doi:10.1093/qjmed/hci063

Transient global amnesia or transient epileptic amnesia?

Sir,

We read with great interest the letter of McCorry and Crowley concerning an attack of transient global amnesia (TGA) considered secondary to herpes simplex viral encephalitis. Some features of the reported attack are unusual. The patient was a relatively young person: 39 years compared with an average age of 61 with TGA. At the beginning the patient suddenly became ‘distressed’; he realized the change and left office. With TGA, the disturbance is usually obvious to witnesses but not to the patient himself. The EEG performed on the second day exhibited right temporal sharp waves. During and after TGA, as a rule the EEG is normal. Epileptiform abnormalities do not occur. TGA has to be differentiated from epileptic seizures with prominent disturbances of mnemonic functions. After the first description, a number of further patients have been reported under different designations: epileptic amnesic attack, pure amnesic seizure or transient epileptic amnesia (TEA). In most cases, the separation from TGA should be easy keeping in mind the frequent recurrence and short duration of TEA, the combination with further seizure types and the abnormal EEG findings including temporal sharp waves. Repetitively asking the same question is typical for TGA, but can also be observed with TEA. A long duration is unusual for TEA. However, pure prolonged transient amnesia can occur in the form of non-convulsive status epilepticus. We would suggest an epileptic aetiology for the attack reported by McCorry and Crowley, rather than TGA.

The aetiology of TGA has been under discussion for years, advocating ischaemic, migrainous, epileptic and other causes. None of these appears completely convincing. Clearly symptomatic cases have been described with mild head injury, stroke and space-occupying lesions, among others. TGA symptomatic to herpes simplex encephalitis would represent a novelty. PCR results should be interpreted cautiously when neither signs and symptoms nor CSF findings or imaging results can readily be attributed to the virus detected. TEA marking the start of a temporal lobe epilepsy—non-lesional so far—seems to be a more plausible interpretation. The occurrence of some inconspicuous simple partial seizures (auras) in the past or further seizures in the future would clarify the situation and offer the opportunity of anti-epileptic drug treatment.

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References

Response

We think Dr Bauer’s suggestion that the underlying cause in our case was not HSVE is extremely unlikely. There is an extensive literature indicating the specificity of HSE PCR is consistently > 99% (i.e. has a very low false positive rate) this being based on testing of large numbers of CSF samples from individuals with conditions other than herpes simplex encephalitis. The reference cited by Bauer et al. provides no evidence to question the validity of this assertion. Our case demonstrated a positive PCR for HSV on two distinct CSF samples taken 10 days apart, and on this basis the diagnosis of HSVE is extremely likely. Further supporting evidence is revealed by a mild CSF lymphocytosis.
in both distinct samples, suggesting an inflammatory or infective process, combined with the clinical presentation of sudden-onset amnesia which, in our view, forms irrefutable evidence that the aetiology of the illness was HSVE.

TGA is a clinical diagnosis, and the presentation was consistent with the clinical diagnostic criteria outlined by Hodges and Warlow for TGA.\textsuperscript{4} We agree the age was below the mean for patients with the condition, and this indeed prompted us to examine the CSF. The EEG abnormality is entirely consistent with HSV infection. We did consider whether HSV infection might have caused an episode of non-convulsive status, but with strictly limited editorial space we did not speculate on this within the article. The learning point from the case is that HSVE can present with symptoms that fit the diagnostic criteria for TGA. The patient has had no further clinical events at over 4 years follow-up; the monophasic nature of the illness is in keeping with TGA and HSVE, but by definition, not epilepsy.

D.J.P. McCorry

References


\textit{doi}:10.1093/qjmed/hci060