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Opinion Article

Neurological Venous Thrombosis: Impaired Dynamic Cognitive Automatic Regulation

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DESCRIPTION

The relatively uncommon cerebrovascular illness known as cerebral venous sinus thrombosis typically affects young people. The diagnosis and treatment of CVT can be challenging due to its widely diverse clinical manifestations, which might include headache, visual impairment, seizures, encephalopathy, and localized neurological abnormalities. Despite the wide range of clinical manifestations, increased intracranial pressure, venous blood drainage blockage, and reduced absorption of cerebrospinal fluid are all contributing factors in the pathophysiology of CVT. Numerous investigations shown that in patients with subarachnoid hemorrhage and traumatic brain damage, intracranial hypertension may hence disrupt the cerebral autoregulation mechanism. Furthermore, a meta-analysis that examined sixteen pertinent researchers found that patients with intracranial hypertension were more likely to have poor autoregulation. While autoregulation plays a crucial role in keeping the blood flow to the brain largely constant despite changes in blood pressure. Brain hyper perfusion or hypo perfusion may result from impaired cerebral autoregulation, which can also cause passive changes in cerebral blood flow in response to blood pressure variations. Increased intracranial pressure, venous cerebral infarction, and hemorrhage can result in cerebral arterioles and capillaries being damaged in CVT patients due to venous blood drainage blockage and impaired absorption of cerebrospinal fluid. Through intricate myogenic, metabolic, and neurogenic processes, cerebral autoregulation was just mediated by the cerebral arterioles and capillaries. And for that reason, people with CVT may have impaired brain autoregulation. The current investigation found that patients with CVT have a marked impairment in their auto regulatory capacity. As a result, blood pressure needs to be closely monitored in CVT patients because ABP fluctuations may cause cerebral hemodynamics to become "venerable." Elevations in ABP may lead to hyper perfusion, which may worsen the auto regulatory capacity and increase intracranial pressure. This vicious cycle can continue. On the other hand, cerebral ischemia and hypo perfusion may be brought on by an ABP decline. Consequently, it is important to evaluate cerebral hemodynamics in individuals with CVT, particularly when directing hypertensive treatment. Numerous studies have shown that in patients with stroke, subarachnoid hemorrhage, traumatic brain injury, and carotid or intracranial stenosis, decreased cerebral autoregulation is an independent predictor of poor neurologic outcomes. Recent research has also demonstrated that in patients with traumatic brain injury, cerebral autoregulation assessment can be used to identify the ideal cerebral perfusion pressure, which can then be adjusted for the best possible result. We anticipate the development of new models in the future that will allow us to analyze autoregulation to determine the ideal blood pressure, improving blood pressure control in CVT patients. Using TCD, the Cerebral Blood Flow Velocity (CBFV) in each bilateral Middle Cerebral Artery (MCA) was determined. A finger plethysmograph with servo control was used to record the Arterial Blood Pressure (ABP) continuously beat-to-beat. After the baseline value stabilized, participants spontaneous breathing in a supine position was monitored for at least five minutes while CBFV and ABP data were collected at a sampling rate of 100 Hz. Using transfer function analysis, cerebral autoregulation was assessed. Auto regulatory parameters (phase shift, gain, and coherence function) were computed in the frequency domain between 0.06 and 0.12 Hz. The step response was computed in the temporal domain to demonstrate the step-wise recovery of CBFV following ABP alterations. In order to quantify the rate of recovery, the rate of recovery of CBFV, or the first three seconds of the step response, was computed.

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