Cerebral Venous Sinus Thrombosis

Definition
Cerebral venous sinus thrombosis (CVST) is simply a thrombosis of the intracranial veins and sinuses.

Epidemiology
Cerebral venous sinus thrombosis accounts for less than 1% of strokes. The incidence of CVST is approximately 5 per million people annually. CVST is more common in young to middle aged persons, with a female to male ratio of 2:1.

Pathophysiology
Causes of CVST can be multifactorial. Most of the causes can be linked to Virchow’s triad for thrombosis formation, which include stasis of blood flow, changes in the vessel wall, and changes in the composition of blood. Approximately 20% of patients, the cause or risk of CVST is unable to be identified.

Alteration of blood composition could be related to the use of oral contraceptives, hematologic disorders, systemic malignancy, trauma, dehydration, postpartum state or infection. Genetic prothrombotic tendencies that increase the risk of CVST include factor V Leiden mutation, deficiencies of protein S and C, as well as antithrombin III.

Cerebral venous sinus thrombosis usually starts in the large venous sinuses. The most common locations for CVST to occur are the superior sagittal sinus (88%) and straight sinus (29%). The mortality rate of CVST is 4.39%.

Manifestations
Manifestations of CVST vary depending on the extension and location of the thrombosis, and the presence of venous collaterals. The most common manifestations of patients with CVST are headache (95%), seizure (47%), altered level of consciousness (54%), fever, nausea and vomiting. Forty to sixty percent of patients presented with some focal neurological signs, including motor or sensory deficits, aphasia, and hemianopsia.

A headache is usually the first presentation and may present weeks to months before the other signs and symptoms occur. The headache usually has a subacute onset, but it can be severe and globalized.

Patients who develop increased ICP may present with headache, papilledema, and diplopia if the 6th cranial nerve is involved. If the ICP is significantly elevated, transtentorial herniation may result, causing the patients to be comatose with or without pupillary dilatation.

Diagnostic Tests
A cerebral angiogram is the gold standard for establishing diagnosis of CVST. However, a cerebral angiogram is invasive and has several complications. With advances in diagnostic technology, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are commonly used to diagnose neurological and neurosurgical diseases. A cerebral angiogram may be used when CT scans or MRI are unable to make the diagnosis or if an endovascular procedure is required.

With the enhancement of cerebral blood vessels by the radio-contrast in CT angiography and CT venography, a diagnosis of CVST can be established. The primary sign of acute CVST on a noncontrast CT is hyperdensity of a cortical vein or sinus. Thrombosis of the posterior portion of the superior sagittal sinus appears as a dense triangle called the “delta sign.”

An MRI is more sensitive in detecting CVST than a CT scan. Non-contrast-enhanced MRIs can show absence of blood flow in the dural sinus. Same as a CT scan, an MRI may show cerebral swelling, and/or hemorrhaging related to CVST. Cerebral MR venography (MRV) is noninvasive and is able to provide visualization of the cerebral venous sinuses.

Laboratory tests such as completed blood count (CBC), erythrocyte sedimentation rate (ERS), prothrombin time (PT), activated partial thromboplastin time (aPTT), and D-dimer should be performed to identify any underlying causes for CVST such as a hypercoagulable state, or infection. Most patients with recent CVST have increased D-dimer concentration. However, the accuracy of using a D-dimer test to diagnose CVST is controversial and using D-dimer to exclude CVST is not recommended.

Treatment Options
The goals of treatment for CVST are to reverse obstruction, re-establish venous return, and to reduce the risk of venous hypertension, cerebral infarction, and pulmonary embolism. The standard treatment for CVST is anticoagulation with intravenous infusion of heparin or a subcutaneous injection of low molecular weight heparin.
Anticoagulant therapy can prevent thrombus extension while the endogenous anti-clotting factors resolve the thrombus. Infusions of unfractionated heparin and subcutaneous injections of low molecular weight heparin have been used. The weight-adjusted subcutaneous injection of low molecular weight heparin is recommended because it has a lower risk of bleeding complications compared with the unfractionated heparin.

In the ISCVT study, 39.3% of CVST patients have intracerebral hemorrhage. Some researchers argue that the use of anticoagulants to treat CVST when patients have concurrent intracranial hemorrhage may increase bleeding. However, research indicates that the use of heparin in CVST patients concomitant with intracranial hemorrhage has not been shown to increase the size of a hemorrhage. CVST is related to the intracranial hemorrhage and is not due to ruptured blood vessels. A heparin infusion prevents the re-oclusion of recanalized blood vessels and reduces capillary pressure, which prevents further outward movement of red blood cells and limits the size of the hemorrhage.

Patients with CVST who deteriorate despite anticoagulant therapy, systemic or local administration of fibrinolytic agents such as recombinant tissue plasminogen activator (rtPA) have been tried to recanalize CVST. Fibrinolytic agents are faster in dissolving a thrombus and re-establishing venous blood flow to prevent further brain damage. Local thrombolyis with an injection of a fibrinolytic agent directly into the thrombus via a transfemoral microcatheter has been performed. The high risk of bleeding complications and vessel dissection, endovascular thrombolytic therapy should be reserved for patients with poor prognosis.

Mechanical thrombectomy is done to remove the clot or break down the clot into smaller pieces by using a mechanical device such as a Merci or penumbra device. A mechanical thrombectomy can be a stand along intervention, but usually is used as a combined therapy together with the administration of fibrinolytic agents. Mechanical thrombectomy is not the first line treatment, and is usually reserved for patients who experience clinical deterioration.

Patients who are male, >37 year-old, comatose, with intracerebral hemorrhage on admission, and increased intracranial pressure (ICP) have a higher mortality rate. Patients with high ICP and transtenorital herniation, decompression hemicrianiotomy has been performed to reduce ICP and remove the immediate threat of cerebral damage.

Nursing Implications

Approximately 50% of patients experience depression or anxiety post CVST. It is important to reassure patients that the recurrence rate of CVST is very low. Patient should gradually resume their normal activities.

Patients may continue with anticoagulation for 6-12 months post discharge, with regular assessment of their clotting profile to maintain the international normalized ratio (INR) between two to three times the normal limit. Patient should monitor for any signs of bleeding while on anticoagulant therapy.

For patients with pregnancy related CVST; the risk of complications for new pregnancy is low. However, women should not become pregnant when they are on coumadin therapy because of its teratogenic effects.

Reference


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