ANSWER TO PHOTO QUIZ (PAGE 217)

A GAP IN MEMORY TAPE

DIAGNOSIS

Based on the clinical scenario alone, a provisional diagnosis of transient global amnesia (TGA) was made and alternative conditions such as acute confusional state, psychogenic amnesia, seizure as well as transient ischaemic attack (TIA) were considered to be possible differential diagnoses. Our clinical suspicion was later confirmed when the MRI showed a typical appearance for TGA (see *figure 1*).

TGA is characterised by sudden anterograde memory impairment of no more than 24 hours and patients recover fully without any long-term consequences. Patients retain personal identification, do not lose consciousness and can function during the episode.

The aetiology of TGA is a matter of continuing debate and although the clinical features are well defined, the exact pathophysiological mechanism remains unclear. Several aetiological possibilities have been suggested including ischaemia, migraine, epileptic phenomena and hippocampal venous congestion.[1] In this text, we provide evidence against the ischaemic hypothesis.

Focal diffusion lesions can be selectively seen in the CA-1 field of the hippocampal cornu ammonis and detection is maximal 48-72 hours after symptom onset.[2] CA-1 neurons are involved in the process of memory stabilisation after its initial acquisition.

Given the diffusion restriction on MRI, TGA is often mislabelled as an acute ischaemic event. Multiple studies, however, have shown evidence against arterial ischaemia as an underlying mechanism in TGA.[3,4] The lack of signal abnormality in FLAIR sequences in repeat imaging (*figure 1*) is against ischaemia as we would expect to see persistent gliotic changes in stroke cases.[4] Secondly, bilateral hippocampal DWI changes are reported in 16.3% of patients with TGA,[5] again making an ischaemic cause unlikely in view of the simultaneous involvement of two different vascular territories. Thirdly, Toledo et al. investigated perfusion MRI in the acute phase of TGA and it was found to be normal.[3]

In summary, TGA is a benign clinical syndrome with a low recurrence rate. No specific treatment is required, however, differentiation from epilepsy and TIA is of paramount importance because these conditions require different diagnostic and therapeutic approach; and have different prognostic implications.

DISCLOSURES

The authors report no disclosures relevant to the manuscript.

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