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Spinal Manifestations of Intracranial Hypotension

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Spinal Manifestations of Intracranial Hypotension

The intracranial and spinal manifestations of CSF hypovolemia, due either to spontaneous intracranial hypotension (SIH) or post-lumbar puncture headache syndrome (PLPHS), comprise an interesting constellation of imaging findings reflective of underlying pathophysiology. It is now well established that reduced CSF volume and pressure in the presence of closed calvarial sutures result in dilatation of both brain and spinal venous and arterial structures (1). This occurs in an effort to maintain intracranial volume and the relationship between CSF, brain parenchyma, and brain vasculature. This phenomenon, known as the "Monroe-Kelly Rule", forms the basis of our understanding of the imaging manifestations of CSF hypovolemia. As a result, venous dural sinus enlargement, diffuse dural enhancement, spontaneous subdural hygromas and hematomas, as well as spinal epidural fluid collections and dural enhancement, all contribute to replacing the CSF volume lost by either a leak, lumbar puncture, or shunting by ventricular catheters. The spinal manifestations associated with SIH—spinal dural enhancement, spinal epidural venous engorgement, subdural or epidural collections (spinal hygromas), and descent of cerebellar tonsils into the upper cervical subarachnoid space—have been reported by several authors.

Yousry et al, in this issue of the *AJNR* (page 1239), bring to our attention yet another spinal manifestation of CSF hypovolemia, that of an extraspinal CSF fluid collection posterior to the C1-C2 vertebral lamina. In their prospective study of 16 patients with postural headache, CSF collections within the soft tissue posterior to C1-C2 were present in seven patients, three of five patients with SIH and four of 11 patients with PLPHS. These collections diminished in three patients following treatment. Additional findings included dilatation of the anterior internal vertebral plexus in 87.5% and subdural hygromas in 63% of patients. The authors hypothesize that the origin of the C1-C2 fluid collection may either result from actual CSF leakage or transudation from suboccipital veins resulting from "hydrostatic pressure changes similar to those seen with cranial subdural fluid collections". The authors suggest that the C1-C2 region is contiguous with rich venous plexi such as the suboccipital venous plexus, the vertebral artery venous plexus, and the vertebral venous plexus. Consequently, a decrease in CSF volume may lead to both compensatory swelling of the cerebral veins as well as enlargement of veins of the deep cervical venous plexus, resulting in transudation of CSF accumulating within the suboccipital region.

The occurrence of fluid collections at the C1-C2 level among patients with PLPHS argues against the former hypothesis. In my view, neither of these hypotheses make sense. I have also observed the described fluid collection behind the C1-C2 level in a patient with SIH fistula who was found to be leaking from a perineural cyst at the C7 level. In our case, extensive epidural spinal fluid collections were visualized from the C1-C2 level to the T11 level. A CSF fistula was localized at the C7 level only after performing myelography under CT guidance and visualizing the first appearance of contrast medium extravasation. Subarachnoid injection from both the lumbar as well as C1-C2 level demonstrated prompt and florid extension of contrast material into the paraspinal soft tissues at the C1-C2 level. The appearance of contrast at the C1-C2 region argues against the proposed hypothesis of transudation. Our patient was cured after surgical repair of the CSF fistula, resulting in improvement in his clinical symptoms, resolution of the epidural and retrocervical fluid collections, and a return to work. We postulate that epidural CSF ascends within the spinal canal from the site of the leak, escaping from the epidural space and extending into the soft tissues at the C1-C2 level. The epidural CSF does not escape at other spinal levels because it is loculated by epidural fat, which forms a pseudocapsule. Thus, one can think of the epidural space as a "gutter" within the spinal canal, allowing CSF to track up or down the spine. Thus, the location of an epidural collection does not necessarily correlate with the site of the CSF fistula, as CSF may spread within the epidural space several spinal segments away from the site of a leak.

