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Case Report

Ischemic cerebral and cerebellar strokes simultaneously following supratentorial cranioplasty: case report

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Abstract: Background: Several complications may take place following supratentorial cranioplasty, such as poor wound healing, scalp infection, epilepsy, bone flap resorption and cosmetic dissatisfaction. However, it is rare that ischemic cerebral and cerebellar strokes can simultaneously happen to patients following supratentorial cranioplasty. We present one case of ischemic cerebral and cerebellar strokes following the right supratentorial cranioplasty simultaneously. Case description: A male of 55-year-old suffered right temporal-parietal subdural hematoma and cerebral contusion and laceration after a sudden traffic accident in October 2016. Decompressive craniectomy was carried out for remitting high intracranial pressure in our hospital. In February 2017, the patient underwent the right temporal-parietal titanium mesh cranioplasty in our Department of neurosurgery. The male had no occurrence of surgery-associated adverse events intraoperatively. However, the consciousness of this patient did not return to normal and the onset of epilepsy had three times postoperatively. Six hours later, postoperative CT scan shown ischemic strokes in the right cerebral and the left cerebellar lobes. Computed tomography angiography (CTA) and Transcranial Doppler (TCD) indicated that the vascular status of the right middle cerebral artery and the left vertebral artery was poor. 7 hours after the cranioplasty, the patient was given the left suboccipital decompressive craniectomy. Medical measures concerning antiplatelet aggregation and improvement of cerebrovascular microcirculation were taken following posterior fossa decompression. The patient was discharged with normal consciousness and 0 grade of muscle tone in the left limbs. The left limbs muscle tone was restored to grade 5 after six months of follow-up. Conclusions: We present a rare case of ischemic cerebral and cerebellar infarction following supratentorial cranioplasty. CTA and TCD were applied to examine the cerebral-vascular abnormalities, reperfusion and venous stasis for Infratentorial and supratentorial infarction following supratentorial cranioplasty. The three major causes should be taken into considerations when assessing the complications of cranioplasty.

Keywords: Cranioplasty, postoperative complication, infarction

Introduction

Cranioplasty is regarded as an effective measurement for cosmetic improvement and neurological protection following decompressive craniectomy (DC). However, several complications associated with cranioplasty are extremely troublesome, including poor healing of incision, postoperative infection, epilepsy, intracranial hematoma, imperfect appearance even accidental death owing to diffuse cerebral edema [1, 2]. It is rare that patients following supratentorial cranioplasty undergo ischemic cerebral and cerebellar strokes simultaneously. We report a male of 55-year-old underwent ischemic strokes following supratentorial cranioplasty who suffered DC for right temporal-parietal subdural hematoma and cerebral contusion and laceration after a sudden traffic accident. In our present study, we aim to share the possible mechanisms concerning the rare postoperative strokes following cranioplasty.

Case history

A male of 55-year-old suffered right temporal-parietal subdural hematoma and cerebral contusion and laceration after a sudden traffic accident in October 2016. Decompressive craniectomy in the injured area was carried out for

