Transcranial Doppler ultrasound findings in cerebral venous sinus thrombosis

Case report


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Transcranial Doppler ultrasound (TCD) findings are described in a patient with acute thrombosis of the sagittal venous sinus. The TCD finding of prominent venous signals adjacent to the middle cerebral artery gave the first indication of the diagnosis, which was subsequently confirmed by computerized tomography. Awareness of the possible TCD findings in patients with a similar history may lead to a more rapid diagnosis of cerebral venous sinus thrombosis.

Key Words • cerebral venous thrombosis • transcranial Doppler ultrasound • delta sign • stroke

Cerebral venous sinus thrombosis may have no obvious precipitating cause, although it has been associated with mastoid or paranasal sinus infection, dehydration, trauma, contraceptive medication, hematological disorders, and neoplasia. The diagnosis of cerebral venous sinus thrombosis can be difficult and is often one of exclusion rather than of positive findings. Although contrast-enhanced computerized tomography (CT) and magnetic resonance (MR) imaging have improved the positive diagnosis of venous sinus thrombosis, difficult diagnostic and management problems still occur in some patients.

Patients with cerebral venous sinus thrombosis may present with headaches, nausea, vomiting, disturbed vision, and symptoms of raised intracranial pressure and may have focal neurological signs mimicking subarachnoid hemorrhage (SAH), acute ischemic stroke, or central nervous system infection. The patient may be unable to cooperate, even with a relatively short procedure such as CT; MR imaging and angiography may require sedation to be successful, making investigation of these patients difficult.

Transcranial Doppler ultrasound (TCD) findings in cerebral venous sinus thrombosis have not been described previously. In the case described here, the abnormal TCD findings gave the clue to the correct diagnosis. Transcranial Doppler ultrasonography is a simple technique that can be performed at the patient's bedside and is possible even in very restless patients. It is not suggested that a TCD study provides the definitive diagnosis but that it may yield sufficient evidence to justify more aggressive investigation in order to establish a positive diagnosis.

Case Report

This 47-year-old woman was admitted with a 2-day history of severe headache associated with vomiting and photophobia but no loss of consciousness. There was no history of note, and her only medication was estrogen hormone replacement therapy. She had returned from a holiday in Spain a few days prior to admission.

Examination. On examination, the patient was confused, with a fluctuating consciousness level (Glasgow Coma Scale score 13 or 14) and mild neck stiffness. There were no focal neurological signs. A CT brain scan without contrast enhancement, obtained on admission, showed no definite abnormality. A lumbar puncture revealed mildly blood-stained cerebrospinal fluid (CSF) and the pressure was elevated (36.5 cm H₂O). A TCD examination using a 2-MHz probe showed normal arterial flow velocity signals from the middle, anterior, and posterior cerebral arteries, but prominent, high-amplitude venous signals were detected adjacent to both middle cerebral arteries (Fig. 1). As venous sig-
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Fig. 1. Transcranial Doppler ultrasound recordings through the temporal bone window of the left (A and B) and right (C and D) middle cerebral arteries (MCAs) at a depth of 45 mm (A and C) or 55 mm (B and D) from the skin surface showing the prominent venous signals immediately adjacent to the MCA signals. The annotations shown in A also apply to B, C, and D. The typical MCA pulsatile arterial signal (thin arrow) is seen above the baseline (arrowhead), indicating flow toward the probe and away from the center of the head. The abnormal venous signal of more constant amplitude (wide arrow) is seen below the baseline, indicating flow away from the probe and toward the center of the head. Identification of both signals simultaneously indicates that the artery and vein must be immediately adjacent to one another.

In our experience, the possibility was raised of a cerebral venous sinus thrombosis with opening of venous collateral channels. ACT brain scan was obtained with intravenous injection of contrast material but was unhelpful due to the extreme restlessness of the patient. The following day, with the patient sedated, a CT brain scan obtained immediately after intravenous administration of contrast material demonstrated an "empty delta" sign in the sagittal sinus, confirming the presence of sagittal venous sinus thrombosis (Fig. 2). Further investigations, such as angiography or MR imaging, were not performed as the CT brain scan was considered to be sufficiently diagnostic in this case.

