An Episode of Acute Amnesia: To CT or not to CT?

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ABSTRACT

Isolated, acute amnesia is an uncommon neurological presentation and may only be mentioned in passing during medical school. This report discusses a typical case and debates whether neuroimaging is required.

CASE INTRODUCTION

Mrs B, a 61-year-old European nurse, was brought in by ambulance to Waikato Hospital with two hours of sudden-onset confusion, repeating sentences and amnesia. She also complained of nausea and a headache.

HISTORY

The episode began at 10:30am while Mrs B was at home on the phone with her sister, with husband Mr B in the adjacent room. All three were present in the hospital to contribute to the history.

There were no preceding symptoms. Midway through a distressing conversation, Mrs B suddenly seemed confused and began to repeat sentences every two minutes, such as, "Is [granddaughter] due to have a baby?" She was crying and could not remember that her granddaughter was pregnant.

Thirty minutes later, she developed a constant, occipital headache that was six out of ten in severity with uncertain acuity of onset. She also felt mildly nauseated but there was no vomiting.

At the hospital, her last memory was of being in the ambulance. She could not remember anything else from that day. She also could not recall that Mr B's birthday was on the previous day.

Mrs B was fully conscious, her language function was intact and she recognised herself and her relatives. There was no visual loss, numbness, weakness, incontinence or history of head trauma. Further questioning revealed no symptoms suggestive of epilepsy or meningitis. Mrs B also denied any chest pain, dyspnoea and palpitations. There was no previous history of similar episodes.

The past medical history was significant for migraines, occurring once or twice per year and associated with unilateral periorbital pain, blurry vision, nausea and vomiting. She also suffered from constant musculoskeletal lower neck pain, and was uncertain if this was causing her headache. Lastly, she had a previous major depressive episode and was on long-term paroxetine.

Her cardiovascular risk factors were reasonably significant with a 5-year cardiovascular disease risk of 5-10%,[1] including the fact that her father had a myocardial infarction before the age of 55; this risk was increased by hormone therapy for menopause. She had hypertension treated with quinapril.

The social history was of a nursing tutor living independently at home with her husband. She was an ex-smoker of six pack years with no alcohol intake.

EXAMINATION

She appeared well, fully alert but slightly anxious. The vital signs were within normal limits. She conversed appropriately but could not remember a conversation from ten minutes ago. The headache increased on neck extension and rotation, and the range of lateral neck flexion was limited by neck pain. She dropped two points in the Ten Point Mental Status Questionnaire shown in Figure 1. Examination was otherwise unremarkable with no focal neurology.

- ☐ Place
- ☐ Year
- ☐ Time to nearest hour
- ☐ Birthday
- ☐ Age

Give address for recall at end of test.

- ☐ Year WWII started or finished
- ☐ Name of the Prime Minister
- ☐ Count backwards from 20 to 1
- ☐ Recognition of two persons
- ☐ Recall address

Total 8/10

Also did not know the date

Figure 1. Ten Point Mental Status Questionnaire used at Waikato Hospital

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Likely transient global amnesia (TGA), with the differential diagnoses listed in Figure 2., of which certain could be ruled out on history:[2]

1. Transient epileptic amnesia also features repetitive questioning but usually has a duration less than one hour, recurrent episodes, and occurs on waking.

2. A temporal lobe stroke or TIA is likely to have associated visual symptoms or signs.

3. Delirium features inattention and Mrs B would not have been able to count backwards from 20 to 1 if this were the case.

4. Korsakoff's syndrome also features inattention and is usually associated with chronic alcoholism or thiamine deficiency, not evident in Mrs B.

5. Psychogenic amnesia may feature a psychosocial or emotional trigger but is characterised by a loss of identity.

Subarachnoid haemorrhage and vertebral artery dissection could not be ruled out on history alone.

Figure 2. Differential Diagnosis

POSSIBLE

Transient epileptic amnesia
Subarachnoid haemorrhage
Temporal lobe stroke or transient ischaemic attack (TIA)
Vertebral Artery Dissection

UNLIKELY

Delirium (Acute Confusional State)
Korsakoff's syndrome
Psychogenic amnesia

MANAGEMENT

Serum electrolytes and glucose were normal. Urine toxicology was clear. A head computed tomography (CT) was performed and also came back normal.

Mrs B was observed in hospital for 24 hours. By then, her amnesia had resolved, though an amnestic gap remained. She was discharged in time to see her granddaughter deliver her baby the next day.

