The diagnosis and management of transient global amnesia in the emergency department

Magnus Harrison, Mark Williams

Transient Global Amnesia (TGA) is a benign and temporary loss of anterograde memory with the preservation of remote memories and immediate recall. TGA was first described in 1956 and since then epilepsy, transient ischaemic attacks (TIA), migraine and now intracranial venous stasis have been implicated in its aetiology. Precipitants of TGA include physical exertion and valsalva-like manoeuvres. In order to diagnose TGA the criteria created by Hodge and Warlow in 1990 can be used. This requires the episode of memory loss to be witnessed and involve anterograde amnesia. The patient must not have any evidence of neurological signs or deficits, features of epilepsy, active epilepsy or recent head injury. Finally the episode must have resolved within 24 h. In this case study the patient's symptoms are mistakenly attributed to a TIA. There is no increased risk of TIA or CVA in patients who have had TGA and there are no increased levels of mortality amongst these patients. In this article we aim to help doctors working in the emergency department to diagnose and manage TGA.

Transient global amnesia (TGA) is a benign, temporary loss of anterograde memory with sparing of immediate recall and remote memories. Worldwide, its incidence is approximately 2.9–10/100 000 and it is thought to be due to intracranial venous stasis leading to ischaemia in the areas of the brain associated with memory. There are many precipitants including physical exertion and valsalva-like manoeuvres. TGA can be diagnosed using the Hodges and Warlow criteria created in 1990. The most important part of managing an episode of TGA is the psychological support of the patient and his or her relatives.

TGA was first described by M Bender in 1956.[1] Although this is not an uncommon presentation of acutely altered neuropsychiatric status, it may be new to the members of an emergency department.[2] TGA needs to be differentiated from the diagnosis of cerebrovascular accident (CVA) and subarachnoid haemorrhage.[3]

In this case study we give an example of a presentation of TGA to the emergency department along with important facts that will help doctors to manage these patients.

CASE STUDY

A woman was strenuously cutting her hedge when she suddenly froze. Afterwards she had no recollection of the previous 2 h and was unable to retain any new information for longer than a few minutes. She had no other symptoms such as headache or confusion. After visiting her general practitioner she was diagnosed as having had a transient ischaemic attack (TIA). However her husband was still worried when her symptoms did not resolve, so he took her to the emergency department.

The patient had no history of hypertension, raised cholesterol, previous myocardial infarction or CVAs, transient ischaemic attacks, recent head injury, epilepsy or migraine. She did not drink and had quit smoking a few years ago, having smoked 20 cigarettes per day for approximately 45 years. The only medication she took regularly was diclofenac that was prescribed for osteoarthritis in her left hand.

On examination she appeared relaxed. Her airways were open with no crackles or wheezes, her respiratory rate and oxygen saturation were normal, her heart rate was 64 beats per minute and had a regular rhythm, and her blood pressure was raised at 180 mm Hg systolic and 84 mm Hg diastolic. Her Glasgow Coma Scale was 15/15 and her abbreviated mini-mental test score was 9/10 (her immediate recall of three words was spared, but she was unable to remember any of the three words after three minutes). Her cranial and peripheral nerves were normal. She was apyrexic and her capillary blood glucose was 5.2 mmol/l. Her ECG and urinalysis were normal and her full blood count, urea and electrolytes and cholesterol were all within normal ranges.

The diagnosis of the patient's problems was transient global ischaemia. By this time her anterograde amnesia was beginning to resolve. After discussion with a neurology registrar she was admitted to the neurology ward where she received a CT scan of her brain that showed old ischaemic change. The neurology consultant agreed with our diagnosis. He saw the patient in a follow-up clinic where he reassured her about the diagnosis and then discharged her with a letter to her general practitioner to monitor her risk factors for CVA.

