Raised intracranial pressure (ICP) from venous sinus thrombosis and cerebrospinal fluid (CSF) hypotension from cerebrospinal fluid leak may both occur after head injury.

**REPORT OF A CASE**

A 14-year-old girl fell off a truck, striking her head. She lost consciousness for 7 minutes. Results of initial evaluation and computed tomography (CT) of the head appeared to be unremarkable on the day of her injury. Three days later, she presented with symptoms of increased ICP including persistent headaches, vomiting, and pulsatile tinnitus, as well as decreased hearing on the right. Head CT and a CT angiogram showed a complex right mastoid petrous fracture and a right sigmoid sinus thrombosis with extravasation of blood from the right sigmoid sinus into the middle ear (Figure, A). The patient’s condition remained stable throughout her 5-day hospital stay, without visual complaints or worsening intracranial hypertension. A repeat CT angiogram demonstrated resolution of active extravasation from the right sigmoid sinus, without change in the degree of thrombosis.

Two weeks later the patient developed increased head swelling, worsening of her headaches (which were relieved when she was lying down), and horizontal binocular diplopia. On examination, she had diffuse head enlargement with a fluctuant fluid wave. A CT scan showed diffuse subgaleal fluid from one temporalsis to the other, suggestive of cerebrospinal fluid (CSF) (Figure, B). Neuro-ophthalmologic examination showed decreased abduction in both eyes with a comitant 12-prism diopter esotropia consistent with divergence insufficiency. There was no papilledema.

The findings of divergence insufficiency in this setting suggested abnormal ICP, either increased or decreased. Therefore, to investigate the possibility of persistent venous sinus thrombosis as a cause of elevated ICP, follow-up CT angiography was performed, which demonstrated resolution of the right sigmoid sinus thrombosis. No measurement of the CSF pressure was obtained, so as not to worsen her symptoms presumed to be related to CSF hypotension. Her diplopia fully resolved within 2 weeks and results of follow-up examination were normal.

**COMMENT**

This patient sustained a head injury with a skull fracture. The trauma resulted in venous sinus thrombosis, which caused increased ICP. Elevated ICP in the setting of a complex skull fracture likely caused the subsequent development of a CSF leak through the fracture site, resulting in the patient’s low pressure symptoms when her venous sinus thrombosis resolved. Although the ICP was never measured in this patient, the clinical presentation highly suggested the sequence of increased ICP followed by low CSF pressure.

Venous sinus thrombosis is a known, but uncommon, complication of head trauma. It results from either propagation of thrombosis from superficial scalp veins draining into the major sinuses, or fracture of the sinus itself.® Venous sinus
thrombosis can cause elevated ICP because of impairment of passive CSF resorption at the level of the arachnoid villi. Our patient's initial symptoms were consistent with elevated ICP from her venous sinus thrombosis. Two weeks later, she developed extensive subgaleal fluid collections consistent with mostly CSF, likely mixed with some blood.

Although posttraumatic CSF leaks due to fistulas or unclosed wounds may frequently result in otorrhea or rhinorrhea, subgaleal collections of CSF are less common, probably because of the substantial amount of force required to separate the scalp from the skull. We suspect the mechanism responsible for our patient's subgaleal collections involved initial increased ICP from venous sinus thrombosis forcing CSF through the temporal bone fracture. Later, with resolution of the venous sinus thrombosis and with a large quantity of CSF displaced extracranially, in the setting of a persistent CSF leak, the patient developed symptoms of low ICP, including divergence insufficiency.

Accepted for publication January 30, 2004.

Author contributions: Study concept and design (Drs Turan, Biousse, and Newman); acquisition of data (Drs Turan and Biousse); analysis and interpretation of data (Dr Biousse); drafting of the manuscript (Drs Turan and Biousse); critical revision of the manuscript for important intellectual content (Drs Biousse and Newman); administrative, technical, and material support (Drs Turan and Biousse); study supervision (Drs Biousse and Newman).

This study was supported in part by a departmental grant (Department of Ophthalmology) from Research to Prevent Blindness Inc, New York, NY, and by core grant P30-EY06360 (Department of Ophthalmology) from the National Institutes of Health, Bethesda, Md. Dr Newman is a recipient of a Research to Prevent Blindness Lew R. Wasserman Merit Award.

Correspondence: Nancy J. Newman, MD, Neuro-ophthalmology Unit, Emory Eye Center, 1365-B Clifton Rd NE, Atlanta, GA 30322 (ophnjs@emory.edu).

References