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CASE REPORT

Cerebral hemorrhagic infarction following cranioplasty in a shunted patient with tension pneumocephalus resulting from depressed skull and craniodural defect



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cerebral infarction;
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Summary A 34-year-old female sustained a severe traumatic brain injury that was treated with decompressive craniectomy and subsequent cranioplasty, then with ventriculoperitoneal shunt about 10 years previously. However, the skull flap was found to be depressed ever since. She was admitted to our hospital for a headache and left hemiparesis with sudden onset. The computed tomography scan displayed tension pneumocephalus in the right frontoparietal region. First, she underwent emergency burr hole drainage and placement of a subdural drain with external ventricular drainage tube. Then her symptoms improved considerably. Unfortunately, 6 months later she was admitted again to our hospital because of headache and left hemiparesis with sudden onset, and the brain computed tomography showed tension pneumocephalus in the right frontoparietal region. She underwent craniectomy to remove the previous depressed skull and simultaneous cranioplasty with Ti-Mesh. On the day of her operation, generalized seizure occurred and her consciousness deteriorated. The magnetic resonance imaging showed hemorrhagic infarction on both sides of the thalamus and the right parieto-occipital region. We think it probable that a sudden increase of cerebral blood flow in the

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cerebral hemisphere where the cranioplasty had been performed caused reperfusion injury and resulted in hemorrhagic infarction.

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1. Introduction

Pneumocephalus, defined as the presence of air in the cranial cavity, most commonly occurs after a head injury or a neurosurgical procedure.¹ Tension pneumocephalus can occur when air becomes entrapped, causing compression of the brain and resulting in severe neurological deficits. Very rarely, it was reported to occur as a complication of ventriculoperitoneal (VP) shunting for hydrocephalus secondary to a variety of etiologies.^{2–4} In this case report, we report the occurrence of cerebral hemorrhagic infarction following cranioplasty in a shunted patient who presented with left hemiparesis, resulting from tension pneumocephalus caused by a depressed skull flap and open scalp wound. The possible mechanisms of tension pneumocephalus and postcranioplasty cerebral hemorrhagic infarction are discussed.

2. Case report

A 34-year-old female visited our emergency room because of severe headache and vomiting for 3 days. The headache was aggravated when she lay down. Tracing back her history, we found that she had sustained severe traumatic brain injury treated by decompressive craniectomy and subsequent cranioplasty, and then VP shunting 10 years earlier. After that event, she did rather well, although she reported mild weakness in the left leg. However, depression of the skull flap was noted thereafter.

On arrival at our emergency room, she was afebrile. Her pulse rate and blood pressure were within normal limits. On examination, her Glasgow Coma Scale score was E4V5M6. Her pupils were equal in size and reactive to light. Mild left leg weakness was noted. There was a tiny wound at the site of the previous old surgical scar of the right depressed temporal scalp. A brain computed tomography (CT) scan showed tension pneumocephalus in both frontal regions (Fig. 1A). Cerebrospinal fluid (CSF) studies, obtained from the VP shunt reservoir, revealed no evidence of infection. Emergency burr hole drainage and subsequent placement of a subdural drain for external ventricular drainage were done. Simultaneously, the scalp wound was debrided and closed primarily. After the operation, her headache improved markedly. A repeated CT scan 10 days later demonstrated good resolution of tension pneumocephalus (Fig. 1B). The wound culture and CSF culture showed no bacterial growth. The patient recovered uneventfully and remained well during the 3-month follow-up.

About 6 months later, she was admitted again to our hospital for sudden headache and left hemiparesis. On examination, her Glasgow Coma Scale score was E4V5M6. Her pupils were equal in size and reactive to light. The muscle power of the left-side limbs was grade 3. There was a tiny wound in the previously debrided scalp. The brain CT displayed a tension pneumocephalus in the right frontoparietal region (Fig. 2). She underwent craniectomy to remove the previous depressed skull and simultaneous cranioplasty with Ti-Mesh (Medtronic, Minneapolis, MN, USA). The surgical procedure was uneventful. On the day of the operation,

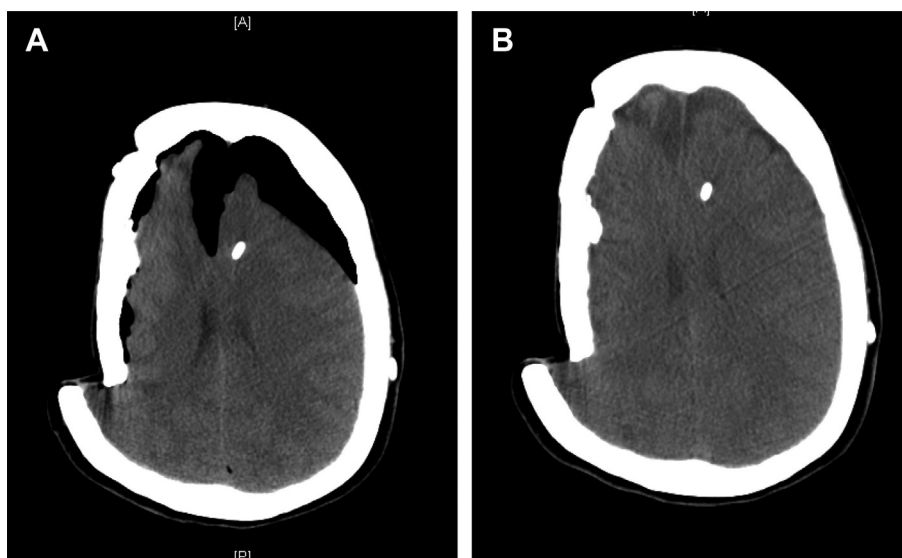


Figure 1 (A) Brain computed tomography showing tension pneumocephalus in both frontal regions. (B) Computed tomography 10 days later demonstrated good resolution of tension pneumocephalus.



