offered immediate chemical or electrical cardioversion, without undue concerns regarding clot formation and consequent embolic phenomena.\textsuperscript{3,4} Patient education and improvement in GP referral systems could increase this number via early expert assessment, and thus allow prompt conversion to sinus rhythm, thereby reducing length of stay and out-patient DCCV lists.

Transoesophageal echocardiography-guided DCCV is suitable for those patients with AF of onset > 48 h, or unknown, who qualify for rhythm control. It shortens hospital stay and enables prompt DCCV without the initial anticoagulation phase. A significant subgroup (28\%) can be potentially identified for this promising management strategy.

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doi:10.1093/qjmed/hci021

\textbf{Transient global amnesia secondary to herpes simplex viral encephalitis}

Sir,

A 39-year-old company director had been under recent financial stress. On the day of hospital admission, he had been at his desk for an hour without complaint. Colleagues witnessed him suddenly becoming distressed, and, although he was not directly spoken to, he left the office and drove three miles home. On arrival, his wife reported that he was agitated, could not explain why he had returned home, and repeatedly asked what day it was; these symptoms lasted around 4 h. No focal weakness was witnessed; the ability to drive suggests praxis was normal, and the content and fluidity of his speech indicates the absence of aphasia.

His agitation had settled by the time he was reviewed by the neurology team 8 h after the onset. He had dense amnesia for events extending from the time he was at work to being in hospital. He was afebrile, and there were no focal neurological signs. Mini-Mental State Examination scored 26/30, revealing errors in recalling the day, ward and short-term recall of 1 of 3 objects.

On admission, serum blood analysis was unremarkable and a contrast-enhanced CT of the brain was normal. Lumbar puncture demonstrated an opening pressure of 20 cm of water, and CSF analysis revealed 6 WBC (lymphocytes), 480 RBC, 211 mg/l protein and 3.9 mmol/l glucose (serum 5.4 mmol/l). In view of the modestly elevated CSF lymphocyte count, intravenous aciclovir was instigated on day one.

An electroencephalogram performed on the second hospital day showed occasionally sharp waves in the right temporal leads. A MRI of the brain on the seventh hospital day revealed no abnormalities. PCR for HSV type 1 was positive on the day 1 CSF. A repeat CSF sample on day 11 was again positive for HSV PCR (Table 1).

The MMSE on day 2 had improved to 29/30 (regarded as normal). The patient returned to work after 4 weeks; formal neuropsychology 2 months after presentation revealed no deficits. No illnesses relating to an immunocompromised status have developed in the 2 years following the illness.

Our patient fits diagnostic criteria for transient global amnesia.\textsuperscript{1} He developed abrupt onset of amnesia without disturbance of consciousness,

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|}
\hline
Day & 1 & 10 & 20 \\
\hline
CSF RBC & 480 & 0 & 4 \\
CSF WBC & 6 & 6 & 0 \\
CSF protein (mg/l) & 211 & 217 & 299 \\
CSF glucose (mmol/l) & 3.9 & 2.7 & 2.6 \\
Serum glucose (mmol/l) & 5.4 & 4.5 & 4.6 \\
CSF PCR-HSV & + & + & – \\
\hline
\end{tabular}
\caption{Test results}
\end{table}

CSF, cerebrospinal fluid; RBC, red blood cell count; WBC, white blood cell count; PCR-HSV, polymerase chain reaction test for herpes simplex virus.
focal neurological symptoms or epileptic features. Repetitive questioning and agitation were prominent features. The modest CSF pleocytosis, localized EEG abnormality and CSF positive for HSV DNA on two separate CSF occasions provides powerful evidence for a diagnosis of HSE.

This the first report to suggest HSE can be expressed with the clinical phenotype of TGA alone. There is one published case of TGA occurring < 24 h prior to the onset of a typical HSVE presentation, with a diagnosis not based on HSV PCR. In our case aciclovir was instigated on the first hospital day, and this may have changed the natural history of the condition in our patient. Normal brain MRI is expected in TGA, and occasionally recognized in HSVE.

In the majority of cases of TGA, no cause can be identified, imaging is invariably normal, and CSF studies are not undertaken. EEGs during episodes have demonstrated no epileptiform abnormalities. Epileptogenic, migrainous and vascular aetiologies have been considered, although there remains no consensus of opinion.

PCR for HSV DNA is the gold standard in diagnosis of HSVE. This case suggests that TGA alone is part of the widening clinical spectrum. Whether aciclovir changed the natural history in this case is unknown. This is the first case to demonstrate that HSV can be an aetiological factor in TGA; the frequency in which it is implicated has not previously been systematically examined and would be a research question of interest.

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doi:10.1093/qjmed/hci022