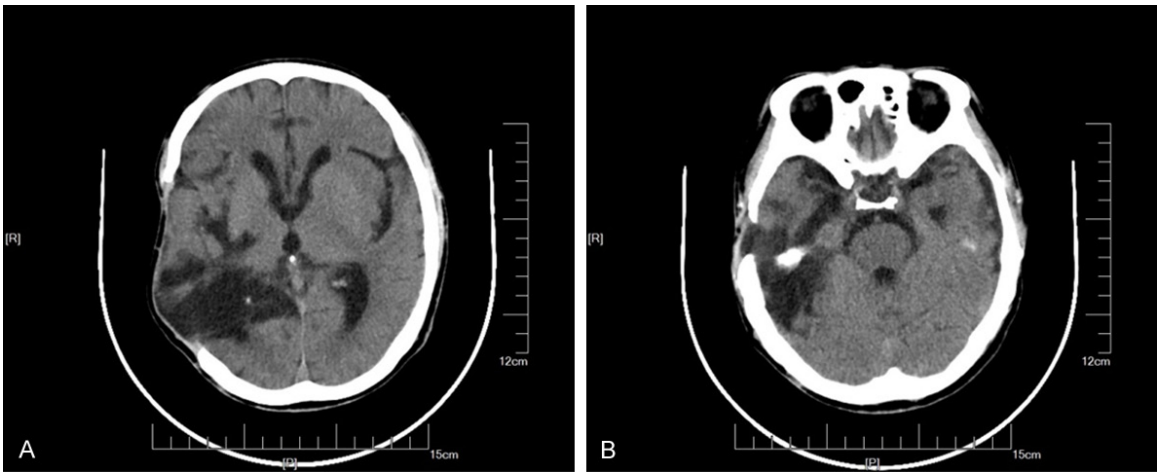

Figure 1. Decompressive craniectomy was carried in the injured right temporal-parietal areas (A, B).

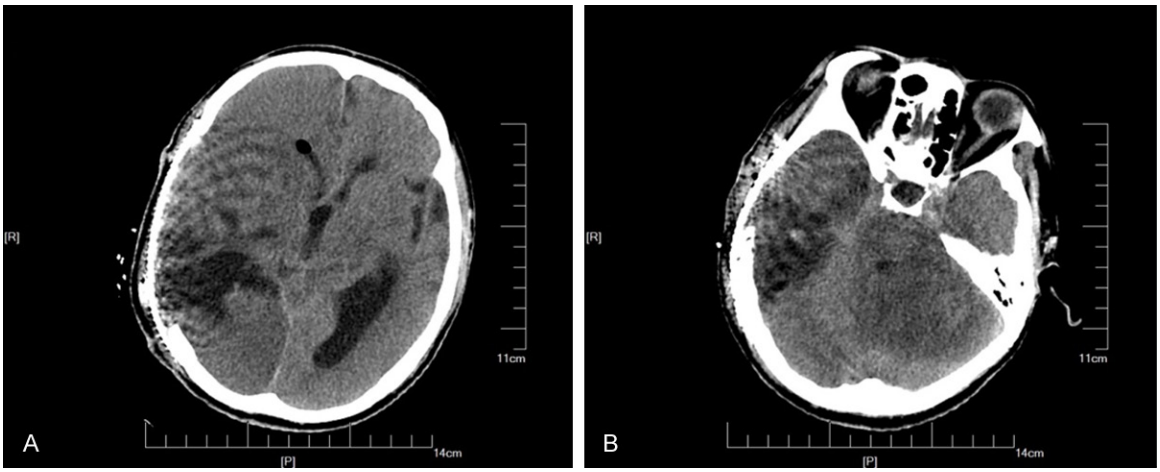

Figure 2. Six hours later, postoperative immediate CT scan shown hypo-dense lesions in the right cerebral (A) and the left cerebellar lobes (B).

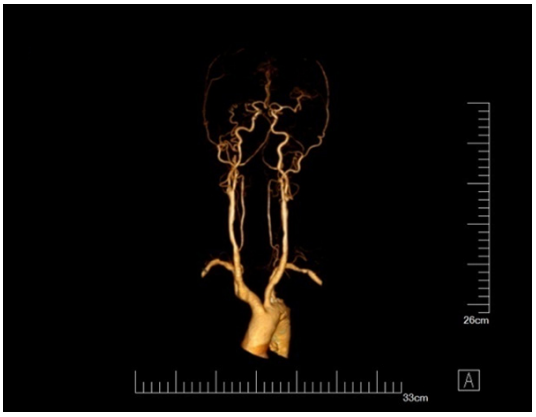

Figure 3. Computed tomography angiography (CTA) revealed the vascular status of the right middle cerebral artery and the left vertebral artery.

treating high intracranial pressure in our hospital (**Figure 1A**, **1B**). The process was uneventful and the patient discharged with good consciousness and 1 grade of the left limbs muscle tone.

