

'What is happening?': a case of transient global amnesia

Introduction

Transient global amnesia is a temporary condition of short-term anterograde memory loss. Patients usually have no other neurological disturbance, are fully awake and have full recall of older established memories, but are frequently anxious or agitated about their current status. The reported incidence is 5 per 100 000 of the population, is most common between the ages of 40 to 80 years (Quinette et al, 2006), has no gender or racial preference and remains a condition of indeterminate origin.

Discussion

Transient global amnesia is a condition presenting as a sudden and almost complete disruption of short-term memory without other neurological features. To diagnose the condition, specific criteria need to be satisfied (Table 1).

This patient had suffered no significant traumatic event, aside from striking

her chin 2 weeks previously, and had not experienced any recent stressful or strongly emotional event. In patients with transient global amnesia it is important to exclude potential differential diagnoses (Table 2) (Owen et al, 2007).

This patient had none of the features of the causes listed in Table 2, was of the age in which this condition is usually seen (Zeman and Hodges, 1997), and in addition had never suffered classical or atypical forms of migraine. She was understandably anxious and, in common with other descriptions, felt nauseous. Perseveration of questioning is a common feature also exhibited by this patient as was complete resolution of her symptoms (except for the

period of amnesia) within 24 hours. Most patients will require no further investigation, although any recurrence should prompt consideration of an electroencephalogram and in some cases cerebral magnetic resonance and positron emission tomographic scanning.

The pathophysiology of transient amnesia remains contentious with several potential causes (Hodges and Warlow, 1990). It is important to exclude a psychogenic, drug- or alcohol-related event. An atypical form of epilepsy has been suggested but electroencephalography does not usually support this, along with the fact that recurrence is unusual. Transient epileptic amnesia is character-

Table 1. Diagnostic criteria for transient global amnesia

Attacks should be witnessed
Acute onset anterograde amnesia must be present
Consciousness is maintained and with no additional cognitive deficit
No focal neurological impairment should be present
Epileptic features must be absent
Attacks resolve within 24 hours
No recent head trauma
Other causes of amnesia must be excluded

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Table 2. Key differentials in the diagnosis of transient global amnesia

Acute confusional states (i.e. infection related)
Epileptic events
Transient epileptic amnesia (a manifestation of temporal lobe epilepsy)
Psychogenic amnesia (usually related to a precipitant psychosocial stressor)
Transient ischaemic attacks or established stroke

Case Report

A 62-year-old woman was referred to the accident and emergency department with a loss of immediate memory of approximately 5 hours' duration. The last thing she clearly recalled was her birthday which occurred 2 days earlier. She repeated the same question 'what is happening?' Examination revealed a healthy but tearful and anxious middle-aged woman. She reported feeling nauseous but had not vomited and had no other symptoms. Past medical history included treated hypothyroidism. The patient had never smoked, drank less than 2 units of alcohol per fortnight and worked in the education sector. She had fallen 2 weeks previously and had struck her chin but suffered no head trauma.

The patient was apyrexial (36.8°C), had a heart rate of 101 beats/min, blood pressure of 134/87 mmHg and an oxygen saturation of 98%. Neurological examination demonstrated an Abbreviated Mental Test Score of 2 out of 10 and Glasgow Coma Scale score of 14 out of 15. No additional deficit was found on a full neurological examination. A full blood count, renal, liver, thyroid function tests and glucose levels were normal, in addition to normal serum calcium, magnesium, vitamin B₁₂, folate, troponin and C-reactive protein levels. A chest radiograph and computed tomography scan of the brain were normal. The electrocardiogram showed previously documented left bundle-branch block.

