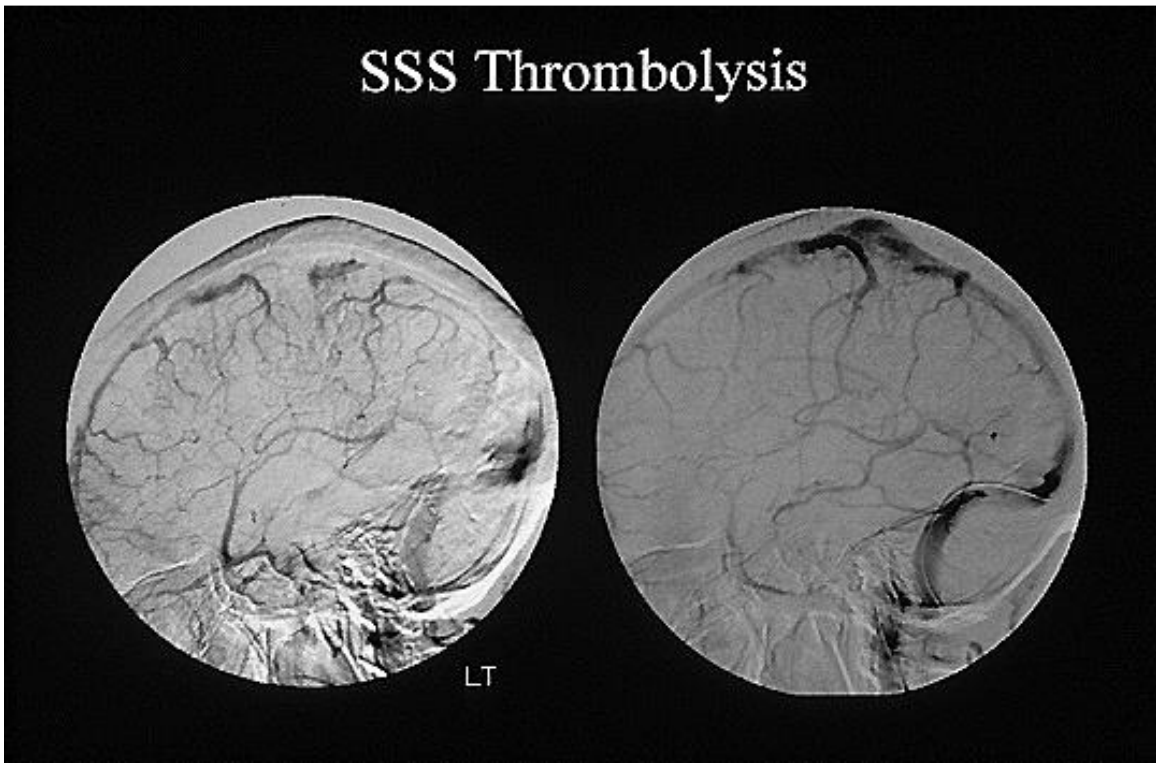
VENOUS SINUS THROMBOSIS: NEWSLETTER

Case Report:

A 32 year old right handed man presented 48 hours with bilateral headache and lethargy. The patient had a history of working outdoors in extreme heat over the previous days. A head CT revealed a Superior Sagittal Sinus (SSS) thrombosis and a small left occipital subcortical hemorrhage. The patient was started on IV heparin (INR goal 2.0). A cerebral MRI/MRA confirmed the CT findings. Catheter angiography showed the SSS thrombosis involving the anterior and posterior SSS. Using right common femoral vein access a microcatheter with multiple side holes (aka: spray catheter) was advanced through the thrombus with its tip positioned at the level of the coronal suture. Over the next 3 days, 3 million units of urokinase were infused into the thrombosed SSS. The SSS gradually recanalized after 48 hours of treatment and the patient was discharged on 6 months of Warfarin therapy without deficits. Etiology for the patient's SSS thrombosis was severe dehydration. Hypercoagulability studies were negative.

Figure 1: The left sided image shows no opacification of the SSS due to thrombosis. The right sided image shows patency of the SSS after two days of urokinase infusion.



Cerebral Dural Sinus Thrombosis:

Dural Sinus Thrombosis (SSS) was first described in 1825 in a 45 year old man with malignancy, headache, seizures and delirium. This diagnosis was confirmed by autopsy (Manual of diseases of the nervous system. 2nd ed. London: Churchill, 1888:416). Additional cases were reported in 1888, 1915, 1936 and 1940. Since 1940 over 200 cases have been described in the literature. While this number is small it is likely that SSS and many other cases of dural and venous sinus thrombosis have gone undiagnosed or misdiagnosed due to lack of suspicion and investigation.

