CASE REPORT

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Paradoxical Herniation after Decompressive Craniectomy for Acute Subdural Hematoma

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Decompressive craniectomy is usually performed to relieve raised intracranial pressure(ICP) caused by various intracranial lesions. A 67-year-old man presented with acute subdural hematoma and traumatic intracerebral hematoma. The patient underwent a decompressive craniectomy. Four weeks later, the patient presented with acute neurological deterioration. Brain computed tomographic(CT) scans revealed the marked concavity of the brain at the site of the craniectomy and associated with midline shift which was reversed by cranioplasty. We report an unusual case of cerebral herniation from intracranial hypotension after decompressive craniectomy for a traumatic subdural hematoma. The cranioplasty may be helpful to prevent paradoxial cerebral herniation.

KEY WORDS : Cerebral herniation · Cranioplasty · Decompressive craniectomy · Subdural hematoma.

Introduction

Decompressive craniectomy is often performed to relieve increased intracranial pressure(ICP) in patients suffering from head injury, stroke, or postoperative complications[16].

The syndrome of the sinking skin flap, suggested by Yamamura[15] in 1977, could explain the neurologic deterioration following decompressive craniectomy. Also the term paradoxical herniation has been suggested to describe this phenomenon[8,11]. In this situation, the skull defect created a siphon effect to the cerebrospinal fluid(CSF) dynamics and the atmospheric pressure was directly transmitted to the intracranial cavity, causing neurological deficits[2,6,14]. After craniectomy, transtentorial herniation is possible even in the absence of increased ICP. It is related to the negative gradient between atmospheric and intracranial pressures, which is enhanced by changes in the CSF compartment following upright position, CSF leakage or dehydration[11]. However, the neurological deterioration by the cerebral herniation following decompressive craniectomy has been less well known in lirteratures with regard to the neurological improvement following cranioplasty.

The authors report an unusual case of spontaneous transtentorial herniation after decompressive craniectomy and discuss possible mechanisms with review of the literatures.

Case Report

A 67-year-old man presented with mental deterioration following head injury. On admission, he was stuporous with motor weakness on the left side. Brain compurted tomographic(CT) scans revealed a large amount of subdural hematoma on the right frontotemporoparietal region and traumatic intracerebral hematoma in the left frontal region (Fig. 1). The

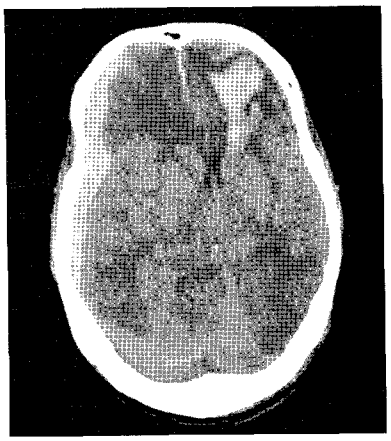

Fig. 1. Preoperative axial computed tomographic(CT) scan showing the acute subdural hematoma on the right frontotemporoparietal region and intraparenchymal hemorrhage in the left frontal lobe with brain shift to the left.

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patient was undertaken right frontotemporoparietal craniectomy with evacuation of the hematoma and a left frontal craniotmy with evacuation of intracerebral hematoma. The patient was improved gradually after operation. He was awake and able to speak a few words, and able to follow simple commands. Follow-up brain CT scans showed a resolution of previous subdural hematoma and recovery of midline shift (Fig. 2). But 4 weeks later, the patient was found obtunded and unresponsive to noxious stimuli, with bilaterally dilated, non reactive pupils. Emergent CT scan was obtained and revealed the marked concavity of the brain at the site of the skull defect with associated midline shift to the left (Fig. 3). A presumptive diagnosis was cerebral herniation secondary to intracranial hypotension. Adequate hydration was followed by cranioplasty. He recovered gradually in a week and brain CT scan after cranioplasty showed the restoration of the shifted midline(Fig. 4).

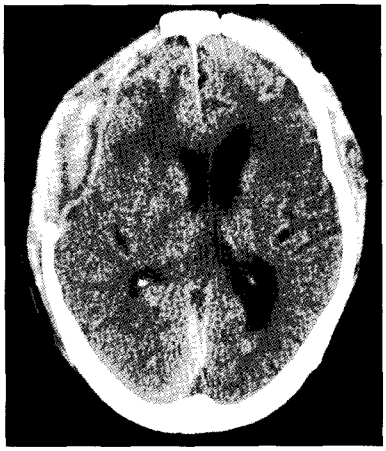

Fig. 2. Postoperative axial CT scan 2 weeks later showing the resolved hematoma in the right frontotemporoparietal region and left frontal lobe. Note the restoration of the shifted midline.

