Intracranial Hypotension Syndrome with Cortical Venous Thrombosis: Letter to the Article Entitled “A Rare Case”

Kortikal Venöz Tromboz ile Beraber İntrakraniyal Hipotansiyon Sendromu: Nadir Bir Olgu Adlı Makaleye İlişkin Mektup

Halil Önder
Yozgat State Hospital, Clinic of Neurology, Yozgat, Turkey

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Dear Editor,

I read with great interest the report by Jain et al., which smartly illustrated the clinical course of a rare patient diagnosed with intracranial hypotension (ICH) syndrome and concurrent cortical venous thrombosis (CVT) (1). I agree with the interesting aspect of this report and appreciate the authors for presenting such a detailed illustration of this patient. However, I would like to comment on this article hoping to provide some new perspectives to the article, as well as the issue of concurrent ICH and CVT.

First, an important point that may be discussed was that the patient had recovered only mildly following conservative treatments with intravenous fluids and steroids. However, anticoagulation treatment had yielded full improvement in the following course. In the literature, there is considerable evidence supporting the occurrence of CVT as a consequence of ICH (2,3,4,5); hence, treatment of solely ICH, as the inducer agent, was generally recommended (6). In a retrospective review by Schievink and Maya (2), ICH was indicated as a risk factor for CVT; however, there was no evidence for CVT preceding ICH in any patient. Thus, this report may give substantial perspectives revealing the role of anticoagulation among the treatment regimens of concurrent ICH and CVT.

However, there may be some limitations while evaluating the presentation of this case. First, the demonstration of recanalization of sinus stenosis in the follow-up using magnetic resonance venography (MRV) might clarify recanalization precisely, and support the rationale of anticoagulation therapy in physiologic aspects. The authors indicated that magnetic resonance imaging (MRI) performed 6 months later revealed a normal study, but an MRV recording was not mentioned in the follow-up. Second, it was not reported as to whether there were any abnormal findings such as empty delta sign (7), which would indicate sinus thrombosis in the first contrast enhancement MRI. Considering that the absence of a flow void and the presence of altered signal intensity in the sinus is a primary finding of sinus thrombosis on conventional MRI (7), conventional MRI findings of the patient have to be enlightened for an accurate diagnosis of the patient. For instance, another crucial diagnosis that might have caused non-visualisation of transverse sinus on MRV is sinus atresia in which conventional MRI does not give any abnormal appearance (7). On the other hand, if the first MRI was negative for CVT, then it can be considered that CVT had developed in the interval period between MRI and MRV recordings. However, a clinical deterioration following the first MRI was not mentioned, in contrary to this hypothesis. Inclusion of these results in the report would also provide information about the evolution time of thrombosis, which may provide substantial insight into the cause-effect relationship of ICH and CVT, as well as their impact seperately on clinical findings.

Address for Correspondence/Yazışma Adresi: Halil Önder MD, Yozgat State Hospital, Clinic of Neurology, Yozgat, Turkey
Phone: +90 512 305 15 80 E-mail: halilnder@yahoo.com

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The occurrence of CVT in patients with ICH has been reported rarely in the literature with a rate of only 2% (2). The cause-effect relationship between CVT and ICH has not been clarified clearly and remains as an interesting issue for further deliberations. Hence the smart illustration of this case constitutes a valuable report for physicians. However, I think that re-evaluation of some points mentioned in this report may add a better understanding and provide substantial perspectives for physicians. Future reports of larger numbers of patients with concurrent CVT and ICH need to clarify the underlying pathophysiology of this cooccurrence, as well as the unknown aspects of CSF dynamics.

Ethics

Peer-review: Internally peer-reviewed.
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References