Thrombosis in a Congenitally Bifurcated Superior Sagittal Sinus

Mark A. Hosley, MD, PhD; Marc Fisher, MD; and James F. Lingley, MD

A 26-year-old woman had a postpartum venous thrombotic stroke involving the right parietal lobe. The initial thrombus was present only in the right channel of a congenitally bifurcated superior sagittal sinus. This diagnosis and subsequent thrombus extension were readily shown by magnetic resonance imaging in contrast to equivocal angiography. A subsequent, prospective review of 100 patients undergoing cranial magnetic resonance imaging showed the presence of similarly bifurcated superior sagittal sinuses in two. The patient stabilized after therapy with intravenous heparin, but switching her medication to oral warfarin sodium was followed by clinical deterioration and propagation of the thrombus, necessitating resumption of intravenous heparin. No coagulopathy was identified. (Stroke 1991;22:396–400)
of a skin rash, her medication was changed to 250 mg valproic acid p.o. q.i.d. without complications. An MRI scan of the head, including gradient recalled (GRASS) images, showed a right parietal infarct and bifurcation of the superior sagittal sinus over 6–8 cm (Figure 2). The thrombosed right channel was
hyperintense, while the open left channel was hypointense due to its flow void on T2-weighted images. The signal intensities were reversed on the GRASS images, confirming this interpretation. Anticoagulant therapy was deferred because of spontaneous improvement in the patient’s condition, with resolving headache, left hemiparesis, and left hemianopsia.

Subsequently the patient improved, but on hospital day 9 her headache recurred accompanied by photophobia, nausea, and vomiting despite an unchanged neurological examination. On hospital day 10, a repeat MRI scan showed thrombosis of both channels of the bifurcated superior sagittal sinus, with extension into the right transverse sinus, and a small intraparenchymal hemorrhage at the site of the initial infarct (Figure 3).

Intravenous heparin therapy was then initiated, maintaining the partial thromboplastin time (PTT) at 2–2.5×control. Oral warfarin sodium therapy was begun on hospital day 14, and the heparin infusion was discontinued on hospital day 18. The patient’s headache resolved, she was free of seizures, and she had only a trace of left hemiparesis. She was discharged home on hospital day 19, with a prothrombin time (PT) of 18/11–13 seconds (patient value/control value).

Five days after discharge, the patient had another relapse of headache. Repeat MRI scan showed right-sided extension of the thrombus along the transverse sinus and sigmoid sinus and into the jugular bulb. Her PT was 19/11–13 seconds at this time. She was readmitted to the hospital, intravenous heparin therapy was resumed, and intravenous vitamin K was given to reverse the effects of warfarin sodium. Her headaches resolved, and the neurological examination demonstrated only minimal left hand weakness. She was subsequently discharged home with continued intravenous heparin therapy.
Findings of the initial admission laboratory studies included a PT of 12.11-13 seconds, a PTT of 21.22-31 seconds, a thrombin time of 9.7-11 seconds, a thyroid stimulating hormone concentration of 19.0-3-7.0 µU/ml, a triiodothyronine resin uptake of 35.6%/25-35%, a thyroxine concentration of 3.904.0-22.0 mg/dl, a free thyroxine index of 1.39/1.1-3.5, and normal levels of protein C, protein S, and antithrombin III; anticoagulant antibody, antinuclear antibodies, and rheumatoid factor were all absent. The patient did have elevated laboratory values for fibrinogen concentration (480/200-400 mg/dl), fibrin split products concentration (40/2-8 mg/dl), and erythrocyte sedimentation rate (76/0-20 mm/hr); all were accountable for the documented thrombus.

Discussion

This patient had a long segment of congenitally bifurcated sagittal sinus that developed initial thrombotic occlusion in only one channel, leading to diagnostic difficulties. Bifurcated superior sagittal sinuses several centimeters long have been described, but their incidence is uncertain.12-15 Chordae willisii are well known in the sagittal sinus, predominantly in the parietal portion, as trabeculae or bands of fibrous tissue, valve-like lamellae, or 2-8-mm-long lamellar sheets.15,16 In large autopsy series, neither Schmutz16 nor Browder et al15 described the presence of a bifurcated sagittal sinus extending for more than 10 mm.

Because of the increased incidence of venous thrombosis in postpartum patients, an MRI scan was obtained in this patient despite the equivocal arteriogram. Usually, with superior sagittal sinus thrombosis and occlusion, venous collaterals bypassing the occluded segment are demonstrated angiographically.4-6 The absence of such collaterals and the presence of minimal arterial irregularities led to the initial arteriographic diagnosis of infarction based on a probable arterial embolic etiology. In retrospect, the presence of the superior sagittal sinus duplication provided an alternative collateral pathway for the venous outflow from the frontal lobes and led to radiological uncertainty.

To assess the radiological prevalence of such bifurcated sagittal sinuses, we prospectively studied a consecutive series of head MRI scans performed on a GE Sigma 1.5-T MRI unit (Milwaukee, Wis.), including both T1- and T2-weighted images. The series consisted of 100 consecutive patients scanned for a variety of diagnostic considerations (e.g., stroke, brain tumor, dementia, gait disorder); patients with a suspected cortical venous thrombosis were excluded. Among this group, two patients demonstrated a longitudinally duplicated sagittal sinus over three or more images, corresponding to a distance of ≥2 cm; none showed evidence of existing thrombosis. A duplicated sagittal sinus is an uncommon but not rare congenital vascular anomaly readily demonstrable on MRI.

Management of venous sinus thrombosis remains controversial and ranges between strictly supportive therapy, anticoagulation, and (potentially) fibrinolytic therapy.5,17-20 Despite the possible risk of exacerbating the hemorheologic component of the infarct, the thrombus propagation in this patient prompted our use of heparin therapy. A preliminary report by Villringer et al20 indicates a significant improvement in outcome and a decrease in morbidity with heparin therapy, even if hemorrhage has already occurred. Our patient did well on heparin therapy, with control of her symptoms and no adverse effects. The thrombus progressed and she worsened clinically despite adequate anticoagulation when heparin was discontinued and warfarin sodium was employed. The basis for this lack of response to warfarin sodium is unclear. The coagulation data did not indicate the presence of an obvious coagulopathy, suggesting the possibility of an undetected coagulation abnormality responsive to heparin but not to warfarin sodium.

This case clearly demonstrates the value of MRI for the diagnosis of cerebral venous thrombosis, especially in this situation with a vascular anomaly. Its advantage relative to angiography regarding safety, speed, cost, and sensitivity suggests that MRI should be the initial diagnostic procedure for evaluating patients with suspected cortical venous thrombotic disease.

References


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