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# Computed Tomography of Dural Sinus Thrombosis

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For 150 years, the variable clinical appearance of dural sinus thrombosis has plagued clinicians. Computed tomography (CT) has alleviated much of the difficulty in making this diagnosis. A high density lesion in the involved sinus on precontrast scans and a filling defect in the sinus on postcontrast scans were the most frequently observed CT abnormalities in 12 patients with sinus thrombosis. Other findings include the "cord" sign and diffuse cerebral edema. Thrombosis of the superior sagittal sinus, transverse sinus, or sigmoid sinus can occur as an isolated event or in association with other disease entities and may cause no recognizable neurologic impairment.

Thrombosis of the dural venous sinuses has been recognized for almost 150 years [1]. Nevertheless, the variable clinical appearance of sinus thrombosis and its association with a wide spectrum of pathologic entities continue to make clinical diagnosis of this disease very difficult [2]. Yet, with computed tomography (CT), the diagnosis may be made with a high degree of reliability. Reported CT features of sinus thrombosis include the "cord" sign, "empty triangle" sign, intracranial hemorrhage, visualization of medullary veins, and diffuse gyral enhancement [3-5]. The occurrence of these signs in 12 patients with sinus thrombosis is reviewed. The most common sign proved to be marked increase in density of the superior sagittal sinus on noncontrast CT scans.

## Materials and Methods

We reviewed the CT scans of 12 patients with dural sinus thrombosis. Eight were neonates and four were adults of up to 64 years old. Surgery or angiography confirmed the diagnosis of sinus thrombosis in all four adults and in one neonate. In the other neonates, the clinical and radiographic findings were believed to be characteristic and no further radiographic studies were obtained. (As our confidence in the diagnosis of sinus thrombosis increased, fewer confirmatory studies were performed.)

One neonate had an intraventricular hemorrhage secondary to a corpus callosum angioma and subsequently developed sinus thrombosis, while in two neonates the sinus thrombosis was a complication of meningitis. The other neonates suffered episodes of severe anoxia or hypoxia; one had a cardiac arrest and another a germinal matrix hemorrhage. In all eight neonates the hematocrit values were normal.

One adult developed sinus thrombosis as a complication of an ischemic stroke and another had radiation therapy for a parietal lobe tumor 12 years before the diagnosis of sinus thrombosis. In the other two adults, sinus thrombosis was an isolated event.

## Results

Of the 12 patients, 11 demonstrated a triangular area of increased density posteriorly at the location of the superior sagittal sinus on precontrast CT scans. In three, the thrombosis extended into a cortical vein, the "cord" sign [3]. Five patients, the four adults and one neonate, had postcontrast CT. In each, a filling defect was demonstrated in the thrombosed

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sinus, including a transverse sinus in one patient and the superior sagittal sinus in four (the "empty triangle" sign) [3, 4]. Eight patients had CT evidence of cerebral edema, but these areas did not show contrast enhancement. Two neonates suffered intracranial hemorrhages, but neither of these hemorrhages was believed to be due to the venous sinus thrombosis (see Discussion). Medullary veins and diffuse gyral enhancement were not observed in any patients. The observations are summarized in table 1.

## Discussion

We found the most consistent CT findings in sinus thrombosis to be an increase in density of the involved sinus on precontrast scans and a filling defect in the involved sinus on postcontrast scans (fig. 1). Although increased density in thrombosed venous sinuses has been reported sporadically [6, 7], this finding is not recognized widely.

In acute superior sagittal sinus thrombosis, the clotted blood within the sinus assumes the same shape as the sinus, a triangle with its base against the calvarium. This dense triangle is most prominent posteriorly, where the sinus is largest. The anterior segment of the superior sagittal sinus is often small or absent, but if it is also involved by thrombosis, the dense triangle will be oriented in the opposite direction from the posterior thrombus. On CT scans near the vertex, areas of both increased and decreased density were seen occasionally, representing either incomplete thrombosis or poor adherence of the thrombus to the wall of the sinus (fig. 2).

