

Severe headache for 5 weeks

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A 39-year-old woman presented to the emergency department because of severe recurrent headache for 5 weeks.

There was no history of a significant previous illness. Imaging studies are shown below (Figures 1–4).

For diagnosis and discussion, see the following page.

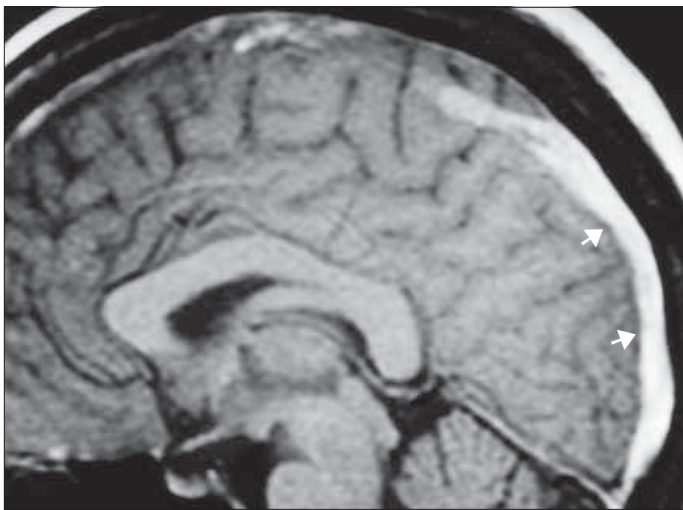


Figure 1. Sagittal T1-weighted magnetic resonance (MR) image shows a high-signal-intensity mass in the superior sagittal sinus (arrows).

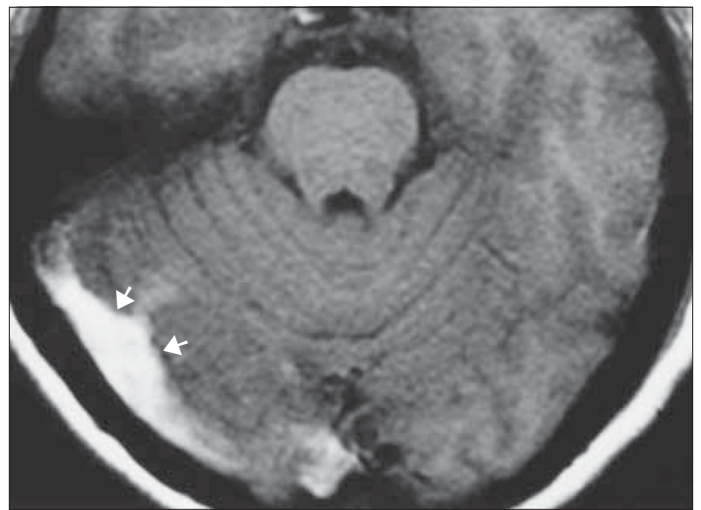


Figure 2. Axial T1-weighted MR image demonstrates a high-signal-intensity mass (arrows) in the right transverse sinus.

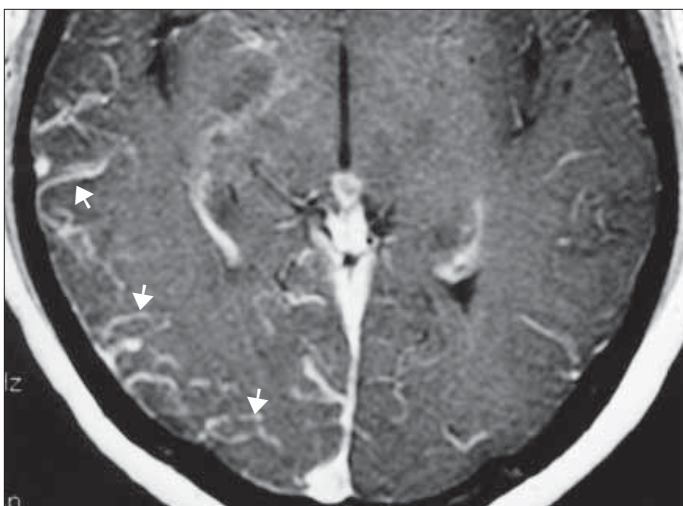


Figure 3. Axial postinfusion T1-weighted MR image shows venous collaterals (arrows) in the right occipital lobe.

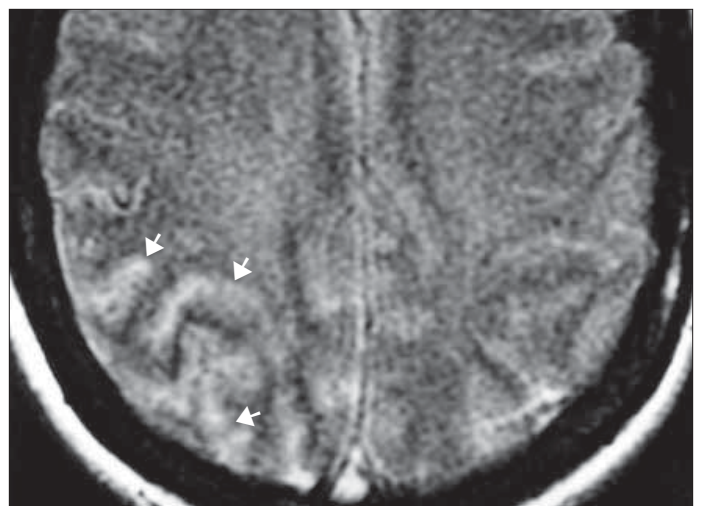


Figure 4. Axial fluid-attenuated inversion recovery (FLAIR) MR image demonstrates edema (arrows) in the right occipital lobe.

