International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2013): 6.14 | Impact Factor (2015): 6.391

A Case of Transient Global Amnesia after Sex

Hesham Khalil, MD

Department of internal medicine, Sahlgrenska University Hospital, Gothenburg, Sweden

Abstract: ***Background: Transient global amnesia (TGA) is a syndrome characterised by a sudden inability to form new memories (anterograde amnesia) and is not associated with any other focal neurological deficit. Various precipitating causes have been reported in the medical literature. No particular treatment is required. Case Report:A case of a 52-year-old man who presented to the emergency department with an episode of acute memory loss. No other focal neurological deficits had been found. His memory returned to normal baseline under 24-hour hospital observation. Conclusion: TGA is a sudden onset of a benign condition that is reversible anterograde amnesia. The condition is associated with repetitive questioning that occurs with an unclear etiology. The presence of a precipitating event and repetitive questioning seem to be key features in making the diagnosis of TGA. Brain imaging is indicated for patients with "high-risk" for more serious pathologies or patients who present with atypical features. TGA requires no treatment as it resolves spontaneously. Clinicians need an awareness of this condition.***

Keywords: TGA, sexual intercourse, repetitive questioning, neurological deficit, memory loss

1. Introduction

Transient global amnesia (TGA) is a syndrome characterised by sudden inability to form new memories (anterograde amnesia), asking repetitive questioning, and is not associated with any other focal neurological deficit. The etiology for TGA remains unknown, although various pathophysiological explanations have been suggested. Treatment is generally not required.

2. Case Report

A 52-years-old man was presented to the emergency department after his wife contacted the ambulance due to an acute attack of confusion. The patient medical history was non-significant and no medication. The patient is a non-smoker. No family history of neurologic disease or atherosclerosis.

The patient is married and he lives with his wife and children. Before the patient was admitted to the emergency department, he had a sexual relationship with his partner. Suddenly after that, he complained of headache in the back of his head. The attack was not associated with light sensation, nausea or vomiting. After the headache attack, the patient started to be confused. He experienced difficulties to remember what happened at that night and to remember his children names although he knew his wife and he could recall old memories. The patient also kept on asking the same questions repeatedly. Despite the headache and confusion, he didn't have any weakness, numbness, slurred speech or double vision. Also, no dizziness, seizure or fever. No history of head trauma, migraine or some previous similar episodes. No intake of alcohol or drugs was identified. He was awake under the attack and his symptoms lasted for approximately 30 minutes with a gradual resolution.

Upon arrival to emergency department, vital signs were stable: BP 145/95 mmHg; pulse 54 beats per minute; and temperature 36,0 C. Glasgow coma scale (GCS) 15/15. Cardiovascular examination revealed regular rhythm without murmurs. A clear vesicular breathing bilateral without wheezes, rhonchi or rales. Abdominal examination was non-significant. No peripheral oedema was noticed. At the time he presented to the emergency department, his symptoms were nearly resolved.

Neurological examination revealed that the patient was awake and oriented to persons, time, and place. His long-term memory was intact and he could recall most of events but for example he couldn't remember his children names. In contrast, his short-term memory was impaired, he kept asking the same questions many times such as what happened? Speech was clear and fluent without dysarthria. Cranial nerve examination showed intact visual field. Eyes movement was normal and no eye deviation, ptosis or nystagmus. Pupils are of normal size and symmetricity and normal direct and indirect light reaction was noticed. His face was symmetrical with normal sensation and normal eye closure and smile. Hearing, head turning, shoulder shrug and tongue protrusion were all normal. Motor examination showed symmetrical normal strength, tone and reflexes bilaterally as well as normal sensation, coordination and gait.

Laboratory investigation including complete blood count showed low normal haemoglobin, normal leukocytes and platelet. Coagulation profile was normal. Similarly, electrolytes, creatinine, lever enzymes, lipid profile and blood sugar were all within the normal range.

Further investigations include: electrocardiogram that showed sinus rhythm and rate with no ST segment changes; computed tomography showed no bleeding or infarction; and telemetry showed no arrhythmias.

After consultation with a neurologist, the patient had been admitted to the hospital for observation. His memory returned to the normal within less than 24 hours and accordingly, he was discharged home with no follow up planed.

3. Discussion

Transient global amnesia (TGA) is a syndrome characterised by sudden inability to form new memories (anterograde amnesia), asking repetitive questioning, lasting up to 24

hours, and is not associated with any other focal neurological deficit[1]. TGA episodes usually last for 2-12 hours. However, a minority (3%) of attacks last < 1 hour. As the attack subsides, the ability to lay down new memories is gradually recovered [2].