In February 2017, the patient was admitted again and his data of 3D CT reconstructions of cranial defect was collected. He underwent the right temporal-parietal cranioplasty with titanium mesh implant according pre-operative data of 3D CT reconstructions in our Department of neurosurgery. The male had no occurrence of surgery-associated adverse events intra-operatively. However, the consciousness of this patient did not return to normal and the onset of epilepsy had three times postoperatively. Six

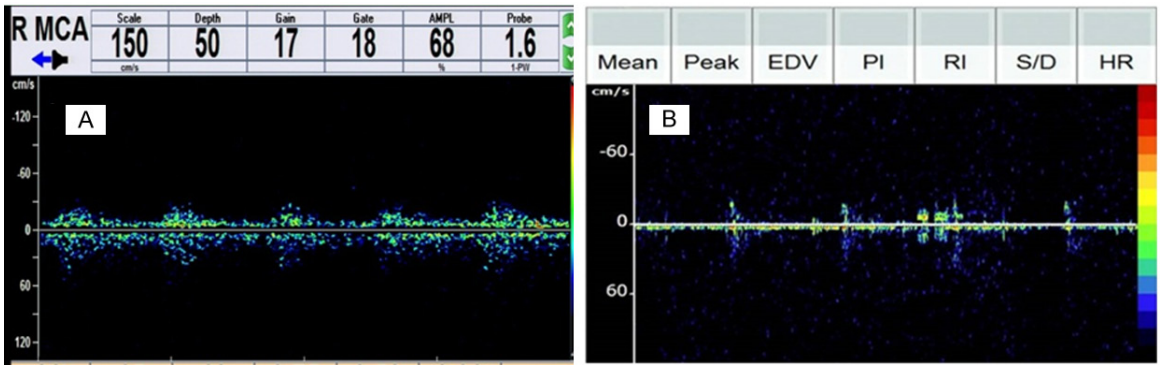

Figure 4. TCD shown the subtotal occlusion of the right middle cerebral artery (A) and the left vertebral artery (B).

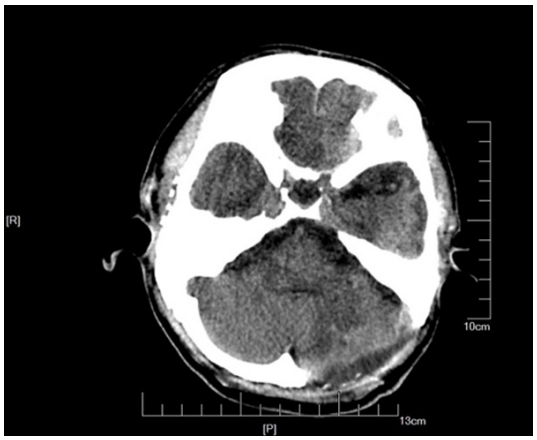

Figure 5. Decompressive craniectomy in the left sub-occipital area.

hours later, postoperative immediate CT scan shown hypo-dense lesions in the right cerebral and the left cerebellar lobes (**Figure 2A**, **2B**). Computed tomography angiography (CTA) (**Figure 3**) and Transcranial Doppler (TCD) indicated that the vascular status of the right middle cerebral artery (**Figure 4A**) and the left vertebral artery (**Figure 4B**) was poor. But no vessel spasms occurred. The subtotal occlusion of the right middle cerebral artery occurred. The inner diameter of the left vertebral artery became smaller and its blood flow volume decreased. 7 hours after the cranioplasty, the patient was given the left sub-occipital decompressive craniectomy (**Figure 5**). Further CT scan proved satisfactory outcomes of posterior fossa decompression and the final infarct size became smaller.

The patient had no history of epilepsy, hypertension, hyperlipidemia, diabetes mellitus, heart disease (e.g. atrial fibrillation, arhythmia) and thrombus of lower extremity. Further, intra-operative carotid artery injuries and low cerebral blood flow, such as hypotension did not occur. Medical measures concerning antiplatelet aggregation and improvement of cerebrovascular microcirculation were taken following posterior fossa decompression. The patient was discharged with normal consciousness and 1 grade of the left limbs muscle tone two weeks after cranioplasty.