DISCUSSION

The incidence of TGA is approximately 2.9–10/100 000 worldwide.[1] Arterial ischaemia (TIA), epileptic seizures and migraine were previously thought to be the most likely factors causing TGA.[4 5] Now the most likely cause of this condition is thought to be intracranial venous congestion leading to ischaemia in the hippocampus and other mesiotemporal structures of the brain associated with creating and storing memories.[1 4 5] This is caused by Valsalva-like manoeuvres increasing the intrathoracic pressure and causing venous reflux in the vessels draining the brain.[1 5 6] A study published in *Neurology* used three-dimensional time-of-flight magnetic resonance angiography and upper extremity digital subtraction venography to show that left brachiocephalic vein occlusion caused retrograde intracranial venous flow in 50% of those having TGA compared with none in the control group ($p<0.001$).[6]

Pre-attack factors associated with TGA include physical exertion, swimming, sexual intercourse and use of marijuana and Viagra.[1 5 7] The greatest risk factor for TGA is a history of migraines.[7] Patients who have had an episode of TGA have no increased risk of having a TIA or CVA, as they have different risk factors.[4]

In order to diagnose TGA the patient must satisfy the following criteria, created by Hodge and Warlow in 1990[8]:

(1) The attack was witnessed and reported.
(2) There was obvious anterograde amnesia during the attack.
(3) There was an absence of clouding of consciousness.
(4) There were no focal neurological signs or deficits during or after the attack.
(5) There were no features of epilepsy.
(6) The attack resolved within 24 h.
(7) The patient did not have any recent head injury or active epilepsy.[8]

As the prognosis of TGA is very good (it has a small recurrence rate and no increased mortality rate), the most important part of management after diagnosis is looking after the psychological needs of the patient and his or her relatives.[2] Seeing a once competent and

healthy partner, sibling or parent become incapable of remembering what was said only a minute ago is very distressing, and hence it is often the relatives who will require reassurance.

Emerg Med J 2007;**24**:444–445.
doi: 10.1136/emj.2007.046565

Authors' affiliations
Magnus Harrison, Mark Williams, University Hospital of North Staffordshire, Stoke-on-Trent, UK

Correspondence to: Dr M Williams, University Hospital of North Staffordshire NHS Trust, North Staffordshire Royal Infirmary, Princes Road, Stoke-on-Trent, Staffordshire ST4 7LN, UK; m.williams@doctors.org.uk

Accepted 17 January 2007

Competing interests: None declared.

Informed consent was obtained for publication of the person's details in this report.

REFERENCES

1 **Quinette P**, Guillery-Girard B, Dayan J, *et al*. What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. *Brain* 2006;**129**(Part 7):1640–58.
2 **Basior J**, Vogel S, Mitton J. Transient global amnesia in the emergency department. *Am J Emerg Med* 1985;**3**:352–7.
3 **Monzani V**, Rovellini A, Schinco G, *et al*. Transient global amnesia or subarachnoid haemorrhage? Clinical and laboratory findings in a particular type of acute global amnesia. *Eur J Emerg Med* 2000;**7**:291–3.
4 **Pinol-Ripoll G**, de la Puerta Gonzalez-Miro I, Martinez L, *et al*. A study of risk factors in transient global amnesia and its differentiation from a transient ischaemic attack. *Rev Neurol* 2005;**41**:513–6.
5 **Rashid J**, Starer J. Transient global amnesia following a transoceanic flight. *Psychiatry Clin Neurosci* 2006;**60**:516.
6 **Chung CP**, Hsu H, Chao A, *et al*. Detection of intracranial venous reflux in patients of transient global amnesia. *Neurology* 2006;**66**:1873–7.
7 **Moreno-Lugris XC**, Martinez-Alvarez J, Branas F, *et al*. Transient global amnesia. Case–control study of 24 cases. *Rev Neurol* 1996;**24**:554–7.
8 **Hodges JR**, Warlow CP. Syndromes of transient amnesia: towards a classification; a study of 153 cases. *J Neurol Neurosurg Psychiatry* 1990;**53**:834–43.