The increased density in the thrombosed sinus on precontrast scans was a reliable indicator of acute sinus thrombosis. However, other conditions can mimic the appearance of sinus thrombosis. Prominent subarachnoid spaces or low density of the brain adjacent to the falx will cause an apparent increase in the density of the falx on CT scans [8]. Also, calcification or ossification will increase the density of the dura or falx (fig. 3). The patent sagittal sinus is a low density within the dural folds, but occasionally volume averaging may make it appear to be increased in density. In such cases, viewing the scans at a higher window width (i.e., 400 or 500) may help exclude sinus thrombosis. If a question still remains, postcontrast scans will demonstrate homogeneous enhancement of a normal sagittal sinus. Thrombosed sinuses will not demonstrate homogeneous enhancement (see Discussion). Occasionally, a bony ridge from the calvarium may protrude into the region of the sagittal sinus mimicking thrombosis, but viewing the scans at "bone" windows will exclude sinus thrombosis (fig. 4).

Nelson et al. [9] reported increased density in nonthrombosed venous sinuses in patients with elevated hematocrits. In these patients, postcontrast scans may be necessary to exclude sinus thrombosis. However, in patients with normal hematocrits, increased density in the venous sinuses is strongly suggestive of sinus thrombosis.

Other lesions that mimic sinus thrombosis are subarachnoid hemorrhage and subdural hematomas, particularly interhemispheric subdural hematomas. It is often difficult to separate these lesions on clinical information, hence the CT scan becomes an important diagnostic tool provided anatomic particulars are familiar.

The superior sagittal sinus is a triangular cavity between dural folds with its base against the calvarium. Both the subdural and subarachnoid space lie outside this dural envelope so that blood in either of these spaces will not give the triangular-shaped density of sinus thrombosis. With bilateral subdural hematomas or subarachnoid hemorrhage on both sides of the falx, the unclotted blood in the sinus will appear as a filling defect in the surrounding blood (fig. 5). Occasionally, thick CT slices will cause volume averaging that may obscure the unclotted blood in the sinus; thin slices will help

TABLE 1: CT Finding with Sinus Thrombosis

Case No.	Increased Density on Precontrast Scans	Filling Defect on Postcontrast Scans	Cord Sign	Cerebral Edema
1 . . . .	+	+	+	+
2 . . . .	+	NA	+	-
3 . . . .	+	NA	-	+
4 . . . .	+	NA	-	-
5 . . . .	+	+	-	+
6 . . . .	+	NA	+	-
7 . . . .	+	NA	-	+
8 . . . .	-	+	-	-
9 . . . .	+	+	-	+
10 . . . .	+	+	-	+
11 . . . .	+	NA	-	+
12 . . . .	+	NA	-	+

Note.— + = present, - = absent, NA = not available.

resolve this problem.

The acutely thrombosed sinus has a high density. In subacute cases, the thrombus may be isodense or hypodense, with little evidence of sinus thrombosis on the precontrast CT scans. However, postcontrast scans demonstrate a filling defect in the involved sinus. This is the "empty triangle" sign reported by Buonanno et al. [3]. The five patients in our study who had postcontrast scans all demonstrated filling defects in the thrombosed sinus. The filling defect represents the thrombus, but the contrast enhancement around the thrombus may be due to peridural venous collaterals, granulation tissue involved in the resorption of the clot, or contrast material around the clot itself.

Another reported sign of sinus thrombosis is cerebral edema [3]; low density areas consistent with edema were seen in nine (75%) of our patients. In one neonate, these areas were thought to represent transependymal resorption of cerebrospinal fluid secondary to hydrocephalus caused by meningitis (fig. 6). In the other eight, the low density areas were interpreted as cerebral edema. In adults the diagnosis of cerebral edema is straightforward, but in the neonate it is more difficult. Recent evidence suggests that periventricular low density may be due to immature, nonmyelinated white matter [10], higher water content in the white matter, or higher metabolic activity in the white matter [11]. However, when the low density lesions are diffuse, bilateral, and accompanied by clinical and radiographic signs of increased intracranial pressure (i.e., splitting of cranial sutures), the diagnosis of cerebral edema can be made with more certainty.

Visualization of medullary veins and diffuse gyral enhancement on postcontrast CT scans have been reported also in cases of sinus thrombosis [3-5], but these did not occur in our patients.

Thrombosis of the straight, transverse, or sigmoid sinuses occurs rarely as an isolated event and is usually accompanied by thrombosis of the superior sagittal sinus (fig. 7). However, in the absence of superior sagittal sinus thrombosis, increased density along the other venous sinuses would be difficult to differentiate from subdural hematoma or subarachnoid hemorrhage.

