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Giant Cervical Epidural Veins after Craniectomy for Head Trauma

Ronald D. Caruso, Mark V. Smith, Ja Kwei Chang, John J. Wasenko, and Arthur E. Rosenbaum

Summary: Markedly dilated cervical epidural veins and right upper extremity weakness developed in a 43-year-old man 4 months after contralateral craniectomy for head trauma. After cranioplasty, his symptoms improved markedly and the size of the veins returned to normal. These findings suggest that enlarged cervical epidural veins may occur without an underlying vascular lesion and that upper extremity weakness may occasionally be attributable to spinal cord venous stasis.

Greatly enlarged cervical epidural veins are rarely seen on imaging studies. Enlargement is usually caused by an underlying vascular lesion such as a malformation or a jugular venous obstructive lesion. This finding may occasionally occur without an associated vascular abnormality.

Case Report

A 43-year-old man was admitted comatose after falling two stories onto concrete. Emergency craniectomy was performed to relieve large left frontoparietal epidural and subdural hematomas, a depressed skull fracture, and bilateral frontal lobe and left temporal lobe contusions. Findings on cervical spine radiographs were normal. The dense right hemiparesis slowly improved and the patient was discharged 6 weeks after surgery with a mild right hemiparesis, moderate cognitive impairment, and a sizable craniectomy defect.

Ten weeks later, the patient returned with markedly worsening weakness in the right upper extremity. Electromyography showed widespread lower motor neuron abnormalities, most severely affecting the right C-8 muscles. MR images of the cervical spine disclosed markedly dilated epidural veins, which largely occupied the bony spinal canal and foramina. The veins were maximal in size at C-1 and tapered to a normal appearance at T-1; signal and contour of the spinal cord were normal (Fig 1). Cerebral MR imaging showed no new lesions, and brachial plexus MR studies were normal. Enhanced two-dimensional phase-contrast MR angiography and selective cerebral and pan-spinal catheter angiography confirmed the presence of dilated epidural veins communicating with a dilated condylar (condyloid) venous system; internal jugular veins were normal (Fig 1). No intracranial or spinal arteriovenous fistula was identified. The upper extremity symptoms were ascribed to a combination of cerebral damage, skull defect, and venous

stasis. Diuretic and steroid therapies were unsuccessful and a cranioplasty was performed.

Over the next 2 months, strength and dexterity of the right upper extremity improved markedly. At 10 weeks postoperatively, a cervical MR study was normal (Fig 1), although cognitive deficits persisted.

