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Commentary

Abnormal Temporal Cerebral Autoregulation in Cerebral Venous Thrombosis

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DESCRIPTION

In traumatic brain injury, cerebral auto regulation is important and may be utilized to determine the ideal intracranial pressure. High intracranial pressure is a characteristic of cerebral venous thrombosis, a cerebral vascular illness for which the auto regulatory mechanism is still unknown. Goal was to learn more about how well people with cerebral venous thrombosis can regulate their brains. In this study, patients with CVT showed a marked reduction in phase shift and RoRc of transfer function analyses, indicating that the cerebral auto regulation was compromised by CVT. There were 16 controls and 23 cases with cerebral venous thrombosis. Transfer function analysis (rate of recovery/phase/gain) from spontaneous oscillations of cerebral blood flow velocity and arterial blood pressure was used to evaluate cerebral auto regulation.

Increased intracranial pressure, venous cerebral infarction, and bleeding, which harm cerebral arterioles and capillaries, can occur in CVT patients due to venous blood drainage blockage and impaired cerebrospinal fluid absorption. While complicated myogenic, metabolic, and neurogenic systems controlled cerebral auto regulation, the cerebral arterioles and capillaries served as its mere effector. And this may be the cause of the deficit in brain auto regulation in CVT patients. TCD was used to quantify the cerebral blood flow velocity (CBFV) in the bilateral Middle Cerebral Arteries (MCA) (DWL, Germany). Using a servo-controlled finger plethysmograph, the Arterial Blood Pressure (ABP) was continuously measured from beat to beat (AD instruments, Australia). After the baseline value stabilized, the CBFV and ABP data were captured for at least 5 minutes while the individuals freely breathed in a supine position at a sampling rate of 100 Hz.

44.7% of the hemispheres in this study were found to have brain parenchymal lesions, including cerebral ischemia, haemorrhage, and subarachnoid haemorrhage, all of which have been shown to be capable of reducing auto regulation. However, based on these findings, brain auto regulation deteriorates even in hemispheres free of lesions and hemispheres with and without lesions did not differ from one another. Furthermore, even in the hemispheres free of significant sinus stenosis and cerebral parenchymal lesions, the bilateral auto regulation impairment was seen. It could support the idea that patients with CVT have impaired brain auto regulation. The auto regulatory capacity was clearly decreased in patients with CVT, according to the current study. Therefore, it is important to carefully maintain blood pressure in CVT patients because cerebral hemodynamics may become "venerable" to ABP fluctuations. Increases in ABP could lead to hyper-perfusion, which could then increase intracranial pressure, which, in turn, could weaken auto regulatory ability and lead to a vicious cycle. The decrease in ABP may cause cerebral ischemia and hypo perfusion. Therefore, evaluation of cerebral hemodynamics in CVT patients should be taken into account, particularly for the direction of hypertensive medication. Numerous investigations showed that individuals with stroke, subarachnoid haemorrhage, traumatic brain injury, carotid or intracranial stenosis, and poor cerebral auto regulation had adverse neurologic outcomes on their own. The best cerebral perfusion pressure for traumatic brain injury patients can be found by evaluating cerebral auto regulation, which can then be utilized to alter cerebral perfusion pressure for a positive outcome. For better blood pressure control in CVT patients, we anticipate new methods for assessing auto regulation to determine the ideal blood pressure. This research reveals that people with CVT have impaired auto regulation. Further research is required to determine whether auto regulation may have a therapeutic benefit in the treatment of patients with CVT.

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