We believe thrombosis of the dural venous sinuses occurs more often than has been recognized. Although patients with sinus thrombosis may be ill, this is not always true. Experimental and surgical removal of large parts of the superior sagittal sinus causes no recognizable neurologic dysfunction [12]. The extent of venous collaterals and the speed at which they develop greatly influence the degree of neurologic impairment. Russell [13] suggests that unless cortical vein thrombosis is also present, intraparenchymal lesions and subsequent neurologic deficits will not develop. Thus,



Fig. 1.—Sinus thrombosis in 25-year-old man. Precontrast scans. Increased density (arrows) in superior sagittal sinus (A) extends into cortical veins (B). C, Postcontrast scan. Filling defect (arrow) in sinus.

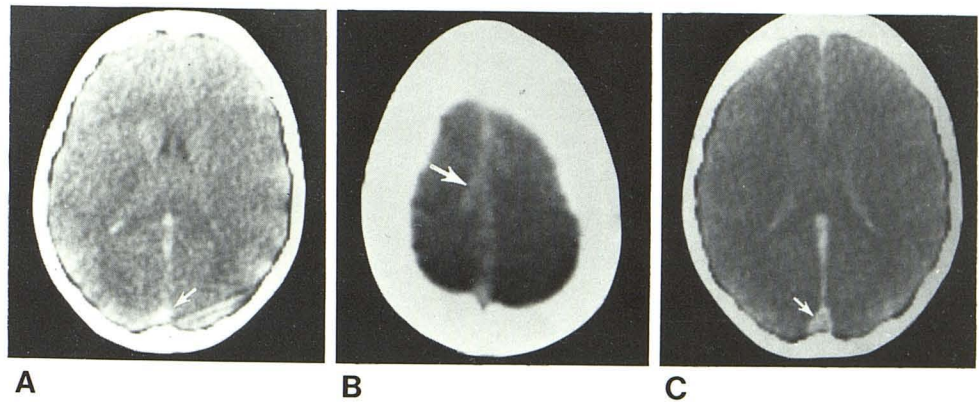
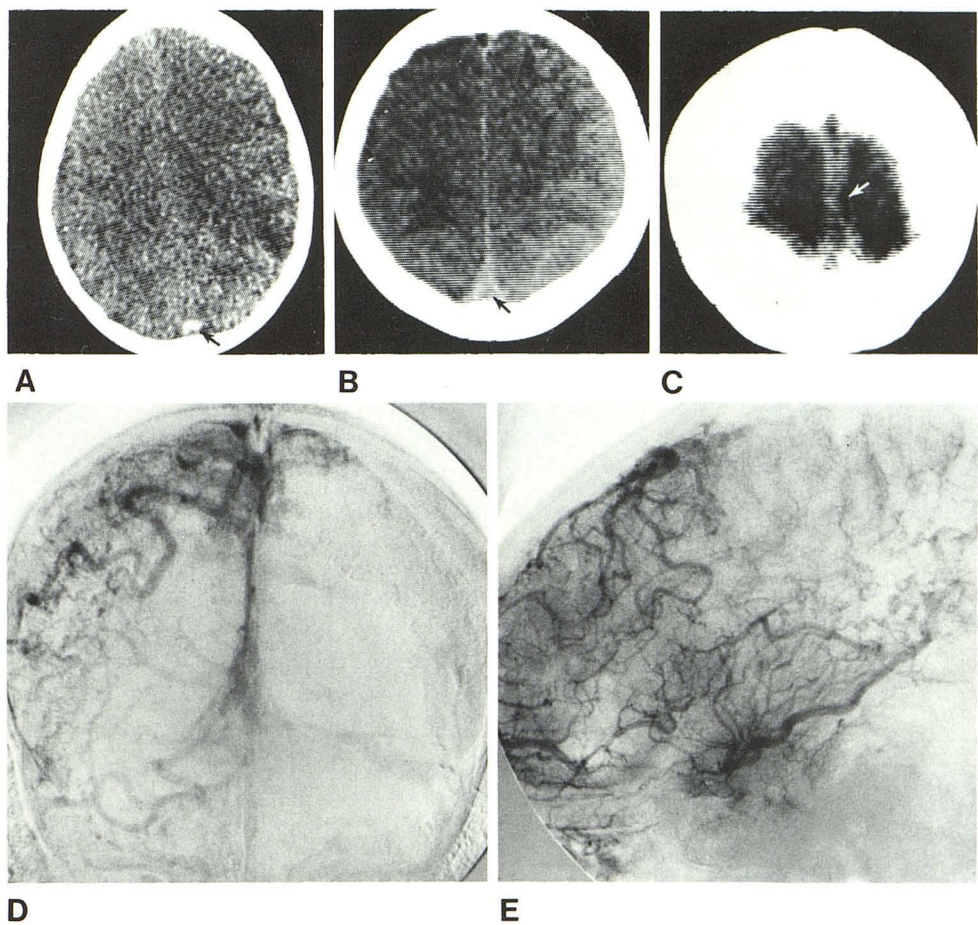