Discussion

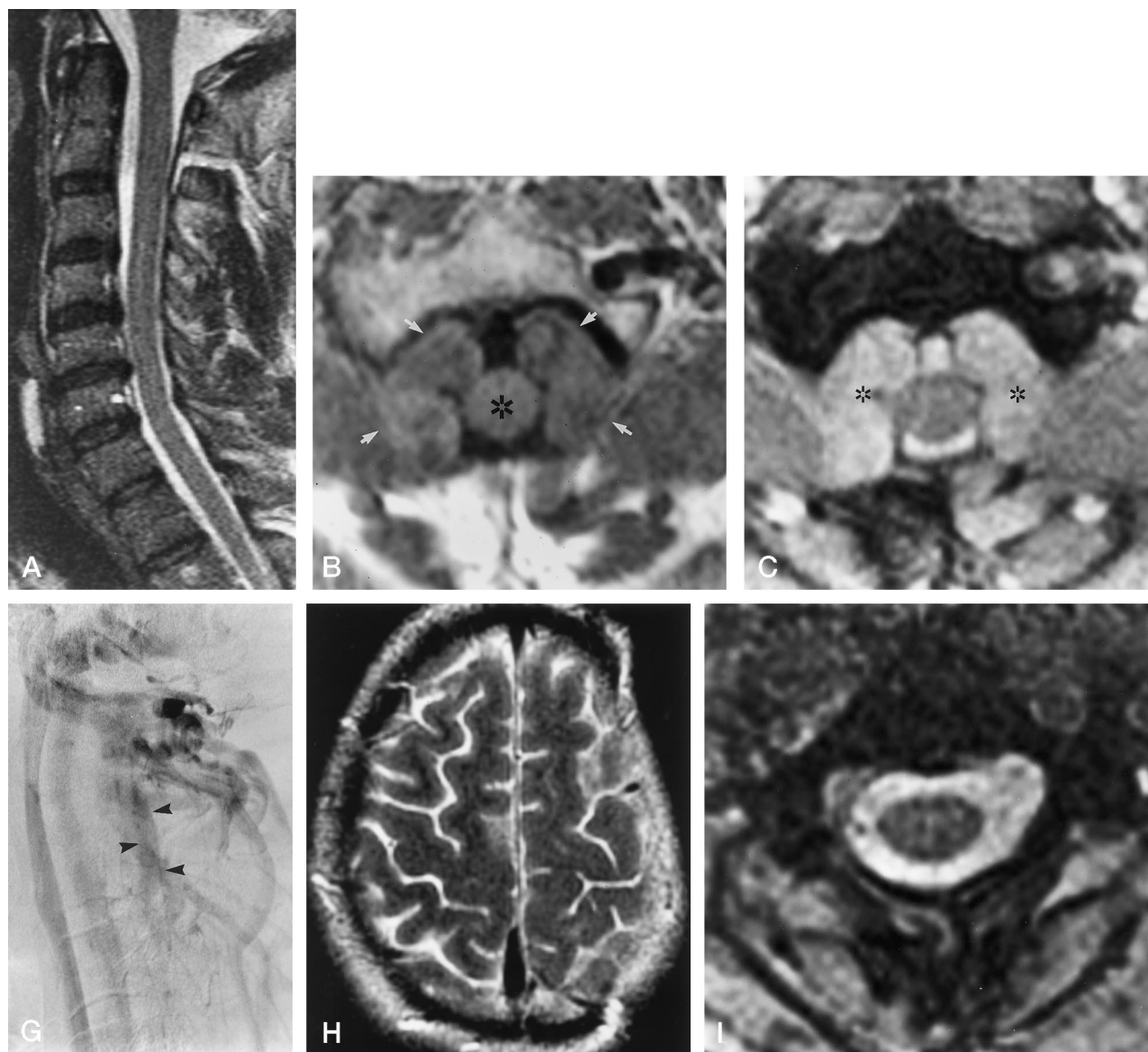
Arnautovic et al (1) elegantly reviewed and expanded information on venous drainage at the skull base, including the multiple interconnections of the cerebral and spinal venous systems. These researchers indicated that the spinal system may function as an alternative cranial drainage system under some circumstances and that the cervical venous system can affect intracranial pressure. Indeed, in healthy persons, the cervical vertebral venous plexus is known to enlarge in the upright position owing to preferential shunting from the internal jugular circulation (2-5). Venous enlargement may also occur from disc disease or compressive lesions of the spinal cord (6). Enlarged epidural or intradural veins are often seen in conjunction with vascular lesions, commonly from arteriovenous fistulas in the thoracolumbar region. When symptomatic, they are typically associated with spinal cord abnormalities (7-13). Brunereau et al (7) reported 12 cranial dural arteriovenous fistulas with accompanying enlarged cervical veins. Half of them had spinal drainage limited to the cervical region without associated extremity symptoms or myelopathy, whereas the other half were accompanied by progressive paraparesis or quadriparesis. Enlarged epidural veins have also resulted from processes causing impaired drainage of the internal jugular circulation, where the epidural veins serve as collateral pathways (eg, superior vena caval thrombosis or cardiac disease). Dickman et al (14) described a case of myelopathy developing from epidural varicose veins compressing the cord at the cervicothoracic junction and improving after surgery.

Several reports have documented neurologic improvement after cranioplasty for repair of postsurgi-

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cal bone defects, although its pathophysiology remains controversial (15, 16). Suzuki et al (15) reported significant improvement after cranioplasty in three of four patients who had prior surgery for acute extraaxial traumatic hematoma and in two other patients with spontaneous putaminal hemorrhage. These authors performed pre- and postoperative dynamic CT of the head and identified findings consistent with increased blood inflow volume. Each of their five patients had at least some cognitive improvement after cranioplasty: four of the five increased their Glasgow coma score from 14 to 15, four had improved speech, three had increased arm muscle power, and two had increased leg strength. Segal et al (16) reported a patient with significant improvement in left upper extremity motor function after cranioplasty performed for a surgical defect from a gunshot wound. They suggested that the patient's improvement was most likely due to correction of localized increased intracranial pressure and of re-

duced cerebral blood flow by the release of cortical scarring and restoration of normal calvarial contour. Fodstad et al (17) found significant abnormalities in intracranial pressure in patients who had undergone craniectomy. Langfitt (18) noted that intracranial pressure was elevated in craniectomy patients in the sitting position. None of these reports noted abnormalities of the cervical epidural veins.

