

Reperfusion Injury after Cranioplasty

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Intracerebral hemorrhagic transformation of the gliotic area after cranioplasty is a very rare complication with only five reported cases to date.^{1–5} After obtaining informed written consent from the next of the kin, we present the case of a young male adult who suffered from bleeding involving the left cerebral hemisphere following cranioplasty.

A 30-year-old man presented at our hospital with past history of decompressive craniectomy done 6 months before due to left-sided acute subdural hematoma with left frontal contusion. On admission, he was conscious without any neurologic deficit. He was posted for elective autologous bone flap replacement. Preoperative computed tomographic (CT) scan showed craniectomy defect and gliosis at the site of previous site of injury (→Fig. 1). He underwent cranioplasty under general anesthesia. Surgery was uneventful, and the dura was not breached. His postoperative

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recovery was uneventful and shifted to the ward with an Aldrete score of 10. Two hours postoperatively, the patient started becoming drowsy and developed right-sided weakness with slurring of speech. Immediate CT scan was done, which revealed hemorrhagic transformation of the previous gliotic area (→Fig. 2). Considering the drop in Glasgow coma score (GCS) from 15 to 8, he was intubated and was taken to the operating theater for bone flap removal. Following the reexploration, he was ventilated overnight and extubated the next day. He was again conscious and obeying commands. Antiedema measures were continued for few more days along with antiepileptics. He was discharged in a conscious state with right-sided weakness and dysphasia. Transmission of atmospheric pressure decreases the volume of the brain and cerebrospinal fluid as the cranium is no longer a closed cavity after craniectomy.⁶ Improvement in

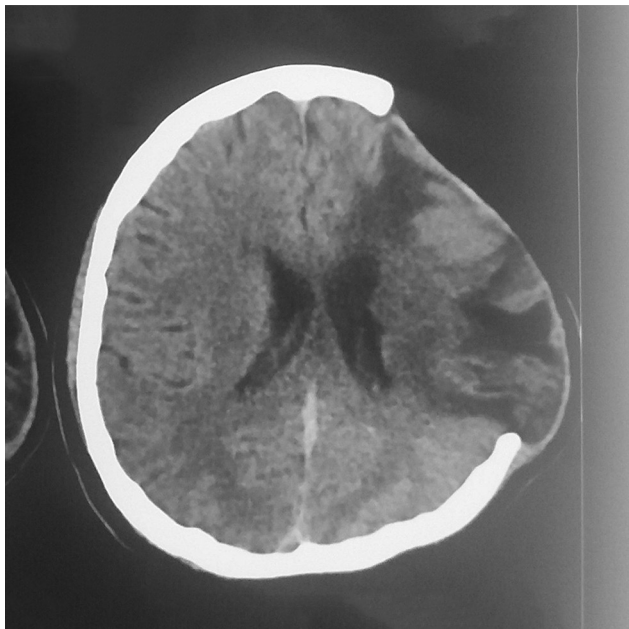


Fig. 1 Preoperative CT showing craniectomy defect and gliosis at the site of previous site of injury. CT, computed tomography



Fig. 2 Immediate CT showing hemorrhagic transformation of the previous gliotic area. CT, computed tomography

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cerebrovascular reserve capacity and glucose metabolism has been shown by transcranial Doppler and 18-fluorodeoxyglucose positron emission tomography.⁷ Yoshida et al reported significant increase in CBF (cerebral blood flow) and cerebral energy metabolism after cranioplasty by using ¹³³Xenon CT and ³¹P magnetic resonance spectroscopy.⁸ Also, increase in bilateral CBF has been demonstrated by CT perfusion studies.⁹ Hemorrhagic infarction has been reported as a complication of cranioplasty procedure. In our patient, hemorrhagic transformation in the gliotic area happened after cranioplasty, on the same side of operation. As there was no apparent injury either to a blood vessel or dura, mechanical cause of bleeding can be ruled out. Hence, our inference is that hemorrhagic transformation is related to the increased CBF to the operated side. Infra- and supratentorial hemorrhagic infarctions have been reported after cranioplasty.² By multiple neuroimaging, reperfusion, vessel injury, and venous stasis after cranioplasty have been held as possible mechanisms for such unique complication.³ In our patient since there was no infarction but only hemorrhage, we propose that rapid increase in bilateral CBF and volume in the chronic dysfunctional brain with questionable status of autoregulation probably resulted in venous stasis and congestion that may have increased the risk of hemorrhage.² It is a rare complication of cranioplasty, but also of worth considering in patients who develop neurologic defect after cranioplasty.

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