Factors associated with sinus thrombosis and venous thrombosis include hypercoagulability during the puerperal period, infections, head trauma, synthetic steroids, hypercoagulability, and autoimmune inflammatory diseases. In warmer climates, dehydration can lead to increased blood viscocity and a hypercoagulable state.

Diagnosis of SSS and other venous sinus syndromes are made using CT, MRI, MRA and catheter angiography. Findings include absence of sinus opacification, sinus filling defects, dilated cortical veins and collateral veins with a corkscrew appearance, increased cerebral transit time due to venous hypertension and possibly reversal of flow away from the obstructed sinus or vein (Neurol Clin. 10:87-111, 1992; Radiology. 98:9-22, 1971). When interpreting the imaging studies, physicians must be aware that in the anterior SSS and one of the Transverse Sinuses (TS) and one of the Sigmoid Sinuses (SS)may normally not opacify. Not being aware of such normal anatomy can lead to false diagnosis. While 10-20% of CT scans may be normal in patients with venous sinus thrombosis, findings can include hemorrhagic and non-hemorrhagic venous infarcts, small ventricles, large ventricles, intense contrast enhancement of the falx and tentorium, thrombosed veins and sinuses, a dense triangle representing fresh thrombus in the posterior SSS and an empty delta sign in the torcula due to enhancement of collateral veins in the SSS wall surrounding a non-enhancing thrombus.

Signs and symptoms of intracranial sinus thrombosis (sagittal, transverse and/or sigmoid sinuses) can include headache, altered mental status, focal neurologic deficits and seizures. Many of the abnormalities may be related to increased intracranial pressure due to decreased cerebrospinal fluid absorption secondary to venous hypertension. Other symptoms are related to intracranial hemorrhage. A forgotten but reported complication of sinus thrombosis includes pulmonary embolism of which 23 cases were reported between 1942 and 1990 (Acta Neurol Scand 86:390-396, 1992).

Once sinus thrombosis is diagnosed, first line therapy is anticoagulation using IV heparin. Depending upon the patient's neurologic condition, more aggressive treatment can include cerebral dehydrating agents (mannitol, urea, hypertonic saline to reduce intracranial pressure, acetazolamide (Daimox) to reduce CSF production, CSF drainage using an external ventricular drain to reduce intracranial

pressure, barbiturates to reduce cerebral metabolism and cerebral blood flow which can reduce intracranial pressure, endovascular thrombectomy using mechanical aspiration devices (Penumbra, Penumbra Inc., Alameda, CA), and microcatheter superselective thrombolysis using urokinase or tissue plasminogen activator (Alteplase, Genentech, San Francisco, CA).

While mortality rates for untreated sinus thrombosis has been reported to be as high as 70%, more recent reviews show rates closer to 5-30% (Neurol Clin. 10:87-111, 1992). In survivors, 15-25% of individuals suffer sequelae. The only randomized, prospective study looking at heparin treatment vs. untreated natural history was carried out by Einhaupl (Lancet. 38:597-600. 1991). At 3 months follow-up, 80% of heparin treated patients were normal and 20% had slight deficits while 10% of the non-heparinized patients were normal, 60% had neurologic deficits and 30% were dead. These authors concluded that heparin therapy for symptomatic sinus thrombosis was safe, effective and beneficial even in the setting of intracerebral venous hemorrhage. No prospective randomized studies comparing heparin therapy to endovascular therapy have been conducted. Nevertheless, such intervention shows very low complication rates with very high recanalization rates especially when thrombus was less than 72 hours old. Using studies on venous thrombus elsewhere in the body, lesions older than two weeks seem to be more difficult to recanalize while those older than six weeks generally show no appreciable change after therapy (Am Heart J. 76:628. 1968).

Conclusion:

Intracranial venous sinus thrombosis should be considered in patients with headache who carry associated risk factors such as dehydration, oral contraceptive use, pregnancy or recent delivery, period, malignancy, and hypercoagulability. Early identification using CT, MR and catheter angiography permits early intervention using IV heparin and endovascular therapy when warranted. These interventions are documented to improve outcomes in randomized and non-randomized studies.