Progressive posttraumatic superior sagittal sinus thrombosis complicated by pulmonary embolism

Case report

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A case of progressive posttraumatic superior sagittal sinus thrombosis is presented, with the complication of pulmonary embolism. A good clinical outcome followed treatment with barbiturate coma.

KEY WORDS • sagittal sinus thrombosis • dural sinus thrombosis • head injury • pulmonary embolus • barbiturate coma • computerized tomography

Superior sagittal sinus thrombosis is rarely observed following closed head trauma. We present a well documented case with the unusual complication of pulmonary emboli, possibly originating from this intracranial source. A good clinical result followed treatment with barbiturate coma.

Case Report

This 20-year-old man was injured when his stalled car was struck by another vehicle. He was found unresponsive and apneic. Mouth-to-mouth resuscitation was followed by the onset of spontaneous respiration.

Examination. On arrival at the hospital, the patient’s vital signs were normal. Pupils were in midposition and reactive. He did not open his eyes or verbalize, but withdrew all extremities to pain (Glasgow Coma Scale score 7). Prothrombin time (PT), partial thromboplastin time (PTT), and platelet, electrolyte, and complete blood counts were normal. Cervical spine, pelvic, and chest x-ray films were normal. Computerized tomography (CT) of the head was within normal limits. Peritoneal lavage was negative. On the following morning, the patient was still lethargic, but could follow commands and give his name. He continued to improve over the next few days, and was able to eat, walk, and follow simple commands.

On the 5th hospital day, the patient became more lethargic. A CT scan showed new focal parasagittal hematomas in the left parietal region and cerebral swelling (Fig. 1 upper scans). The following day the patient became stuporous, had a right focal motor seizure, and was placed on Dilantin (phenytoin) therapy. On the 7th hospital day, he had a grand mal seizure, became unresponsive, and required intubation and ventilation. A repeat CT scan demonstrated an increased number of small parasagittal intracerebral hematomas and diffuse cerebral swelling (Fig. 1 center scans). Digital angiography confirmed the diagnosis of progressive superior sagittal sinus thrombosis.

Coagulation studies revealed normal PT, PTT, thrombin time (TT), and platelet count, and negative cryoglobulin, but showed elevated fibrinogen (465 mg/dl) and fibrin split products (32 to 64 µg/ml). Low-dose heparin and Rheomacrodex (low molecular weight dextran) therapy was begun. A Richmond bolt was placed and pentobarbital was instituted when the intracranial pressure (ICP) persisted above 20 mm Hg. Pentobarbital coma was maintained for 72 hours with good control of ICP (less than 10 mm Hg).

Course. On the 10th hospital day the patient’s oxygenation deteriorated, with PaO2 dropping from 208 to 62 mm Hg (fraction of inspired oxygen 50%). Chest x-ray films showed a left lower-lobe atelectasis, and a ventilation-perfusion lung scan showed a right-sided segmental defect. Bronchoscopy was performed and the atelectasis resolved. Following a similar drop in PaO2 36 hours later, a second ventilation-perfusion lung scan showed a complete lobar defect in the right lower lobe.

FIG. 1. Evolution on serial computerized tomography of superior sagittal sinus thrombosis. *Upper Scans:* Noncontrast scans 5 days after admission. The superior sagittal sinus (arrows), straight sinus, and vein of Galen are abnormally dense due to acute thrombosis and associated stasis. Focal parasagittal hemorrhages are present unilaterally at the vertex. Scans at other levels showed increased tentorial density (see text) and effacement of cerebrospinal fluid cisterns, indicating cerebral swelling. *Center Scans:* Post-contrast scans 7 days after admission. The density of thrombus within the dural sinuses is largely obscured by contrast enhancement. Parasagittal hemorrhages have increased in number and are now strikingly symmetrical. Scans at other levels showed intense tentorial enhancement (see text) and persistent cerebral swelling. *Lower Scans:* Post-contrast scans 8 weeks after admission. Old clot within the superior sagittal sinus is now seen as a low-attenuation filling defect inside the enhancing sinus (arrows). The thrombus is more apparent at high display levels and narrow display windows (middle scan). Parasagittal hemorrhages are resolving. A small frontal subdural collection is seen. Scans at other levels showed reduced cerebral swelling.
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Fig. 2. Selective pulmonary arteriogram 12 days after admission. A large intraluminal filling defect (arrow) occludes a segmental lower-lobe branch. Smaller emboli are seen elsewhere (arrowheads).