Smith and Hodge (19) reviewed abnormalities of the upper cervical spinal cord and emphasized that symptoms of upper cervical cord lesions may include findings of damage to the lower cervical cord. Taylor and Byrnes (6) reviewed high cervical cord compressive lesions and their effects, citing evidence suggesting that compressive lesions in the upper cervical spine can cause venous distention in the cervical gray matter as low as T-1, thereby causing lower motor neuron findings in the upper extremities from venous congestion and resulting damage to spinal cord motor neurons. They concluded that venous drainage from

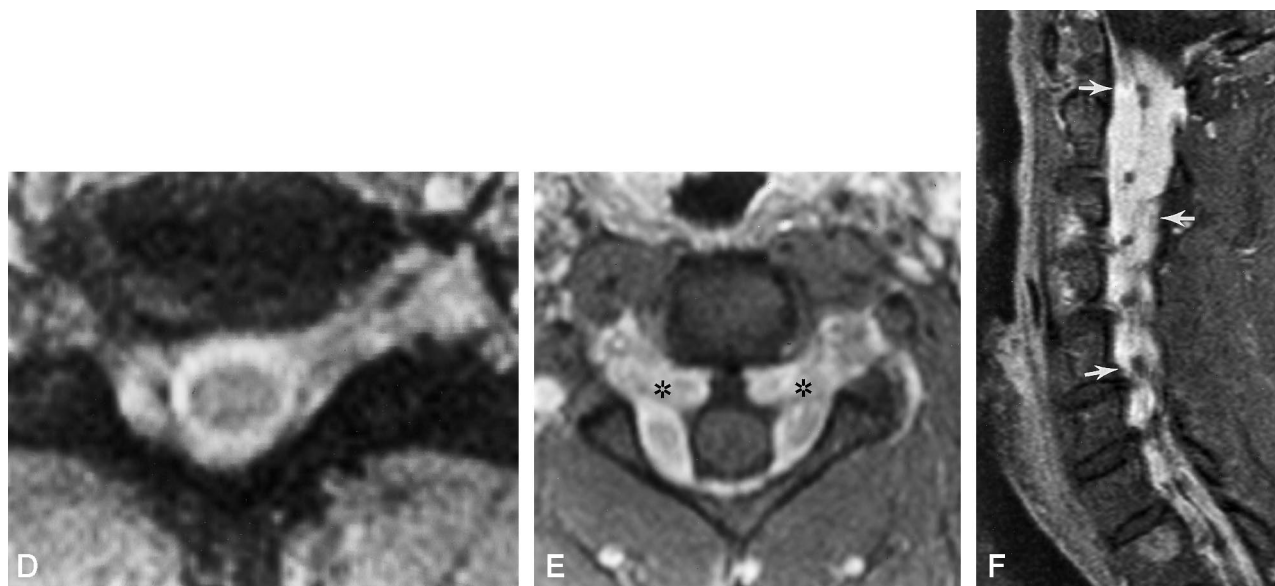


FIG 1. 43-year-old man with contralateral upper extremity weakness and impaired coordination after craniectomy for head trauma.

A, Midline sagittal fast spin-echo T2-weighted MR image shows normal spinal cord signal and contour. Mild degenerative disk disease is present.

B, Axial spin-echo T1-weighted MR image at the C-2 level shows "masses" (arrows) isointense with the spinal cord (asterisk) that largely occupy the spinal canal and that were thought to be greatly dilated epidural veins.

C, Axial gradient-echo T2-weighted MR image at the C-2 level shows the "masses" (asterisks) to be hyperintense relative to the cord and hypointense relative to CSF.

D, On axial gradient-echo T2-weighted MR image at the C7-T1 level, "masses" are much less prominent.