The authors are to be commended for their prospective evaluation and their recognition of the fact that the fluid collection behind C1-C2 does not necessarily represent the site of the CSF fistula. Indeed, the recognition of a fluid collection behind the C1-C2 level in combination with dilatation of the anterior internal vertebral plexus should certainly raise suspicions for the condition of CSF hypovolemia, particularly in a patient with clinical manifestations of postural headache. Other pitfalls include the opacification of the anterior internal vertebral plexus following contrast medium administration. The anterior vertebral plexus can become markedly enlarged in the setting of SIH, and contain flow voids, indent the thecal sac, and displace the dura on either side of the midline. I have seen this misdiagnosed as a meningioma at the foramen magnum. The authors also point out that the syndrome of CSF hypovolemia may occur in the ab-

sence of descent of the cerebellar tonsils or dural enhancement. Given the proper clinical setting, the ancillary findings of CSF hygroma, epidural venous collections, paraspinal fluid collections behind the C1-C2 level, and intracranial and extracranial venous dilatation are clues to the diagnosis. If one remembers that the epidural space is a gutter within which extrathecal CSF can travel long distances from the site of egress, an understanding of the MR imaging appearance of CSF hypovolemia becomes clear.

A search for the leak must first begin with fat-suppressed fast spin-echo imaging of the spine. In the absence of a frank leak, plain-film myelography, performed while the patient is in the decubitus position, is helpful because CT myelography, obtained after a low-dose injection of contrast material, is often insufficient for identification of the site of a small CSF fistula. We've had the occasion to watch the epidural contrast leak several vertebral body segments away from the site of CSF fistula in the course of 3 to 5 minutes. Our approach to these difficult patients who do not respond initially to epidural blood patch include a simultaneous puncture for myelography as well as isotope cisternography. Myelography is performed, preferably from the lumbar region, with attention to the tho-

racic spine. The patient is placed in a decubitus position and cross-table anteroposterior films are obtained every minute for 5 minutes. When the radiologist is satisfied a leak is not occurring, the patient is repositioned on his opposite side, and the films are repeated. CT myelography (3–5 mm) is then performed from the cervical through the lumbar region.

Because the imaging manifestations of SIH have become well known, many patients have been diagnosed who otherwise would have been misdiagnosed with migraine, headache of unknown origin, aseptic meningitis, or subdural hematomas. Attention to the myriad manifestations of CSF hypovolemia both intracranially and extracranially will prevent such errors of diagnosis and facilitate prompt treatment of CSF fistula.

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Diffusion-weighted Imaging as a Surrogate Marker for Stroke as a Complication of Cerebrovascular Procedures and Devices

Ischemic stroke may occur as a complication of any vascular intervention from the heart to the head. These interventions include diagnostic arteriography, coronary revascularization, valve repair, carotid endarterectomy or angioplasty, and endovascular obliteration of aneurysms by use of detachable coils. Nearly all strokes associated with these procedures are attributable to embolic material lodging in distal cerebral arteries.

Efforts to reduce the stroke risk from cerebrovascular interventions are limited by several complicated factors. First, the nature of the embolic material generated during these procedures varies widely and, consequently, the potential for causing ischemic injury to the brain is variable. Embolic material can range from air bubbles, microscopic cholesterol or lipid particles, to larger fragments of atherosclerotic plaque and organized thrombus (1–3). Second, the embolic insult can vary in the total number of particles in the temporal profile of the particle shower. A slow trickle of microscopic particles may have less potential for ischemic injury than the same number of particles delivered in a sudden shower. Third, the potential for an embolic event to result in injury depends on the condition of the cerebral tissue. Given an identical embolic insult, cerebral tissue with reduced perfusion pres-

sure (due to proximal stenosis or occlusion, for example) has a greater risk of permanent ischemic injury than does brain with normal perfusion pressure. Finally, the low frequency of stroke in these procedures makes it difficult to perform studies with adequate power to detect changes in stroke risk with different devices or pharmacological adjuncts.

A surrogate marker for clinical stroke that occurs at a greater frequency would have considerable utility in studies of stroke risk reduction during neurointerventional procedures. At present, there are two complementary methods that have potential for this application: transcranial Doppler (TCD) (4) and, as discussed by Jaeger et al in this issue of the *AJNR* (page 1251), diffusion-weighted imaging (DWI).

The primary advantage of TCD is its capability for real-time monitoring of embolic events during the procedure. Embolic signals, with no clinical sequelae, have been detected by TCD in the middle cerebral artery stem among patients with symptomatic carotid stenosis, artificial heart valves, and polycythemia rubra vera. Asymptomatic signals have also been recorded during diagnostic arteriography, carotid endarterectomy or angioplasty, and endovascular treatment of aneurysms. The sen-