Fig. 2.—Sinus thrombosis in 64-year-old woman with ischemic stroke. A, Pre-contrast scan. Increased density (arrow) in superior sagittal sinus. B and C, Postcontrast scans. Filling defects (arrows) in sinus. D and E, Angiography confirms thrombosis.



thrombosis of the major dural sinuses, in the absence of cortical vein thrombosis, may cause no clinically recognizable focal neurologic dysfunction. One of our four adult patients, whose sinus thrombosis was proven angiographically, had a single generalized seizure, but no focal neurologic abnormality (fig. 1).

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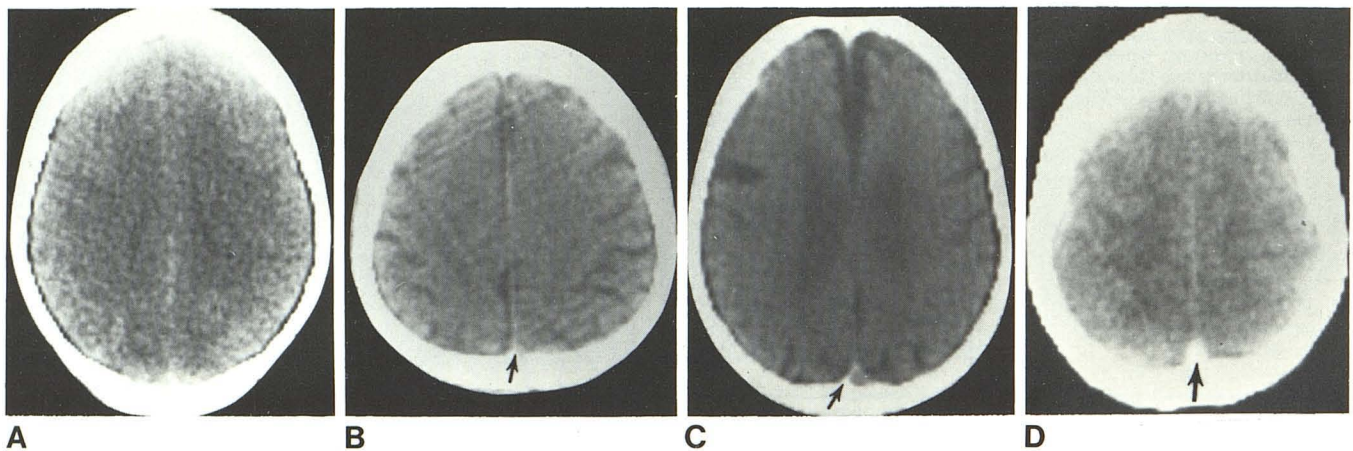


Fig. 3.—Varying densities of superior sagittal sinus in four patients. A, Normal sinus. B, Prominent sinus (arrow) secondary to prominent subarachnoid spaces. C, Prominent sinus (arrow) secondary to calcified dura. D, Sinus thrombosis (arrow) (precontrast scan).

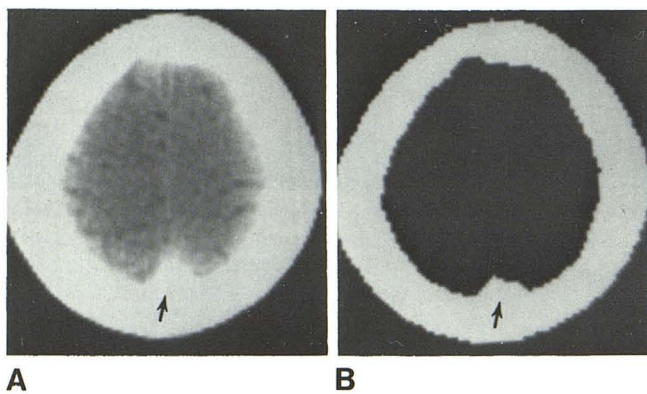


Fig. 4.—Bony ridge (arrows) simulates superior sagittal sinus. Normal (A) and bone window (B) settings.

