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Tremor and Other Hyperkinetic Movements

In Response To:
Salazar R. Spontaneous intracranial hypotension associated with kinetic tremor and ataxia. Tremor Other Hyperkinet Mov. 2016; 6. doi: 10.7916/D8HQ3ZN5

Letters

Spontaneous Intracranial Hypotension and its Association with Movement Disorders?

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To the Editor:

I read with great interest the article by Salazar et al. in which the author described an interesting patient with bilateral kinetic hand tremor, gait ataxia and gait start hesitation.[1] Following clinical and laboratory investigations, he received a diagnosis of spontaneous intracranial hypotension (SIH) and associated his symptoms with that condition. In my opinion, this is a crucial case that is very rare in literature. However, I would like to comment on some aspects of the article, hoping to provide new perspectives about the underestimated issue of SIH and associated clinical manifestations of movement disorders.

First, a major limitation of this case may be that the site of the cerebrospinal fluid leak could not be determined, making it impossible to perform a target-oriented treatment (epidural blood patch). Due to the lack of an efficient therapy, it was not possible to comprehensively illustrate the patient's clinical presentations, as symptom recovery following adequate therapy provides valuable data for proper diagnosis.[2] For instance, the patient could have an underlying primary type I Chiari malformation, and new-onset SIH might have provoked findings of mild brainstem sagging.

Secondly, it was stated that the patient decided not to pursue any further work-up and only proceeded with conservative management with a partial response. I think that if the author had described which symptoms had resolved in more detail, the report would have provided a better understanding of the case and the mechanisms responsible for the patient's clinical manifestations. At this point, considering that movement disorder is a considerably uncommon component of the SIH clinical presentation, the underlying pathophysiology remains unclear.[3] In addition, follow-up neuroimaging in this patient might give important data about the clinical concerns regarding the responsible mechanisms. Furthermore, although the patient's mental examination was reportedly normal, other differential diagnoses such as Parkinson plus syndromes were not excluded. This is important as findings of tremor and gait abnormalities should lead to the consideration of degenerative processes in the foreground of differential diagnoses. I agree that neuroimaging findings and lumbar puncture pressure results supported the existence of SIH in this patient. However, the mechanisms of the occurrence of movement disorders in association with SIH can only currently be explained on a hypothetical level (mechanical factors such as nerve traction and cerebellum-brainstem compression).[3–5] Conversely,

these manifestations are accepted as cornerstone findings in Parkinson plus syndromes, as the underlying mechanisms have been repeatedly explained in the setting of degenerative processes. Taken together, I think that the rationale of attributing newly developed symptoms of tremor, gait start hesitation, and cerebellar function abnormality to SIH may be questionable.

In conclusion, I would like to draw attention to the issue that movement disorders associated with SIH have been reported only very rarely in a few case series.[3–5] For example, postural tremor was smartly reported in a unique report by Turgut et al. in a patient with an association with SIH (distinct from the patient of Salazar et al., who only had kinetic symptoms). The case described by Salazar et al. also adds very substantial contributions to this mysterious and unsolved issue. However, re-evaluation of some points in light of the comments in this letter will probably provide important perspectives to understanding these manifestations in SIH. Future larger case studies need to clarify the unknown aspects of SIH and related movement disorders. Furthermore, studies focusing on these mechanically explained and probably reversible symptoms can also provide an opportunity for another point of view to investigate movement disorder pathophysiologies and the responsible pathways.

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