Treatment and Posttreatment Course. The patient was treated with intravenous heparin and a plasma expander. She improved, becoming orientated with a normal consciousness level. Over the next 2 weeks, her condition continued to improve, with an occasional recurrence of headache, and she was well enough to be discharged 3 weeks after admission on a course of oral anticoagulant agents.

The prominent venous signals had disappeared by 2 weeks after admission. No definite precipitating cause of the sinus thrombosis was found, and it is suggested that the combination of hormone replacement therapy plus possible mild dehydration while on holiday in a hot climate was the likely etiology.

Discussion

Cerebral venous sinus thrombosis is relatively uncommon and may be overlooked unless specifically sought. Patients may present with acute or chronic symptoms. In one recent series, six of 24 patients diagnosed with benign intracranial hypertension had thrombosis of the dural sinuses on angiography. Further difficulties may be encountered in establishing the diag-
nosis radiologically. High-dose dynamic contrast-enhanced brain CT may exclude major thrombosis of the cavernous, sagittal, or sigmoid sinuses but not the smaller sinuses or superficial veins. Magnetic resonance imaging may be more sensitive than CT, although thrombus may appear atypical possibly due to associated infection. Cerebral angiography is the definitive examination at present (but may be superseded by new MR imaging-angiography techniques); however, the procedure requires meticulous attention to detail and, due to the wide range of normal variants of cerebral venous anatomy, may (like CT and MR imaging) be difficult to interpret in individual cases.

Treatment of cerebral venous sinus thrombosis is controversial. The relative infrequency of the condition makes the establishment of large randomized controlled treatment trials difficult. However, a randomized controlled trial of treatment with intravenous heparin versus placebo administration was carried out by Einhäupl, et al., and suggested considerable benefit in the heparin-treated group, even in patients in whom hemorrhagic infarction was already visible on a CT brain scan obtained before the start of heparin treatment. Therefore, despite the blood-stained CSF in our patient, she was treated with intravenous heparin administration.

Deep cerebral veins can be detected in the midline of normal subjects with TCD examination (a 70- to 80-mm depth) but neither prominent venous signals in the region of the middle cerebral artery with TCD (a 35- to 60-mm depth from the temporal bone window) nor the TCD findings in cerebral venous sinus thrombosis been described previously. In our patient with sagittal venous sinus thrombosis, prominent venous signals were observed paralleling the middle cerebral arteries with the flow directed toward the center of the head, presumably due to increased flow via collateral venous channels to the deep cerebral veins bypassing the thrombosed sagittal sinus. The venous signals disappeared as the patient improved.

In our experience, we have occasionally observed prominent venous signals in patients with SAH or who have suffered an ischemic stroke (on the ischemic side); however, these signals have never been a persistent finding and are often neither prominent nor bilateral. We have never observed prominent venous signals in the region of the middle cerebral artery in normal individuals.

Since the current case report, we have observed two other patients with a presumed diagnosis of deep cerebral vein thrombosis, neither of whom showed this pattern of prominent venous signals in the region of the middle cerebral artery on TCD examination. However, this finding might not be expected if the deep, as opposed to the superficial, cerebral veins were affected by thrombosis.

Conclusions

In our patient the finding of prominent venous signals adjacent to the middle cerebral arteries raised the possibility of cerebral venous sinus thrombosis and led to the correct diagnosis being made using contrast-enhanced brain CT. We think that the observation of prominent bilateral venous signals paralleling the middle cerebral arteries on TCD examination is not specific for cerebral venous sinus thrombosis, nor would it be observed in all such cases; however, the finding should raise the possibility of the condition in patients with appropriate clinical symptoms.

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References

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