Figure 2 Brain computed tomography displaying tension pneumocephalus in the right frontoparietal region.

generalized seizure occurred and her consciousness deteriorated after the attack. The magnetic resonance imaging showed infarction—hemorrhage on both sides of the thalamus and the right parieto-occipital region (Fig. 3). She underwent rehabilitation and recovered gradually with residual left hemiparesis and dysphasia 3 months later.

3. Discussion

Based on a literature review, it is stated that there are two requirements for tension pneumocephalus to develop after a VP shunt: the presence of a CSF diversion system causing a decrease in intracranial pressure and the existence of a craniodural defect.⁴ In the presence of a craniodural defect, the negative pressure resulting from the siphoning

effect of the shunt will allow the entry of trapped air into the brain, causing pneumocephalus. We think these mechanisms can explain the occurrence of tension pneumocephalus in our case.

The mechanism of the cerebral hemorrhagic infarction following cranioplasty for depressed skull is not clear in our case. Cecchi and colleagues⁵ reported a case of hemorrhagic infarction occurring after an autologous cranioplasty in a patient with sinking flap syndrome. They thought that a sudden increase of cerebral blood flow in the cerebral hemisphere where the cranioplasty had been performed caused reperfusion injury because the autoregulation might have been impaired during the previous stroke insult. In contrast to the patient of Cecchi et al,⁵ who experienced ipsilateral hemispheric hemorrhagic infarction, our patient developed bilateral hemorrhagic infarction.

Eom et al⁶ reported a patient with right internal carotid artery occlusion who underwent intra-arterial thrombolysis with urokinase and subsequent decompressive craniectomy, and then had diffuse hemorrhagic infarction following cranioplasty. However, the patient had not recovered from anesthesia and became comatose. A cranial CT showed increasingly diffuse brain swelling and multifocal hemorrhagic transformation in both frontal lobes and basal ganglia. They then stated that deep cerebral venous occlusion or congestion was the possible cause for the bilateral hemorrhagic infarction. This patient underwent decompressive craniectomy for a large cerebral infarction, and all these might have diffusely damaged the small intracranial vessels. When collaterals are insufficient, cranioplasty can cause a rapid increase in cerebral blood flow and blood volume bilaterally in the chronic dysfunctional brain, resulting in venous stasis, secondary venous congestion and thrombosis, and subsequent extensive intracerebral hemorrhagic infarction. To sum up the above-mentioned opinion, we think that the explanation of Eom et al⁶ may be applied to our case.

In conclusion, although the exact pathogenic mechanism of bilateral hemorrhagic infarction following cranioplasty

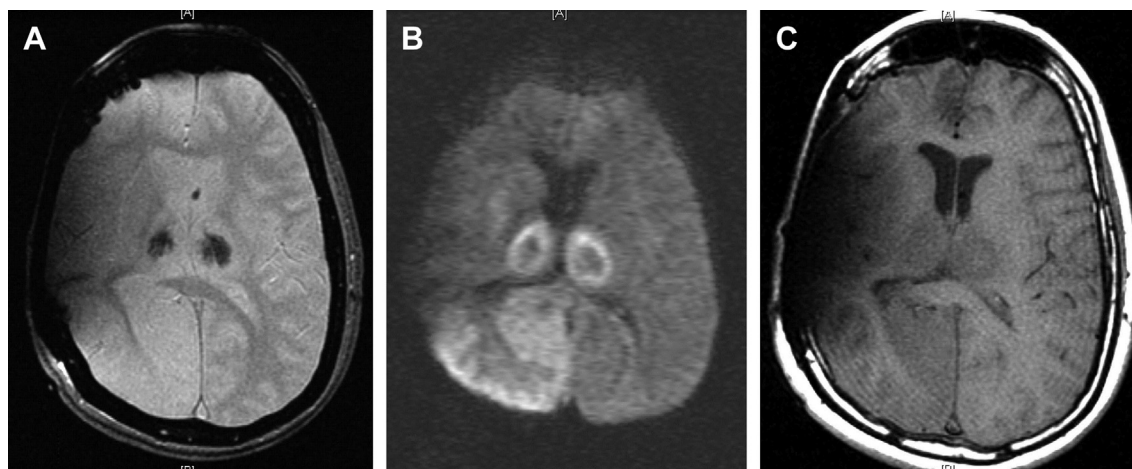


Figure 3 (A) Magnetic resonance (MR) imaging study on T2 weighted gradient—echo images showing marked hypointensity on both sides of the thalamus. (B) Diffusion-weighted MR image revealing a lack of diffusion restriction within both sides of the thalamus and right occipital lobe. (C) Axial images, T1-weighted image, showing hypointensity lesion in the thalamus bilaterally, and in the right parieto-occipital lobe.

remains ambiguous, the possibility of serious complications in such patients should be kept in mind. We have to conduct further research to prevent this kind of serious complications from occurring.

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