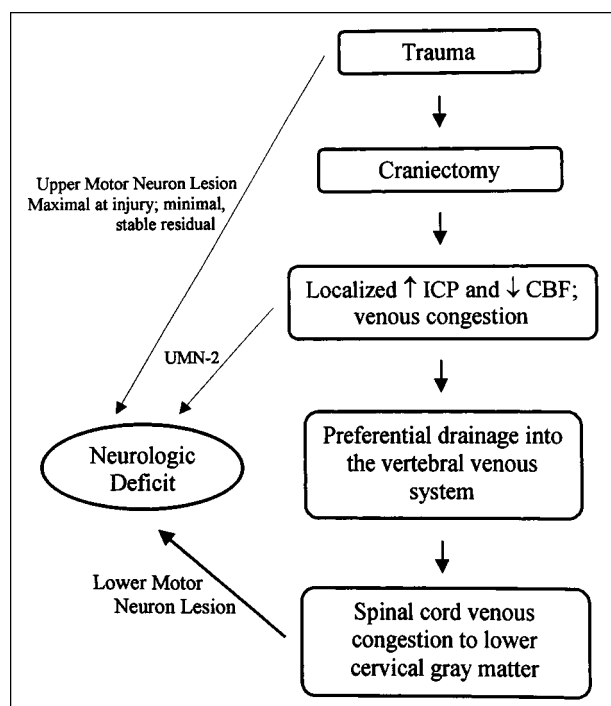
E, Contrast-enhanced, fat-suppressed, axial T1-weighted MR image at the C2-3 level shows intense enhancement (asterisks) extending into the intervertebral foramina, consistent with dilated epidural venous plexus.

F, Contrast-enhanced, fat-suppressed, parasagittal T1-weighted spin-echo MR image shows extension of the venous enlargement (arrows) into the lower cervical region, tapering inferiorly.

G, Digital subtraction catheter angiogram, venous phase of minimally off-lateral oblique projection after a left vertebral artery injection, shows markedly dilated epidural veins (arrowheads) and prominent condylar venous collaterals to the internal jugular veins. Prolonged imaging showed more inferior extent of the dilated epidural veins. No arteriovenous fistula or venous occlusion was discerned.

H, Axial fast spin-echo T2-weighted MR image at the centrum semiovale shows the craniectomy; there is no new lesion in the motor cortex. The brain is compressed by atmospheric pressure. Clinically, the visible scalp concavity was noted to increase in the upright position.

I, Axial gradient-echo T2-weighted MR image at upper C-3 level 10 weeks after cranioplasty shows return of the epidural veins to normal. Right upper extremity clinical findings were markedly improved.



lower cervical cord gray matter is normally superior to the C-2 level (6). The lower motor neuron weakness of the upper extremity that manifested before cranioplasty in our patient is similar to that observed with upper cervical spinal cord lesions.

On the basis of the above information, we hypothesize that our patient's large craniectomy defect allowed compression of his brain in the fixed cranial compartment by atmospheric pressure. This resulted in localized increase in intracranial pressure, alteration of superficial blood vessels, and reduction in cerebral blood flow from increased venous pressure. We believe that this combination of events caused preferential drainage via collateral pathways at the skull base (described in [1]) and the shunting of blood into the epidural and spinal cord venous systems. Venous distension in the cord, as low as the lower cervical cord gray matter, caused dysfunction of lower

FIG 2. Flow diagram for hypothesized pathogenesis of the dilated epidural venous system and predominately lower motor neuron neurologic deficit. ↑ = increased, ↓ = decreased, ICP = intracranial pressure, CBF = cerebral blood flow, UMN-2 = upper motor neuron lesion (minimal, delayed).

spinal motor neurons supplying the muscles of the right upper extremity (Fig 2). Although venous stasis is usually a symmetric process, we propose that the right hand was more affected because of the concurrent cranial lesion. We speculate that the deterioration that occurred later in the patient's recovery phase may have been due to his spending more time in the upright position. This is known to further increase intracranial pressure in craniectomy patients (18) and to cause shunting into the spinal venous system even in healthy persons (2–5). We assume that the venous distension in the cord was insufficient to cause signal changes on MR images. We have no explanation for the absence of previous reports of dilated epidural veins in patients with a large cranioplasty. It may be that this patient had higher intracranial pressure than the typical patient, or it may relate to the location of the defect or to an as yet unrecognized coexisting abnormality. Although impaired cerebral blood flow due to the craniectomy contributed to the upper extremity findings, the increasing weakness with a stable cognitive deficit and the lower motor neuron findings on EMG indicate that cerebral dysfunction was not the main mechanism. Our patient had no initial cervical cross-sectional imaging study (ie, prior to the second admission MR study) to document the precise time of appearance of the enlarged cervical veins; however, disappearance of the enlarged epidural veins after the cranioplasty strongly suggests a causal relationship.

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