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Short Communication

Clinical Outcomes in Patients with Cerebral Venous Thrombosis

Sarah Palareti*

Department of Angiology and Blood Coagulation, University of Bologna, Bologna, Italy

DESCRIPTION

Cerebral Venous Thrombosis (CVT) can mimic other acute neuropathy and can only be detected in optimal and timely brain images, often overlooking or delaying diagnosis. CVT was found in 9.3% of consecutive autopsy series. This suggests that CVTs are often overlooked in life. CVTs generally have a good prognosis when diagnosed and treated early. Anticoagulant therapy with parenteral heparin is the mainstream of acute treatment, but patients whose condition worsens despite treatment are eligible for intravascular treatment like intravascular thrombolysis or thrombolysis, or neurosurgery decompressive craniotomy [1].

CVTs account for 0.5%-1.0% of unselected stroke hospitalizations and are probably associated with pregnancy, childbirth, and the use of estrogen-containing oral contraceptives and are found in women about three times as often as men.

Blood flows from brain through the small cerebral veins into the larger veins of the deep vein system. This includes the inner cerebrum, basal ganglia (von Rosenthal), and veins of the Galen vein. It then flows into the hard sinus including straight sinus and transverse sinus, and the sagittal sinus; these flow primarily into the internal jugular vein. Blood stasis, abnormalities in the walls of blood vessels, and changes in blood composition (virchow's triad) lead to an imbalance between the thrombus-promoting process and the fibrinolytic process, predisposing to progressive venous thrombosis. Occlusion of venous vessels leads to increased venous pressure, decreased capillary perfusion, and increased local cerebral blood volume. Initially compensated for by dilation of the cerebral veins and mobilization of collateral vessels, the continued increase in venous pressure was due to angiogenic edema due to disruption of the blood-brain barrier and blood with cerebral perfusion pressure and tissue infarction. It can cause a drop in flow. Therefore, both cytotoxic edema and angiogenic edema can occur [2,3].

The venous region is less clearly defined than the arterial region due to the extensive anastomosis between cortical veins that allows the development of alternative venous drainage pathways after occlusion. CVTs can also block CSF absorption by arachnoid villi. These are increase intracranial pressure with or without tissue damage and is usually associated with superior sagittal sinus obstruction.

The cerebral venous system is divided into superficial and deep venous systems. They are a group of venous channels located intracranial between the endosteal and meningeal layers of the dura. Unlike systemic veins, cerebral veins have no valves and do not follow the area of the cerebral artery. In particular, the superior sagittal sinus also drains cerebrospinal fluid from the subarachnoid space [4].

Superficial systems include the dural venous sinus and cortical veins. It drains the cerebral cortex and superficial white matter. The two major dural venous sinuses include the superior sagittal sinus, which drains the dorsolateral region, and the cavernous sinus, which drains the anterior ventral region. The superior sagittal sinus flows into the transverse sinus, and the transverse sinus flows into the straight sinus. The cavernous sinus is drained posterolaterally into the transverse sinus and inferior-laterally into the sigmoid sinus along the inferior petrosal sinus. The superficial cortical veins are the superior and inferior cerebral veins such as Labbe and Sylvian veins or superficial middle cerebral veins.

The deep system includes drainage of straight, lateral, and S-shaped sinuses, as well as deeper cortical veins (venous von Galen, internal cerebral vein, Rosenthal or basal vein, medulla and subependymal veins). These blood vessels drain the basal ganglia, thalamus, upper brain stem, and deep white matter of the brain. Both the superficial and deep vein systems eventually flow into the internal jugular vein [5,6].

Cerebral veins and dural venous thrombosis are less common than most other types of stroke, but can be more difficult to diagnose. With the widespread use of Magnetic Resonance Imaging (MRI) and growing clinical awareness, CVTs are becoming more and more frequently recognized. In addition, it is now known to have a more diverse clinical spectrum than previously thought. Due to its myriad causes and symptoms, CVT meets not only neurologists and neurosurgeons, but also

Correspondence to: Sarah Palareti, Department of Angiology and Blood Coagulation, University of Bologna, Bologna, Italy, E-mail: palareti_sarah@unibo.it

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emergency medicine specialists, internists, oncologists, hematologists, gynecologists, pediatricians, and general practitioners.

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