Typically, TGA affect middle-aged or elderly patients. Its annual incidence has been reported to be 3.4 to 10.4 per 100,000. The incidence increases to 23.5 per 100,000 per year in people older than 50 years [3].Several precipitating situations for TGA were described by Fisher and Adamsand include: swimming in cold water, taking a hot shower, sexual intercourse, pain, emotional stress, angiography and Valsalva physical activity[4]and myocardial ischemia[5].However, it has been shown that risk factors associated with stroke such as hypertension, diabetes, and hypercholesterolemia are not associated with TGA, while migraines have been found to be strongly associated with TGA[6], [7].

The etiology of TGA remains unknown. However, several hypotheses were suggested in TGA etiology and include: vascular etiology (arterial ischemia and venous congestion theory), migraines, epilepsy, and psychogenic disorders [8]. The arterial ischemia hypothesis is mostly supported by the diffusion-weighted imaging (DWI) changes on magnetic resonance imaging (MRI). However, limitations of this hypothesiswere encountered and included that these changes are inconsistently present, the changes are reversible with time, and they do not respect a clear arterial territory[3]. Another hypothesis for arterial ischemia via arterial thromboembolism has also been considered as mechanism as in transient ischemic attacks (TIA)and TGA. In fact, both TIA and TGA share certain features such as duration of less than 24 hours for the attack and the occurrence in older patients. A limitation for that hypothesis is that TGA episodes usually last longer and patients with TGA tend to have a lower atherosclerotic risk burden[8], [9].

The venous congestion theory has been proposed in which Valsalva maneuver and increased intrathoracic pressure would prevent venous return to the superior vena cava, consequently, would lead to retrograde jugular venous flow in the presence of jugular valve incompetence. This lead to elevated venous pressure to the cerebral venous system which may result in venous ischemia of the mesial temporal lobes [3], [10], [1]. However, several aspects are still not clear regarding the venous congestion theory: (i) why venous congestion is so anatomically selective; (ii) why there is persistence ofmemory impairment long after resolution of the typically short-lasting intrathoracic hypertension; and (iii) why TGA is not seen more commonly in patients with cerebral venous thrombosis provided that this theory is the only explanation. Migraineetiology for TGA has also been proposed. TGA may be similar to an aura via cerebral spreading depression perhaps triggered by hippocampal glutamate release [3], [10]. An argument against this theory is that migraine tends to present in younger individuals, have a recurrent nature and patients do not usuallyexperience symptoms of migraine during TGA.

Epileptiform etiologieshave also been suggested as a cause of TGA, given the transient amnesia may manifest some seizures [6]. However, like migraines, seizures are recurrent and cases of electroencephalogram monitoring during TGA episodes have not revealed any epileptiform activity[6], [11]. Psychological causes of TGA have also been proposed based on findings that subgroups of TGA patients have certain phobia and other personality disorder such as anxiety and depression [9], [10].

The differential diagnosis of TGA includes TIA or stroke, focal seizures (including TEA), postictal state, psychogenic amnesia, metabolic disorders such as hypoglycemia [2], [3],intoxication and drug withdrawal[12]. In these differentials, patients usually experience more global impairment. A diagnostic algorithm for patients presenting with acute-onset anterograde amnesia was proposedby Arena and Rabinstein 2015 [3].

The diagnosis of TGA is based on the clinical history and physical examination. TGA is an exclusion diagnosis and accordingly, an appropriate work-up must be done to exclude alternative diagnoses. To achieve that, serum electrolytes, glucose, ECG and a toxicology screen should be performed. However, transient episodes of amnesia can be caused by seizures which is known as transient epileptic amnesia (TEA). For suspected TEA, an electroencephalogram (EEG) should also be performed beside the tests that were mentioned earlier. Another differential diagnoses are trauma and acute ischemia in which neuroimaging is important for confirmation of such pathologies [3], [10], [13].TGA diagnostic criteria have been suggested as the following[3], [10], [14]: (a) Attack must be witnessed and information about the beginning of the attack is available from an observer (b) head trauma or loss of consciousness at the onset must be excluded, and clouding of consciousness or loss of personal identity should be absent; (c) no accompanying focal neurologic signs or symptoms besides anterograde amnesia; (d) resolution of the memory loss within 24 hours; (e) epileptic features must be absent, and (f) patients with recent head injury or active epilepsy are excluded.