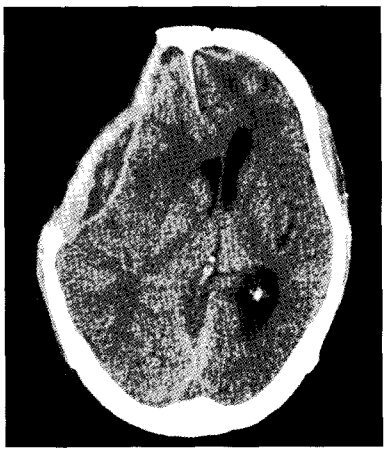

Fig. 3. Postoperative axial CT scans 4 weeks later showing the marked concavity of the brain at the site of the craniectomy with associated midline shift to the left.

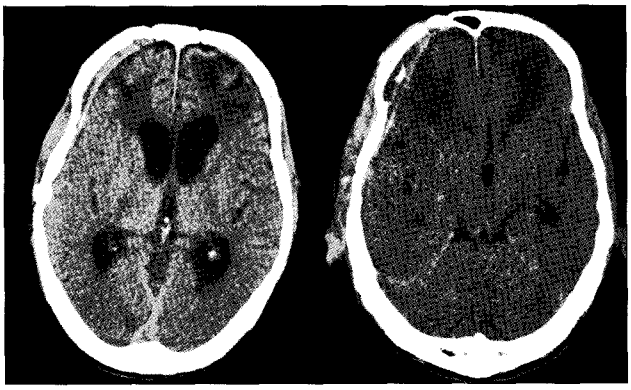

Fig. 4. Second postoperative axial CT scans showing the restoration of the shifted midline after cranioplasty.

Discussion

In patients with craniectomy, the cranium do not maintain a rigid structure. Therefore, the Monro-Kellie doctrine that the sum of volumes of brain, CSF, and intracranial blood is constant, can not be applied as it is stands[1]. Another fator, atmospheric pressure, can influence this equilibrium. Increased transmission of atmospheric pressure will decrease volume of brain or CSF in patients with craniectomy. This phenomenon will be exacerbated by upright position, CSF leakage or dehydration, causing shift of brain from supratentorial compartment to infratentorial one.

The first pathophysiologic explanation for this phenomenon was suggested by Gardner et al.[3] in 1945, who claimed that, unlike the brain in the closed calvaria, the brain pulsated with every alteration of arterial or venous pressure in a trephined skull. In 1968, Langfitt[6] suggested that the atmospheric pressure was transmitted to the intracranial cavity, causing inward rotation of the scalp over the cranial defecits. This pressure acting over the cerebral cortex may cause neurological deficits. Some authors[2,9,12] claimed that the skull defect creates a siphon effect on the CSF dynamics related to the distortion of the dura, underlying cortex, and venous return by scarring and direct pressure.

There have been a few studies about the neurological improvement after cranioplasty in patients with craniectomy. Richaud et al.[9] reported that in their all cases, there was a 15 to 30% increase in cerebral blood flow in the area of cortex adjacent to the cranioplasty. They proposed restoration of normal cerebral hemodynamics as a mechanism for neurological recovery after cranioplasty. In 1985, Stula[13] suggested that the atmospheric pressure acting on the unprotected brain, produced brain compression and cranioplasty normalized this situation. Segal et al.[12] reported a case that the patient improved significantly in motor function after cranioplasty, emphasizing on the restoration of local hemodynamics under the altered pressure and removal of scarring.

Since Yamamura et al.[15] named the phenomenon "the syndrome of the sinking skin flap", there were many reports about treatment of patients with neurological deterioration following craniectomy or craniotomy. Tabaddor et al.[14] reported a case with severe sinking at the skull defect and contralateral hemiparesis after craniectomy, and the patient was recovered after cranioplasty. They believed that neurological improvement following cranioplasty was due to the relief of the pressure gradient between the atmosphere and the intracranial space. In 1997, Schiffer et al.[10] reported five cases of this syndrome

that were improved by cranioplasty and recommended early prophylactic cranioplasty in cases with large, concave, or fluctuating skull defects, especially if any neurological deterioration occurs without an apparent cause. Some authors[4,5,7,8] claimed that lumbar puncture should be avoided in patients with craniectomy or craniotomy, because a position-dependent transtentorial herniation of the brain might occur. The maneuver of placing a patient in the Trendelenburg position or intrathecal saline infusion with aggressive intravenous fluid resuscitation could be potentially life saving in this situation. In our case, we postulated that aggressive exercises consisting of mobilization and upright positioning may have adversely affected the underlying process, and the negative pressure gradient between atmospheric and intracranial pressure might cause cerebral herniation. After we performed cranioplasty and adequate rehydration, the good outcome was achieved in a patient who was deteriorated neurologically.

Conclusion

We report a rare case of paradoxical cerebral herniation after decompressive craniectomy. It should be kept in mind that this herniation may cause neurological deterioration after craniectomy, and early cranioplasty with adequate hydration warrants good outcome.

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