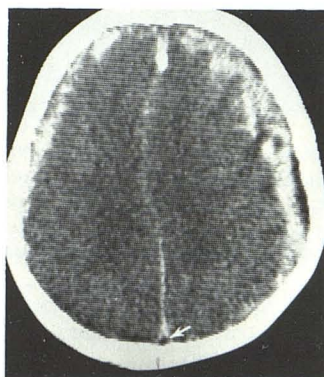


Fig. 5.—Subdural hematoma and patent superior sagittal sinus. Sinus appears as filling defect (arrow) in subdural blood.

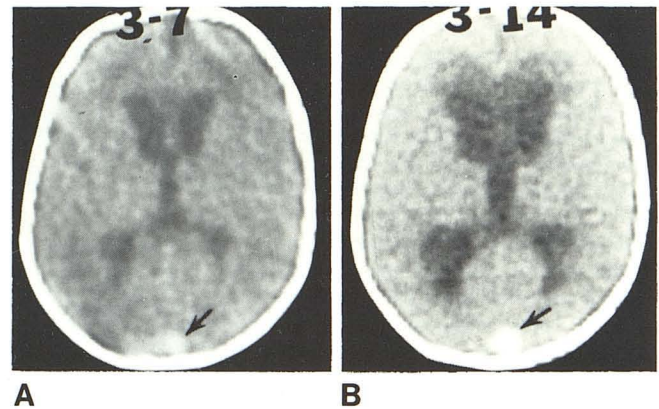


Fig. 6.—Neonate with meningitis and sinus thrombosis. A, Before enhancement. Superior sagittal sinus (arrow) probably thrombosed. B, Follow-up scan 1 week later. Definite sinus thrombosis (arrow).

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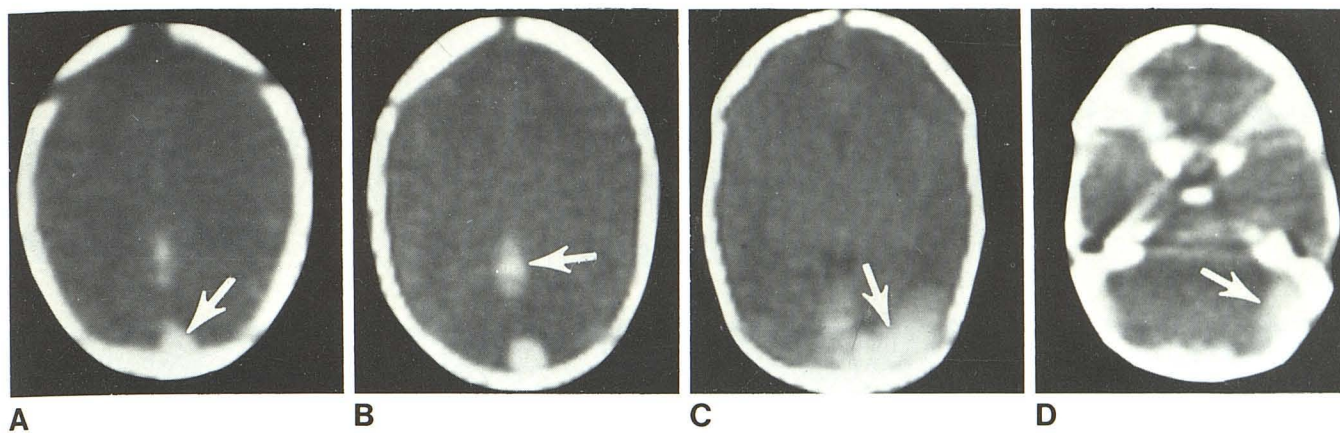


Fig. 7.—Neonate with cardiac arrest after cesarean section. Precontrast scans demonstrate thrombosis (*arrows*) of sagittal, straight, transverse, and sigmoid sinuses, respectively. (Hematocrit was normal).