

Transient Global Amnesia: A Bright Spot Not to be Missed

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Transient Global Amnesia (TGA) is a clinical disorder characterized by transient anterograde and retrograde amnesia with ambiguous pathogenesis [1]. It has also been associated with life-threatening medical conditions such as myocardial infarction arrhythmias or dissecting aortic aneurysm [2]. The diagnosis of this clinical syndrome is established when the clinical diagnostic criteria are fulfilled. The seven points of Hodges and Warlow's diagnostic criteria are the following: 1.) The information must be available from a trusted observer who was present during the maximum duration of the episode, 2.) The presence of anterograde amnesia must be clearly observed during the episode, 3.) It is necessary for the cognitive deficit to be limited only to amnesia in the absence of a.) Consciousness clouding and b.) Loss of recognition of personal identity, 4.) Absence of any focal neurologic sign and symptom, 5.) Epileptic phenomena should not be observed, 6.) Episodes must subside within 24 hours, 7.) All patients with a recent history of head injury or active epilepsy, defined by the use of antiepileptic medications or the presence of at least one episode of seizure in the last 2 years, are excluded from the above diagnosis [3,4].

The TGA must be differentially diagnosed by other amnesic syndromes or disorders that amnesia is a major clinical component [4].

Transient ischemic attack or stroke in the posterior cerebral circulation
Focal epileptic seizures, including transient epileptic amnesia
Postictal stage
Post-traumatic amnesia
Metabolic disorders, mainly hypoglycemia
Drug toxicosis or side effects
Limbic encephalitis
Migraine
Hypoxia
Psychogenic amnesia

Table 1: The main alternative diagnoses include.

The diagnosis of TGA is easy enough after a thorough history and performing neurological examination, when we focus on the fulfillment of the clinical diagnostic criteria [3,4]. When these criteria are not fulfilled, it must be investigated an alternative diagnosis. The combination of the medical history, the neurologic examination during clinical practice and the results of magnetic resonance imaging, electroencephalography and laboratory tests will establish the definite differential diagnose [1,3,6].

The significance of DWI sequence is crucial for the differential diagnosis of TGA. The diagnostic criteria for the diagnosis of TGA were first formulated by Caplan, taking into account the typical symptomatology of that clinical syndrome. Hodges and Warlow, and subsequently Quinette, et al. later certified these criteria, which have a great value in establishing the diagnosis of TGA against the most of the alternative diagnoses and especially the transient epileptic amnesia [4]. In the first days after the manifestation of the TGA, restriction in the DWI sequence can be found in at least one hippocampus, as high signal punctuate lesion. These lesions are transient and are located in the CA-1 section of the hippocampus [1,7]. The imaging findings of diffusion disturbance in the hippocampus are specific and sensitive enough to make the diagnosis of TGA in conjunction with the clinical diagnostic criteria. Approximately 84% of the patients who meet the diagnostic criteria have imaging findings in DWI. Disturbances of in DWI are more likely to be found between 36 and 48 hours after the initiation of the first clinical symptomatology, approximately 82-87% of the patients [7].

However, a review of the literature can reveal rare case reports of patients who met the diagnostic criteria of TGA whereas suffering of stroke outside of hippocampus. This could be explained by the functional connectivity of several brain areas with the hippocampus, resulting in an indirect impairment of the hippocampus function, when these areas are disturbed, generating the TGA symptomatology. For example, cases with the clinical diagnosis of TGA have been reported after a small left putamen infarction [8], thalamo-mesencephalon infarct [9], transient lesion of the anterior

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