and de Fine Olivarius,¹⁴ 9 (32%) had strokes and 4 (14%) had recurring transient ischemic attacks. Transient global amnesia by itself still has a good prognosis, but the presence of other transient ischemic manifestations or risk factors for cerebrovascular disease increases the chance of further problems. Nevertheless, follow-up neuropsychological testing may reveal a persistence of deficit in verbal IQ and impairment of verbal memory even in patients who have had a single episode of transient global amnesia and have returned to work and family life with no complaint of memory loss or other mental difficulties.²¹

Providing reassurance to both patient and family and avoiding invasive investigation are the mainstays of treatment of transient global amnesia.⁹ Patients with migraine and risk factors for cerebrovascular disease need to be followed closely. Crowell and associates²² believe that prophylaxis with antiplatelet therapy may have value if migraine plays a

role in some cases of transient global amnesia.

Summary

Transient global amnesia is often attributed to a seizure, vascular cause, or migraine, but the outcome is usually benign. The presence of migraine and important

risk factors for stroke necessitates close patient monitoring. Antiplatelet therapy should be considered. PGM

The authors wish to thank Ms Susan Tapia for her help in the preparation of this manuscript.

Address for correspondence: Gilbert J. Toffol, DO, Valley Neurology Associates, 4244 N 19th Ave, Phoenix, AZ 85015.

References

1. Adams RD, Victor M. Principles of neurology. 2d ed. New York: McGraw-Hill, 1981:292-3
2. Fisher CM, Adams RD. Transient global amnesia. *Trans Am Neurol Assoc* 1958;83:143-6
3. Bender MB. Syndrome of isolated episodes of confusion with amnesia. *J Hillside Hosp* 1956;5: 212-5
4. Fisher CM. Transient global amnesia: precipitating activities and other observations. *Arch Neurol* 1982;39(10):605-8
5. Caffarra P, Moretti G, Mazzucchi A, et al. Neuropsychological testing during a transient global amnesia episode and its follow-up. *Acta Neurol Scand* 1981;63(1):44-50
6. Rollinson RD. Transient global amnesia: a review of 213 cases from the literature. *Aust N Z J Med* 1978;8(5):547-9
7. Corston RN, Godwin-Austen RB. Transient global amnesia in four brothers. *J Neurol Neurosurg Psychiatry* 1982;45(4):375-7
8. Croft PB, Heathfield KW, Swash M. Differential diagnosis of transient amnesia. *BMJ* 1973; 4(892):593-6
9. Godwin-Austen RB. Where am I? (Editorial) *BMJ (Clin Res)* 1982;285(6335):85-6
10. Hall JA. Transient global amnesia in neurologues. *Practitioner* 1982;226(1367):953-5
11. Fisher CM, Adams RD. Transient global amnesia. *Acta Neurol Scand* 1964;40(Suppl 9):1-83
12. Benson DF, Marsden CD, Meadows JC. The amnesia syndrome of posterior cerebral artery occlusion. *Acta Neurol Scand* 1974;50(2):133-45
13. Mathew NT, Meyer JS. Pathogenesis and natural history of transient global amnesia. *Stroke* 1974; 5(3):303-11
14. Jensen TS, de Fine Olivarius B. Transient global amnesia: its clinical and pathophysiological basis and prognosis. *Acta Neurol Scand* 1981; 63(4):220-30
15. Caplan L, Chedru F, Lhermitte F, et al. Transient global amnesia and migraine. *Neurology* 1981;31(9):1167-70
16. Fogelholm R, Kivalo E, Bergstrom L. The transient global amnesia syndrome: an analysis of 35 cases. *Eur Neurol* 1975;13(2):72-84
17. Rowan AJ, Protas LM. Transient global amnesia: clinical and electroencephalographic findings in 10 cases. *Neurology* 1979;29(6):869-72
18. Smyth VO, Winter AL. The EEG in migraine. *Electroenceph Clin Neurophysiol* 1964;16(Jan-Feb):194-202
19. Shuping JR, Rollinson RD, Toole JF. Transient global amnesia. *Ann Neurol* 1980;7(3):281-85
20. Nausieda PA, Sherman IV. Long-term prognosis in transient global amnesia. *JAMA* 1979;241(4): 392-3
21. Mazzucchi A, Moretti G, Caffarra P, et al. Neuropsychological functions in the follow-up of transient global amnesia. *Brain* 1980;103(1):161-78
22. Crowell GF, Stump DA, Biller J, et al. The transient global amnesia-migraine connection. *Arch Neurol* 1984;41(1):75-9

Readers are invited to submit brief reports of perplexing cases related to primary medical care. Address submissions to: Manuscript Administrator, POSTGRADUATE MEDICINE, 4530 W 77th St, Minneapolis, MN 55435.