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Letter to the Editor

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Before Cerebral Venous Thrombosis Iis Attributed to Lumboperitoneal Shunt Overdrainage, Other Causes Must be Excluded

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LETTER TO THE EDITOR

We read with interest the article by Iimori, *et al*., about a 76-year-old man with idiopathic normal pressure hydrocephalus treated with a lumboperitoneal shunt reportedly complicated by bilateral subdural hematoma (SDH) and left cerebral venous thrombosis (CVT) [Iimori, T. *et al*., 2024]. Overdrainage of CSF was blamed for both the CVT and SDH [Iimori, T. *et al*., 2024]. The patient received intravenous heparinization followed by warfarin and surgical removal of the SDH [Iimori, T. *et al*., 2024]. The study is excellent but has limitations that are of concern and should be discussed.

First, we disagree with the statement that the index patient is the first reported case of cerebral venous thrombosis after lumboperitoneal shunt placement [Iimori, T. *et al*., 2024]. On the contrary, intracerebral hematoma, subarachnoid haemorrhage, and venous sinus thrombosis have been reported as a complication of lumboperitoneal shunt placement in a 40-year-old woman with idiopathic intracranial hypertension [Castillo, L. *et al*., 2008].

The second point is that differential causes of cerebral venous thrombosis have not been adequately considered and excluded [Iimori, T. *et al*., 2024]. Cerebral venous thrombosis is multicausal and can be caused by acquired or hereditary coagulopathy, thrombocytopathy, paraneoplastic, infectious (e.g. chronic sinus infections), traumatic or neoplastic causes [Ordieres-Ortega, L. *et al*., 2024]. Other causes of CVT that have not been adequately ruled out are heart disease, including atrial fibrillation, sickle cell anaemia, chemotherapy, meningitis, dehydration, heparin-induced thrombocytopenia, Behcet's disease, hypothyroidism or reversible cerebral vasoconstriction syndrome (RCVS) [Ordieres-Ortega, L. *et al*., 2024]. As the case possibly occurred during the pandemic, we should also know whether the patient was SARS-CoV-2 positive or negative at the time of CVT. This is important as it has been repeatedly reported that SARS-CoV-2 infection can be complicated by venous sinus thrombosis [Scutelnic, A. *et al*., 2024].

The third point is the discrepancy between the CT venography in Figure 2b and the cortical lesions on FLAIR in Figure 2c and T2 in Figure 2d [Iimori, T. *et al*., 2024]. Since CT venography shows a lack of filling in almost the entire left hemisphere venous bed, one would expect a much more extensive lesion on FLAIR and T2 and a much more intense clinical deficit. This discrepancy should be clarified.

The fourth issue is that shunt function on readmission was not reported, and there is no mention of whether CSF pressure was actually reduced on a second tap test on readmission as suspected [Iimori, T. *et al*., 2024]. During the initial hospitalization, the index patient had received a Codman-Certas plus programmable valve (CSF shunt valve, Integra LifeSciences Holdings Corporation, Plainsboro Township, New Zealand), and the pressure was initially set at 400 mmH_2O to prevent overdrainage in the early postoperative period [Iimori, T. *et al*., 2024]. Was the shunt still set to this value on re-admission or was it set to a lower value?

The fifth point is that comorbidities and concomitant medications of the index patient were not specified [Iimori, T. *et al*., 2024]. Since he was 76 years old, it is conceivable that he suffered not only from normal pressure hydrocephalus but also from other diseases. Various comorbidities and concomitant medications may be complicated by cerebral thrombosis or haemorrhage.

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In conclusion, this interesting study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and support the study's message. Before lumboperitoneal shunt overdrainage is considered to be the cause of cerebral venous thrombosis, all possible differential causes of cerebral venous thrombosis must be excluded with certainty.

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