Four hours after admission to hospital, the Abbreviated Mental Test Score had risen to 9 out of 10. By the following morning she had fully recovered and was discharged home with an outpatient neurology follow-up appointment.

ized by a briefer duration of usually less than 1 hour, recurrent episodes and associated epileptiform stigmata (aura, temporary unresponsiveness). An association with migraine has not been confirmed. It seems likely that the aetiology may be multi-factorial but localized (hippocampal region) cerebral arterial ischaemia or possibly venous cerebral congestion (related to jugular vein incompetence) may be responsible (Schreiber et al, 2005). **BJHM**

Hodges JR, Warlow CT (1990) Syndromes of transient amnesia: towards a classification of 153 cases. *J Neurol Neurosurg Psychiatry* 53(10): 834–43

Owen D, Paranandi B, Sivakumar R, Seevaratnam

M (2007) Classical diseases revisited: transient global amnesia. *Postgrad Med J* 83: 236–9 (doi: 10.1136/pgmj.2006.052472)

Quinette P, Guillery-Girard B, Dayan J et al (2006) What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. *Brain* 129: 1640–58 (doi: 10.1093/brain/awl105)

Schreiber SJ, Doepp F, Klingebiel R, Valdeuz JM (2005) Internal jugular vein valve incompetence and intracranial venous anatomy in transient global amnesia. *J Neurol Neurosurg Psychiatry* 76(4): 509–13 (doi: 10.1136/jnnp.2004.043844)

Zeman AZ, Hodges JR (1997) Transient global amnesia. *Br J Hosp Med* 58(6): 257–60

LEARNING POINTS

- The diagnosis of transient global amnesia can frequently be made from the history of a reliable witness to events.
- The exclusion of all frequent causes of impaired cerebral function is required to make this diagnosis.
- Transient global amnesia may have a multi-factorial aetiology with no single pathophysiology identified.

IMAGES IN MEDICINE

Peripheral T-cell lymphoma with left ankle swelling, ulceration and fever

Peripheral T-cell lymphoma constitutes about 10–15% of all cases of non-Hodgkin's lymphoma in adults, according to the World Health Organization classification (Savage, 2007). These lymphomas are prevalent in Asia and parts of central and south America, but rare in Europe or north America (Rüdiger et al, 2002).

A 50-year-old man presented to the authors' hospital several times, with left ankle swelling, ulceration and fever. Physical examination showed slightly pitting oedema of the left leg and an ulcer about 3x2 cm in size at the left ankle, with a small amount of yellow transparent liquid penetration (Figure 1). Laboratory data were almost normal. A week later, the

patient, who was still in hospital, developed fever, and the lesion was even worse. Pathological examination of the skin tissue indicated that the lesion was left ankle extranodal NK/T-cell lymphoma. Positron emission tomography-computer tomography showed that the tumour had involved the lymph nodes of the bilateral popliteals, the thigh root and groin (Figure 2). **BJHM**

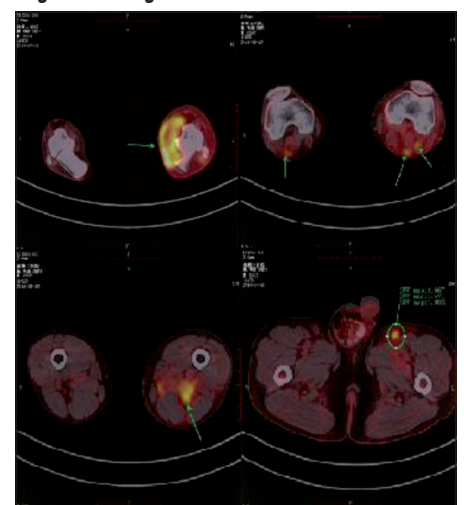
Figure 1. Left leg showing slightly pitting oedema, and an ulcer about 3 x 2 cm in size at the left ankle, with a small amount of yellow transparent liquid penetration.



Rüdiger T, Weisenburger DD, Anderson JR et al (2002) Peripheral T-cell lymphoma (excluding anaplastic large-cell lymphoma): results from the Non-Hodgkin's Lymphoma Classification Project. *Ann Oncol* 13: 140–9

Savage KJ (2007) Peripheral T-cell lymphomas. *Blood Rev* 21: 201–16 (doi: 10.1016/j.blre.2007.03.001)

Figure 2. Positron emission tomography-computer tomography showed that the tumour had involved the lymph nodes of the bilateral popliteals, the thigh root and groin.



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