Paradoxical brain herniation

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Paradoxical brain herniation is frequently underestimated. It results from the pressure difference between the atmospheric pressure and the intracranial pressure causing brain shift inward at the craniectomy site.

It is a rare and potentially life-threatening complication of decompressive craniectomy (DC) and results from the combined effects of brain gravity, atmospheric pressure and intracranial hypotension causing herniation in the direction opposite to the site of the DC with subsequent brainstem compression. To date, the cases of PBH reported in literature are spontaneous or provoked by a lumbar puncture (LP), a cerebrospinal fluid shunt or ventriculostomy [1) 2)].

Although rare, paradoxical herniation in the setting of a large craniectomy defect may occur in the absence of cerebrospinal fluid drainage [3)].

This entity should be suspected whenever transtentorial herniation occurs in conjunction with direct or indirect signs of intracranial hypotension. Placing the patient in the Trendelenburg position should be attempted, because this simple maneuver may turn out to be life-saving [4)].

Those treatments for lowering ICP, such as mannitol, CSF drainage, and hyperventilation, all of which follows the Monro-Kellie doctrine will exacerbate paradoxical herniation, because lowering intracranial pressure increases the pressure gradient across the craniectomy defect [5)].

This phenomenon is related to the negative gradient between atmospheric and intracranial pressures, which can be exacerbated by an upright posture, CSF leakage, or dehydration [6)].

Patients who have undergone CSF drainage, such as, external ventriculostomy, ventriculoperitoneal shunt placement, or lumbar puncture are more susceptible to this phenomenon, for these conditions can lower ICP states relatively than that of extra-cranial pressures. In these situations, the brain is sucked down through the tentorial incisural notch essentially and the foramen magnum potentially [7)].

Not surprisingly, the pressure acting over the cerebral cortex may cause neurological deficits. Several authors have claimed that skull defects may create a siphon effect on CSF dynamics, which distorts the dura, underlying cerebral cortex, and venous return, due to scarring and direct pressure to the brain [8) 9)].

Clinical features

Symptoms may include focal deficits, brainstem release signs, autonomic instability, changes in level of consciousness, and pupil changes [10) 11)].

Case reports

2016

Nasi et al present an uncommon case of PBH provoked by percutaneous drainage of a huge subdural hygroma (SH) ipsilateral to the decompressive craniectomy causing mass effect and neurological deterioration. After percutaneous evacuation of SH, the patient became unresponsive with dilated and fixed left pupil. A brain CT scan showed marked midline shift in the direction opposite to the craniectomy site with subfalcine herniation and effacement of the peripontine cisterns. Paradoxical brain herniation (PBH) was diagnosed. Conservative treatment failed and the patient required an emergency cranioplasty for reverse PBH.

The present case highlights the possibility that all forms of CSF depletion, including percutaneous drainage of subdural CSF collection and not only CSF shunting and/or lumbar puncture, can be dangerous for patients with large craniotomies and can result in PBH. Moreover, an emergency cranioplasty could represent a safe and effective procedure in patient not responding to conservative treatment [12)].

2015

Two patients who underwent decompressive craniectomy after head trauma deteriorated secondary to paradoxical herniation, one after lumbar puncture and the other after ventriculoperitoneal shunting. They motivated the authors to investigate further provoked paradoxical herniation.

The authors reviewed the records of 205 patients who were treated at a single hospital with decompressive craniectomy for head trauma to identify those who had had lumbar puncture performed or a ventriculoperitoneal shunt placed after craniectomy but before cranioplasty. Among the patients who met these criteria, those with provoked paradoxical herniation were identified. The authors also sought to identify similar cases from the literature. Exact binomials were used to calculate 95% CIs.

None of 26 patients who underwent a lumbar puncture within 1 month of craniectomy deteriorated, whereas 2 of 10 who underwent a lumbar puncture 1 month afterward did so (20% [95% CI 2.4%-55.6%]). Similarly, after ventriculoperitoneal shunting, 3 of 10 patients deteriorated (30% [95% CI 6.7%-65.2%]). Timing of the procedure and the appearance of the skin flap were important factors in deterioration after lumbar puncture but not after ventriculoperitoneal shunting. A review of the literature identified 15 additional patients with paradoxical herniation provoked by lumbar puncture

and 7 by ventriculoperitoneal shunting.

Lumbar puncture and ventriculoperitoneal shunting carry substantial risk when performed in a patient after decompressive craniectomy and before cranioplasty. When the condition that prompts decompression (such as brain swelling associated with stroke or trauma) requires time to resolve, risk is associated with lumbar puncture performed ≥ 1 month after decompressive craniectomy [13].

2014

A 56-year-old woman with no interesting medical history, who, after an olfactory groove meningioma surgery, presented a haemorrhage located in the surgical area with an important oedema. The patient required a second emergency surgery without any chance of conserving the cranial vault. During the post-operational period, great neurological deterioration in orthostatic position was noticed, which resolved spontaneously in decubitus. This deficit was resolved with bone replacement afterwards [14]

2012

A 38-year-old man underwent decompressive craniectomy for severe brain swelling. He remained neurologically stable for five weeks, but then showed mental deterioration right after a lumbar puncture which was performed to rule out meningitis. A brain computed tomographic scan revealed a marked midline shift. The patient responded to the Trendelenburg position and intravenous fluids, and he achieved full neurologic recovery after successive cranioplasty [15].

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