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Cortical Venous Thrombosis in Intracranial Hypotension Syndrome Patient: Revisited

İntrakraniyal Hipotansiyon Sendromlu Hastada Kortikal Venöz Tromboz: Gözden Geçirme

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Dear Editor,

Halil Onder has nicely and critically reviewed the various aspects regarding intracranial hypotension (ICH) and cortical venous thrombosis (CVT) and has pointed towards some interesting points for further deliberations (1). I fully agree with the issues raised and will try to highlight these concerns.

A patient presented with fever and headache for 7 days. Based on characteristic magnetic resonance imaging (MRI) findings, prominent bilateral symmetric pachymeningeal enhancement with prominent engorged venous sinuses, prominent enhancement of the tentorium cerebelli, small ventricles, effaced sulci, prominent pituitary gland, and low pressure on cerebrospinal fluid (CSF) examination, a diagnosis of ICH syndrome was made and the patient was started on conservative treatment with intravenous fluids and steroids, but the patient showed only mild recovery. Further MR venography (MRV) showed non-visualization of the left transverse and sigmoid sinus, which was suggestive of CVT. The patient was started on anticoagulant treatment and showed complete clinical recovery. We agree fully with points raised by Halil Onder that follow-up MRV could have been given more strength to our diagnosis, but normal MRI performed at follow-up was normal and the patient improved considerably so we did not undertake further studies. As we mentioned, the patient was started on conventional treatment after MRI. MRV was considered when the patient did not show significant improvement despite treatment. Moreover, CVT might have developed during this interval. The underlying pathophysiologic process as described by the Monroe-Kelly doctrine or endothelial injury caused by stretching of venous sinuses due to decreased CSF buoyancy might hold true in our case (2,3,4). We stated in our case report that the cause-effect relationship of ICH and CVT has not yet been established and many hypotheses have been put forward, which are mentioned in the case report.

CVT occurs in about 2% of patients with spontaneous intracranial hypotension (SIH) and ICH is a known risk factor for CVT (5). There is considerable evidence supporting the occurrence of CVT as a consequence of ICH, as in our case. The use of anticoagulants in this scenario is not well established and therefore controversial. The rationale behind anticoagulation for CVT was to facilitate recanalization and to prevent further venous thrombosis. Further reporting of more cases with longer follow-up will further add to our knowledge of other hidden aspects of CSF dynamics and the pathophysiologic aspect of the presence of rare complications of rare diseases and effective management, particularly regarding the use of anti-coagulation in these patients. A high degree of suspicion leading to timely diagnosis and early management can reduce morbidity and financial burdens. However, it is pertinent to say the presence of SIH should not prevent the search for other thrombotic risk factors in the presence of CVT.

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