TGA requires no further treatment other than education, explanation, reassurance and psychological support to the patients and their family [2]. The patients have no increased risk of mortality, epilepsy, or stroke following TGA[15].

In our presented case, the previously mentioned criteria for TGA were fulfilled and include: the episode was witnessed; with a clear precipitating event, i.e. sexual relationship; the patient maintained his consciousness throughout the episode; neurological examination was normal; and the patient exhibited a spontaneous return to the baseline level of functioning and memoryin less than 24 hours under hospital observation.

4. Conclusion

Transient global amnesia (TGA) is a sudden onset of a benign conditionthat is reversible anterograde amnesia. The condition is associated with repetitive questioning that occurs with an unclear etiology.TGA may cause a

considerable distress for the patient as well as the relatives. The presence of a precipitating event and repetitive questioning seem to be key features in making the diagnosis of TGA. Brain imaging are indicated for patients with "high-risk" for more serious pathologiesor patients who present withatypical features. Nevertheless, brain imaging relieves the anxiety about more dangerous causes of such events.TGA requires no treatment as it resolves spontaneously. Clinicians need an awareness of TGA condition, diagnosis and reassurance of patients.

References

[1] S. L. Lewis, "Aetiology of transient global amnesia," *The Lancet*, 1998.
[2] R. Shekhar, "Transient global amnesia--a review.," *Int. J. Clin. Pract.*, vol. 62, no. 6, pp. 939–942, Jun. 2008.
[3] J. E. Arena and A. A. Rabinstein, "Transient global amnesia.," *Mayo Clin. Proc.*, vol. 90, no. 2, pp. 264–272, Feb. 2015.
[4] C. M. FISHER and R. D. ADAMS, "TRANSIENT GLOBAL AMNESIA.," *Acta Neurol. Scand., Suppl.*, vol. 40, pp. SUPPL 9:1–83, 1964.
[5] C. Agosti, B. Borroni, N. M. Akkawi, T. Bordonali, and A. Padovani, "Acute myocardial infarction presenting with transient global amnesia.," *J Am Geriatr Soc*, vol. 54, no. 6, p. 1004, Jun. 2006.
[6] K. Sander and D. Sander, "New insights into transient global amnesia: recent imaging and clinical findings.," *Lancet Neurol*, vol. 4, no. 7, pp. 437–444, Jul. 2005.
[7] J. W. Miller, R. C. Petersen, E. J. Metter, C. H. Millikan, and T. Yanagihara, "Transient global amnesia: clinical characteristics and prognosis.," *Neurology*, vol. 37, no. 5, pp. 733–737, May 1987.
[8] M. Zorzon, L. Antonutti, G. Masè, E. Biasutti, B. Vitrani, and G. Cazzato, "Transient global amnesia and transient ischemic attack. Natural history, vascular risk factors, and associated conditions.," *Stroke*, vol. 26, no. 9, pp. 1536–1542, Sep. 1995.
[9] P. Quinette, B. Guillery-Girard, J. Dayan, V. de la Sayette, S. Marquis, F. Viader, B. Desgranges, and F. Eustache, "What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases.," *Brain*, vol. 129, no. 7, pp. 1640–1658, Jul. 2006.
[10] S. Kremen, M. Mendez, and J. Wilterdink, "Transient Global Amnesia" In: UpToDate, Post TW (Ed), UpToDate, Waltham, MA. (Accessed on October 24, 2016.), 2016.
[11] D. E. Jacome, "EEG features in transient global amnesia," *Clinical EEG and Neuroscience*, 1989.
[12] P. Bonnet, P. Niclot, F. Chaussin, M. Placide, M. P. Debray, and A. Fichelle, "A puzzling case of transient global amnesia.," *Lancet*, vol. 364, no. 9433, p. 554, Aug. 2004.
[13] D. Owen, B. Paranandi, R. Sivakumar, and M. Seevaratnam, "Classical diseases revisited: transient global amnesia.," *Postgrad Med J*, vol. 83, no. 978, pp. 236–239, Apr. 2007.
[14] J. R. Hodges and C. P. Warlow, "Syndromes of transient amnesia: towards a classification. A study of 153 cases.," *J. Neurol. Neurosurg. Psychiatr.*, vol. 53, no. 10, pp. 834–843, Oct. 1990.
[15] L. Pantoni, E. Bertini, M. Lamassa, G. Pracucci, and D. Inzitari, "Clinical features, risk factors, and prognosis in transient global amnesia: a follow-up study.," *Eur. J. Neurol.*, vol. 12, no. 5, pp. 350–356, May 2005.