consistent with a high probability of pulmonary embolus. This was confirmed by pulmonary angiography (Fig. 2). Bilateral leg venograms were performed, with no evidence of thrombi. Heparin was increased to 5000 IU subcutaneously every 8 hours, and with mechanical ventilation the patient was able to maintain satisfactory blood oxygenation.

Throughout the hospital course, PT, PTT, and TT were normal, but fibrinogen and fibrin split products remained elevated. The platelet count rose steadily to a peak of 740,000 on the 26th hospital day. The patient gradually improved, and on the 61st hospital day he was discharged to a course of rehabilitation with minimal weakness in his right upper extremity and diplopia secondary to right fourth nerve paresis.

Discussion

Sagittal sinus thrombosis is often mentioned but rarely observed as a complication of closed head injury. Traumatic thrombosis was first reported by Bagley1 in 1934, and Carrie and Jaffé4 reported two cases diagnosed at autopsy in 1954. A syndrome of “benign posttraumatic intracranial hypertension” was described by Beller2 in 1964 in seven patients who developed signs of intracranial hypertension several days to many months after head injury. The cases were ascribed to longitudinal sinus thrombosis, but are poorly documented, with normal angiograms in two patients, normal ventriculograms in two patients, and no studies in three patients. There are no reports of posttraumatic sinus thrombosis since the advent of CT scanning.

Digital angiography is an excellent tool for the diagnosis of superior sagittal sinus thrombosis, and was confirmatory in our case. However, the initial diagnostic clues are usually present on CT scans, and their recognition can lead to early suspicion and appropriate therapy.

Many of the characteristic CT findings are well illustrated in this case.6,7,9-11 Direct visualization of thrombus as increased attenuation within the sinus on a non-contrast scan may be seen in cases of acute thrombosis. This abnormal sinus density is obscured by adjacent enhancement on post-contrast images. On the other hand, subacute or chronic thrombus may not be apparent on plain scans, but is detected as a filling defect within the sinus on contrast-infused images (Fig. 1 lower scans). A narrow CT display window and/or a high display level may be necessary to demonstrate the difference in attenuation between intraluminal clot and contrast medium circulating in the blood.

Secondary CT findings may also be highly suggestive of superior sagittal sinus thrombosis. Focal parasagittal hemorrhages are the most specific secondary feature on plain scans, which may also show small ventricles and effaced cisterns due to cerebral swelling. Contrast-infused scans may demonstrate gyriform cortical and prominent tentorial enhancement. The increased tentorial density probably represents dural congestion due to stasis and collateral venous flow, and is especially prominent with involvement of the straight sinus or torcular.

To our knowledge, this is the first report of a possible pulmonary embolus from thrombus in a dural sinus. The association in our case of well documented sinus thrombosis, proven angiographically, and pulmonary embolism remains circumstantial, but the negative leg venography suggests the possible intracranial source.

Treatment of superior sagittal sinus thrombosis is controversial. Surgical thrombectomy with survival has been reported.12,13 Gettellfinger and Kokmen8 used anticonvulsant and anti-edema agents successfully, and recommended avoiding anticoagulant drugs because of fatal hemorrhage in two patients. Di Rocco, et al.,5 reported a series of five patients treated with heparin and urokinase, with complete recovery of non-traumatic thrombosis.

In a head-injured patient, therapeutic options are limited. Our patient had a successful outcome following treatment with low-dose heparin and Rheomacrodex to limit clot propagation, Dilantin to control seizure activity, and pentobarbital coma to treat increased ICP.13

References


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