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Letters to the Editor

Diagnosing Transient Global Amnesia Requires Exclusion of Alternative Differentials

To the Editor:

With interest, we read the article by Tso et al.[1] about a 62-year-old woman who experienced Takotsubo syndrome (TTS) supposedly triggered by multiple subacute stressors and associated with a short episode of unconsciousness that was interpreted as transient global amnesia (TGA). We have the following comments and concerns.

The main shortcoming of the report is that the diagnosis of TGA remains unproven. Missing is conventional magnetic resonance imaging of the brain to exclude ischemia, bleeding, mass lesion, venous thrombosis, or another structural lesion, and electroencephalography (EEG). Normal conventional magnetic resonance imaging and normal EEG results are prerequisites to diagnose TGA. If the standard EEG is non-informative, video EEG recordings should be carried out.

The patient had a history of 3 cerebral events. The first event manifested as an amnestic episode (anterograde and retrograde amnesia) shortly after awakening months before admission, being interpreted as TGA.[1] We should know how this TGA was diagnosed. The second event manifested as loss of consciousness for 2 to 3 minutes on the day of admission.[1] The third event manifested as unconsciousness related to polymorphic ventricular tachycardia and was not recalled by the patient.[1] Events 2 and 3 cannot be interpreted as TGA but rather as syncopes due to ventricular arrhythmia or a seizure.[2] In this regard, we also should be informed about the current medication the patient was regularly taking. This is because certain drugs are epileptogenic and arrhythmogenic.

Because the most frequent cerebral triggers of TTS are subarachnoid bleeding, seizures, cerebral ischemia, and intracerebral bleeding,[3] it is crucial that these conditions are excluded as the cause not only of TTS but also of polymorphic ventricular tachycardia. Ventricular tachycardia may be a manifestation of epilepsy.[4]

We do not agree with the notion in the "Discussion" that absence of "automatisms or other features of epilepsy" excludes seizures. There are certain seizure types (e.g., absences, nonconvulsive state) that manifest without clonic or tonic activity.

Missing in the report are the reference values for troponine and creatine kinase. Without knowing reference limits, pathogenicity of the provided values cannot be assessed.

Overall, the presented report has a number of shortcomings that need to be addressed before diagnosing TGA. Seizures need to be excluded as a trigger of TTS. Although TGA and TTS may occur concomitantly, a plausible trigger needs to be provided to explain the development of TTS.

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