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DIAGNOSIS: Dural sinus thrombosis.

DISCUSSION

Dural sinus thrombosis (DST), also known as *cerebral venous thrombosis*, describes thrombosis of cortical and deep cerebral veins as well as that of the dural sinuses. DST is relatively rare (the cause of 1% of acute strokes) and has a nonspecific clinical presentation; thus, it is frequently a delayed or missed diagnosis. A high level of clinical suspicion and proper imaging techniques are critical for prompt diagnosis and treatment. The site of thrombosis is variable, with the superior sagittal sinus most commonly involved, followed by the transverse, sigmoid, and cavernous sinuses (1).

DST results from a multistep process. Virchow's triad of venous thrombosis includes endothelial injury, venous stasis, and hypercoagulability; any or all of these may be present (2). DST usually begins with a partially occlusive thrombus within the dural sinus that progresses to obstruct the involved dural sinus and then extends to bridging cortical veins. Increased venous pressure results, with breakdown of the blood-brain barrier, vasogenic edema, and then hemorrhage. Up to 50% of cases of DST progress to venous infarction. Venous infarctions are frequently bilateral, parasagittal, and hemorrhagic. Petechial perivascular hemorrhages may also be seen with cortical venous infarcts. If thrombosis and occlusion of the straight sinus occurs, bilateral thalamic infarcts may result.

Although numerous systemic diseases and conditions predispose to DST (>100 have been described), approximately 25% to 40% of cases occur with no identifiable cause (1, 2). Common predisposing conditions include dehydration, trauma, pregnancy, oral contraceptive use (present in the current case), coagulopathies (protein C and S deficiency and factor V Leiden deficiency most commonly), and association with arteriovenous malformations or arteriovenous fistulas. Local infection (sinusitis, otitis, meningitis) remains a common cause of DST through direct spread of infection and alterations in the coagulation pathway. Because of more effective antibiotics, infection has decreased as a cause in past years. Local or invading tumors, particularly meningiomas, can also be associated with DST. Hematologic illnesses, particularly leukemia and sickle cell disease, have been reported to cause DST (1). Systemic diseases such as vasculitis, thyrotoxicosis, or inflammatory bowel disease are also reported but are less commonly associated with DST. All age groups and both sexes can be affected; however, the disorder is more common in young women and aging adults.

Although the clinical presentation of DST is variable, headache is the most common symptom and is believed to be secondary to increased intracranial pressure. Nausea, vomiting, visual changes, focal neurologic deficits, and seizures are other symptoms associated with DST. When cavernous sinus thrombosis is present, eye pain, ophthalmoplegia, pupillary changes, and retinal hemorrhages may occur. Intraparenchymal hemorrhage and infarcts are more serious presenting clinical findings. If DST is secondary to central nervous system, ear, nose, or throat infections, alterations in mental status and fever are commonly re-

ported. The clinical presentation may vary from asymptomatic to coma or profound neurologic deficit.

Although computed tomography (CT) is usually the first diagnostic imaging examination performed in patients with suspected DST, MR imaging is the preferred modality for diagnosis of the disorder. MR venography is especially useful. With MR techniques, the clot can be directly visualized, the extent more accurately characterized, and associated cerebral ischemia or hemorrhage more completely evaluated. MR signal characteristics vary with the age of the thrombus, with the acute clot appearing isointense when compared with normal brain on T1-weighted images and hypointense on T2-weighted images. In the subacute stage, the signal is hyperintense compared with that of normal brain parenchyma on both types of imaging sequences. Associated cerebral edema will typically be hyperintense on FLAIR and T2-weighted images. When DST has progressed to venous infarction, contrast enhancement of the infarcted parenchyma can be seen secondary to blood-brain barrier breakdown, which may mimic an enhancing mass. The differential diagnosis includes giant arachnoid granulations (mimicking a filling defect in the sinus) and a congenitally hypoplastic transverse sinus. Although conventional cerebral angiography was the standard in diagnosing DST in years past, it is now indicated only when MR imaging is nondiagnostic or when intervention is desired.

Management of DST involves 1) a diagnostic workup to determine any underlying conditions or causes, such as infection or a mass, 2) aggressive treatment of the cause, and 3) supportive care for associated symptoms. In cases of septic thrombus that is associated with central nervous system, ear, nose, or throat infections, antibiotic therapy is the mainstay of treatment. Treatment with antithrombotics such as heparin is controversial (3, 4).

More recently, interventional neuroradiologic techniques have been used to deliver thrombolytic agents directly to the clot. Reported series are small, but the results are promising. Most authors describe the following basic endovascular treatment technique. Cerebral venography is performed to determine the extent of venoocclusive disease. Afterward, a microcatheter is maneuvered into the dural sinus system and, ideally, is placed across the clot. Unlike systemic delivery, this method allows the thrombolytic agent to be infused directly into the clot. The microcatheter is then left in place for continuous infusion of the thrombolytic agent. After approximately 18 to 24 hours, the patient is reexamined with venography to determine if the sinus is patent or if repeat direct thrombolysis is needed (2). Initial results of endovascular treatment have been encouraging, with little morbidity reported in stable candidates.

1. Osborn AG. *Diagnostic Neuroradiology*. St. Louis: Mosby-Year Book Inc, 1994:145-150, 238.
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3. Volturo GA, Repetra RJ Jr. Non-lower extremity deep venous thrombosis. *Emerg Med Clin North Am* 2001;19:877-893.
4. Einhaupl KM, Villringer A, Meister W, Mehraein S, Garner C, Pellkofer M, Haberl RL, Pfister HW, Schmiedek P. Heparin treatment in sinus venous thrombosis. *Lancet* 1991;338:597-600.