Traumatic Thrombosis of Dural Venous Sinuses in Closed Head Injuries

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In 1946, Ecker first described the relationship of blunt skull injury to thrombosis of the dural venous sinuses. In a period of 12 months he observed 11 cases in which either a linear or a diastatic fracture of the skull crossed a major dural venous sinus. He pointed out that a fracture of the skull could cross an underlying sagittal or lateral sinus without being accompanied by impairment of the venous blood flow through the sinus. In almost all of his cases, however, there was sufficient obstruction of the venous outflow to cause mental confusion, headache, and choked discs. He postulated that, in these cases, the cerebral veins became engorged, causing increased intracranial pressure accompanied by papilledema. When these signs occurred after head trauma, it was logical to suspect the presence of intracranial hemorrhage. But if trauma could be eliminated as a cause of the symptoms, the clinical picture could be attributed to the mechanism described. It is to his credit that he gave the clinical picture so accurately without the aid of dural sinus venography.

Martin, in 1955, described a group of patients with head injuries characterized by paralysis resulting from obstruction and thrombosis of the cortical veins which drained into the sagittal sinus. More common were cases in which the signs of increased intracranial pressure developed. In these cases without evidence of severe intracranial bleeding, there was ultimate recovery without operation. Their many points of similarity to cases of otitic hydrocephalus were attributed to complete or partial obstruction of the superior longitudinal or lateral sinus. This syndrome Martin called “traumatic hydrocephalus.” He pointed out that in such cases injury to the dura in the walls of the sinus and extension of the thrombus from abrasions of the scalp or injured emissary veins were probably the most important factors causing sinus thrombosis.

Carrie and Jaffe reported two cases in which thrombosis of the superior sagittal sinus occurred as the result of trauma without penetrating injury or fracture of the skull. Studies indicated that intramural hemorrhages caused by rupture of small sinusoids was a common sequela of trauma. The resulting injury to the endothelium lining predisposed the sinus to thrombosis.

In an analysis of 95 cases of benign intracranial hypertension, Foley found that intracranial hypertension without visible cause followed a mild head injury in four cases. There was little clinical evidence of thrombosis of the superior longitudinal sinus, and he was reluctant to ascribe the condition to probable thrombosis in the lateral sinus.

Recently, Beller described the clinical characteristics in seven cases of a benign form of traumatic intracranial hypertension. The studies suggested that partial or complete thrombosis of the sinuses, with subsequent interference in the venous blood flow and in the normal absorption of cerebrospinal fluid, was the mechanism responsible for this clinical syndrome.

In a previous paper the pathological findings were discussed in 10 cases of head injury associated with various forms of trauma to the dural venous sinuses. Non-fatal injury to the dural venous sinuses appeared to be more common than had been formerly realized.

The present paper summarizes 17 cases of thrombosis of the dural venous sinuses, including three of the four presented in 1957. Fourteen cases are examples of fracture of the occiput with ipsilateral thrombosis of the lateral or sigmoid sinus. Three cases illustrate traumatic thrombosis of a dural venous sinus without fracture of the skull but with associated ipsilateral injury. In each case, the clini-
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Case Reports

Case 1. A 4-year-old boy was struck by an automobile and rendered unconscious for 1 week. There was bleeding from the left ear, but this lasted only for a short time. The patient showed improvement until 1 month after the accident when he had a sudden onset of headache, nausea, and vomiting.

Examination. The boy was cooperative, but could not sit up because of weakness and truncal ataxia. MacEwen's sign was present on percussion of the skull. Two diopters of papilledema were noted bilaterally, with incipient hemorrhages in the left disc. Old caked blood was found in the left ear canal, but there was no deficit in hearing.

X-ray findings. Roentgenograms of the skull showed separation of the coronal and sagittal sutures. The lateral sinus markings on the occiput were present bilaterally. A fracture line extended through the left occipital bone inferiorly and anteriorly into the left petrous pyramid. This fracture line crossed the groove for the left lateral sinus marking. A ventricular puncture disclosed a xanthochromic cerebrospinal fluid under increased pressure. The x-rays showed a moderate degree of communicating hydrocephalus.

Dural sinus venography (Fig. 1) showed a dilated sagittal sinus, torcular Herophili, and right lateral sinus. In the left lateral sinus, there was an oblique obstruction to the passage of the opaque medium 2.5 cm from the torcular. The fracture line crossed the left lateral sinus just medial to the site of thrombosis. There was no evidence of drainage of blood via the collateral diploic or scalp veins or vertebral plexuses.

Case 2. This 5-year-old girl sustained a minor blow to the right occiput in an automobile accident. Shortly after the accident she developed increasing vomiting and headache.

Examination. Six days after the injury, there were no localizing signs but there was bilateral papilledema. Lumbar puncture disclosed a cerebrospinal fluid pressure of 330 mm; the fluid was clear and colorless. Jugular compression tests were not done. Bilateral trephines disclosed no epidural or subdural collections of blood. Cerebral cortex bulged into the trephine sites.

Discussion

The clinical picture of increased intracranial pressure expressed by headache, visual disturbances, papilledema, and elevated CSF pressure was uniform in all 17 patients in this series.

Fourteen of these patients had fractures of the occiput, all topographically related to the underlying sinus. In most instances, the symptoms of increased intracranial pressure arose shortly after the injury, unaccompanied by signs of localization. In 13 of these 14 patients, the occipital fracture was accompanied by unilateral thrombosis of the ipsilateral sinus; in one patient the occipital fracture was bilateral, accompanied by unilateral thrombosis of the lateral sinus. There was thrombosis in the right lateral sinus in seven cases and in the left lateral sinus in seven cases. The thrombosis extended usually from the site of fracture to the torcular. Evidence of collateral drainage via diploic and scalp veins and vertebral plexuses was present in the majority of cases. The remaining cases seemed to overcome the thrombosed lateral sinus by dilatation of the intact contralateral sinus system.

Three were three cases of increased intracranial pressure due to trauma that did not show a fracture on x-ray. Although these cases appear to be in the minority, they do illustrate the fact that thrombosis of a lateral sinus may occur without fracture of the occiput. Clinically they resemble those cases in the first category in which the thrombosis
was accompanied by a visible fracture that crossed the lateral sinus marking in the occiput. Only one thrombosis was seen at the junction of the lateral and sigmoid sinus; in this case, the fracture involved the mastoid process of the same side.

It may be assumed that mechanical obstruction of the cerebral venous outflow by thrombosis of the sagittal, dominant lateral, or sigmoid sinus produces an increased venous pressure within the sinus system proximal to the thrombosis. This increase of venous pressure prevents the normal absorption of the CSF, thus producing increased intracranial pressure. In the majority of cases, the collateral venous drainage via the diploic and scalp veins and the vertebralplexuses, and the dilatation of the intact contralateral sinus system, seems to temporarily interrupt the venous circulation. The benign clinical course and spontaneous recovery of these patients from the syndrome of increased intracranial pressure may be attributed to recanalization of the thrombosed sinus.

**Summary**

The clinical picture of benign intracranial hypertension following traumatic thrombosis of a dural venous sinus has been substantiated by dural sinus venography. An analysis of 17 cases showed that, in 14 cases, the thrombosis was ipsilateral to the side of a fracture in the occiput while in three cases,
the thrombosis occurred without evidence of fracture in the occiput. In both fracture and non-fracture cases, the clinical picture was the same, regardless of the site of thrombosis.

References