DISCUSSION

OVERVIEW

Described in 1956,[3] TGA is a benign neurological syndrome lasting less than 24 hours characterised by acute onset, severe anterograde amnesia and repetitive questioning in the absence of focal neurology.[2]

The incidence is 1 in 20000 per year, rising to 1 in 4000 per year in those aged 50 years and older.[4] It mainly affects individuals aged 40 to 80 with an average age of 61 – Mrs B's age exactly.[2] There is no gender predilection and no relationship with cardiovascular disease. Higher rates of migraine and psychiatric disease are seen in those with TGA compared to healthy controls, both notable in Mrs B's past medical history.[2]

Diffusion-weighted magnetic resonance imaging (MRI) confirms that the neurological lesions are located in the CA1 subfield of the hippocampus. During an episode the CA1 neurons undergo a physiological disturbance that resolves without any structural changes or long-term consequences.[5] The mechanism of injury to these neurons is still uncertain. Arterial, venous, migrainous, epileptic and psychological hypotheses have not been confirmed.[2]

Arterial or thromboembolic aetiology is tempting to entertain given the sudden onset of this condition, but a meta-analysis[6] has shown no relationship between stroke risk factors and TGA; and the lesions seen on MRI do not look or act like infarcts. Venous aetiology was considered since Valsalva manoeuvres are often the precipitating event but dedicated studies of jugular vein flow have not found a relationship with TGA. The cortical spreading depression of migraine has not been replicated in the hippocampus for TGA, although there may be a link given that there are higher rates of migraine in these patients.[7] Electroencephalography (EEG) recordings during and after TGA attacks have never supported the epileptic hypothesis. Finally, there may be a link with psychiatric illness given that higher rates of psychiatric disease are seen in these patients.[2] Because no single hypothesis has explained the aetiology, it may be that TGA is a syndrome with multiple causes but the same presentation.

DIAGNOSIS

Figure 3: Diagnostic criteria [8]

Must have reliable collateral history from witness who was present during episode

Must have anterograde amnesia

No clouding of consciousness

No loss of personal identity

No cognitive impairment other than amnesia

No focal neurological symptoms or signs

No epileptic features

Must resolve in 24 hours

No recent head injury, no seizures in the last two years and not on medication for epilepsy

TGA is a clinical diagnosis that can be made by fulfilling all seven criteria outlined in Figure 3. Despite advances in neuroimaging the criteria have not changed since first derived from 153 cases in 1990 by Hodges and Warlow.[8] The key differential diagnoses have been discussed during the case presentation. Although non-specific, Figure 4 lists other clues that may clinch the diagnosis to the astute clinician.

Figure 4: Other features in a typical history [2,4]

Triggering events – strong emotions, physical activity, Valsalva manoeuvre, changes in posture, medical procedures, high altitude, contact with water, and changes in body temperature

Retrograde amnesia – from hours to years

Disorientation – to time and place

Associated symptoms – agitation, anxiety, nausea, paraesthesias; headache occurs in 10 to 40 per cent

Duration – six hours on average

MANAGEMENT

Blood glucose, serum electrolytes and urine toxicology are important screening tests.[4]
EEG recording should be performed if querying the diagnosis of transient epileptic amnesia. The debate around neuroimaging is discussed below.

If symptomatic, the patient should be admitted to hospital for safety reasons until the episode resolves. No treatment is indicated. No driving restrictions are necessary following a single episode, as recurrence is rare.

TO CT OR NOT TO CT?

The problem is that there is no way to diagnose TGA with the criteria unless the episode resolves within 24 hours. Serious differential diagnoses must be excluded and cannot wait.

Despite being over two decades old, the diagnostic criteria have not changed since they were outlined in Hodges and Warlow's 1990 study.

However, their recommendations on neuroimaging are more difficult to adhere to. In their study, 95 of the 114 cases that fulfilled all seven diagnostic criteria (83%) had serial head CTs; all of the CTs were normal. Thus they recommended against routine CT scanning of patients who fulfilled the criteria, with an episode over one hour, and no focal neurology. They recommended CT for episodes less than one hour, recurrent attacks or for those who did not fulfil the criteria.[8]

Consensus remains that there are no CT changes to be seen in TGA. Its purpose is to rule out sinister pathology. Given the availability of CT scanning nowadays, current guidelines recommend for routine neuroimaging even if all of the diagnostic criteria are fulfilled.[4]

The detection rate of hippocampal lesions is up to 85% with ideal MRI parameters and timing of 24 to 72 hours after the onset of the episode.[7] However, routine MRI for every patient is expensive and the costs are not justified: MRI performed earlier than 24 hours has a low detection rate and its purpose at this stage to rule out other pathology could be carried out by CT; after 24 hours the clinical diagnosis can be confirmed, and so an MRI positive for hippocampal lesions would not change management.

For Mrs B, a "textbook" account of the episode was provided, which satisfied six out of seven of the diagnostic criteria and all other features of a typical history. She was the average age of those affected, and had both risk factors of previous migraines and psychiatric disease. The dilemma arose from Mrs B's occipital headache. The fact that she had amnesia made it difficult to determine whether this was new or old. In the end a head CT was justified, as she did not satisfy all seven of the diagnostic criteria on admission. With her background of hypertension the benefits of ruling out vascular pathology outweighed the harms of irradiation.

Had Mrs B's episode resolved by the time she was admitted to hospital, then she would have fulfilled all seven criteria, and a CT would not have been necessary according to Hodges and Warlow's recommendations. However, most clinicians would still perform a CT scan even if all seven criteria were fulfilled3,4 because TGA is rare compared to stroke, which is common in this age group. CT is quick and easy and it is reassuring to rule out sinister pathology for such an unusual presentation.

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