Follow up

The left limbs muscle tone was restored to grade 5 after six months of follow-up. For economic reasons, the patient did not review the imaging data of the central nervous system.

Discussion

Cranioplasty is commonly regarded as a safe and straight procedure for skull reconstruction due to decompressive craniectomy [3]. However, several severe complications including ischemic strokes, influenced prognosis following cranioplasty. Several factors concerning ischemic cerebral and cerebellar strokes following supratentorial cranioplasty should be paid more attention: hypo-perfusion, carotid and vertebral arteries lesion or injuries, cerebral or cerebrovascular disfunction or self-regulation, dys-autonomia.

Hypo-perfusion was firstly regarded as the most probable reason associated with ischemic strokes following supratentorial cranioplasty [4]. Inter-operative hypotension or hypo-oxygen could be the main cause of cerebral or

cerebellar ischemia [5]. However our anesthesiologist colleagues suggested that there were no hypotension or respiratory dis-function interoperatively. Furthermore, the patient suffered no pre-operative and post-operative hypotension or hypo-oxygen. However, neurological imaging revealed cerebral and cerebellar ischemia post-operatively. The two factors could not explain ischemic strokes in the right cerebral and the left cerebellar lobes. Usually, watershed infracts and focal ischemic injuries occurred with exception of cerebral and cerebellar ischemia post-operatively.

Carotid and vertebral arteries or deep veins lesions and injuries may be responsible for the ischemic event [4, 6]. In fact, the traffic accident and further decompressive craniectomy were not the culprit of carotid and vertebral arteries lesions or injuries in the present patient. In other words, there was no evidence that the two factors led to cererovascular injuries. Furthermore, in the process of cranioplasty, the patient suffered no positional stenosis of the right middle cerebral and vertebral arteries intra-operatively. After all, the male was turned his head towards the opposite less than 30 degrees. However, the postoperative imaging of CTA and TCD of the patient revealed subtotal occlusion of the right middle cerebral and the left vertebral arteries following cranioplasty. What was worse, he suffered ischemic strokes in the right cerebral and the left cerebellar lobes. Deep vein thrombosis may happen to patients following cranioplasty [3]. However, the patient had no history of hypertension, hyperlipidemia, diabetes mellitus, heart disease (e.g. atrial fibrillation, arhythmia) and thrombus of lower extremity.

Abnormal self-regulation and re-equilibration of intracranial vessels was another major factor. Because of severe cerebral or cerebellar injuries, intracranial vessels tended to abnormal self-regulation and re-equilibration [7]. The ability of self-regulation of intracranial vessels suffered severe injury following brain trauma or cerebro-vascular strokes [8]. However, in cerebro-vascular microcirculation, the reperfusion increased suddenly, which can be the reason of hemorrhage or infarction [4, 9]. Owing to re-equilibration of intracranial pressure and the atmospheric pressure following cranioplasty, the atmospheric pressure imposed on brain disappeared. Intracranial vessels, including infratentorial and supratentorial vessels underwent improper extension and damages, which led to dramatic decrease of cerebral or cerebellar blood supply and further infarction [10]. However this theory is controversial and need further arguments.

In all, only hypo-perfusion, carotid and vertebral arteries lesions or injuries, cerebral or cerebrovascular disfunction or self-regulation, dys-autonomia were taken considerations, can neurosurgeons reduced the rate of ischemic cerebral and cerebellar infarction effectively following supratentorial cranioplasty.

Conclusion

We present a rare case of ischemic cerebral and cerebellar infarction following supratentorial cranioplasty. Hypo-perfusion, carotid and vertebral arteries lesions or injuries, cerebral or cerebrovascular disfunction or self-regulation, dys-autonomia may be major culprits associated with the ischemic stroke. Neurosurgeons should pay more attention to the above reasons for reducing the rate of ischemic cerebral and cerebellar infarction effectively following supratentorial cranioplasty. Owing to limited cases, the mechanism concerning the rare infarction need further investigated.

Disclosure of conflict of interest

None.

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