

## Serial Diffusion-Weighted Imaging in Transient Global Amnesia?

Sir,

I read with great interest the article by Sivakumar and Indiran in which they illustrate a case with transient global amnesia (TGA) developing in the setting of bilateral hippocampal diffusion restriction.<sup>[1]</sup> I agree with the interesting aspect of this case; however, I would like to comment on the article for a better understanding of the report and provide some new perspectives.

TGA is one of the most mysterious syndromes in clinical neurology.<sup>[2]</sup> The involvement of hippocampal parenchyma in the pathophysiology of TGA is rather a validated view, supported by the recent reports.<sup>[2]</sup> On the other hand, although there are many hypotheses regarding the etiological mechanisms of TGA, there is still not a consensus at this point. In a crucial study by Sedlacek *et al.*, 31 consecutive patients were investigated by serial magnetic resonance imaging (MRI) examinations for 3 days from symptom onset.<sup>[2]</sup> In 26 patients, they found diffusion-restricted lesions in the hippocampal area which were all confirmed in T2-weighted images. They emphasized on the specific hippocampal localization corresponding to subcortical vascular borderzone and suggested that relative hypoperfusion might be responsible from diffusion restriction in this localization. In conclusion, they emphasized on a model of delayed ischemia as most possible mechanism of TGA. In the case by Sivakumar and Indiran, follow-up diffusion-weighted imaging (DWI), repeated after 10 days, had showed no abnormality.<sup>[1]</sup> DWI is a highly sensitive method for ischemic stroke but not specific and can occur due

to other mechanisms and origins such as neoplastic, infectious, toxic-metabolic, ictus-related changes. Hence, considering the controversies in this area, evaluation of serial imaging changes in diffusion-restricted regions may be a reasonable way of understanding the responsible mechanism. However, the authors did not make any explanation about the possible mechanism underlying this completely normalization of DWI abnormality?<sup>[1]</sup> For instance, in ischemia model, we would expect to see the effects of pseudonormalization (baseline apparent diffusion coefficient values and hyperintense DWI) in the 2<sup>nd</sup> week of the clinical presentation (corresponding to the time of second MRI). Technical problems may also be a possible explanation which might be clarified by a third MRI follow-up? I wonder if the authors may also include the T2-weighted images (if present) for a better evaluation of the underlying pathophysiology. I would also like to remark that the DWI sections, illustrated in the report (DWI at presentation and 10 days after), are not similar which complicates a proper interpretation of the lesion evolution. Of note, the proper demonstration of the follow-up image is also important to distinguish the initial lesions from possible DWI artifacts. Future reports of larger case series including follow-up imaging results may provide crucial contributions regarding the unknown aspects of TGA pathophysiology.

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### Conflicts of interest

There are no conflicts of interest.

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