



Brain Swelling after Cranioplasty and Aneurysm Clipping. A Rare Complication. Case Report and Literature Review

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Abstract

Background: The malignant brain edema is the most serious complication of cranioplasty and has an un-favorable prognosis, including death. It presents only in a 1% of the cases.

Clinical Case: 18-year-old male with decompressive craniectomy and ventriculo-peritoneal shunt. We performed a cranioplasty with autologous graft. After surgery presents malignant brain swelling and contralateral subdural hematoma. After 2 weeks of medical treatment and a new surgery for decompressive contralateral side was discharged.

Conclusion: Cerebral swelling after a cranioplasty is a complication that is probably reported less frequently than what happens. The cascade of events that precede this complication is not yet well defined; however, it is worth taking into consideration what has already been described in this regard, to try to avoid this situation.

Keywords: Cranioplasty; Brain Swelling; Cerebral Edema; Subdural Hematoma; Syndrome of Sinking Skin Flap

Background

Cranioplasty is used for patients who have undergone a craniectomy for refractory intracranial hypertension, caused by a variety of pathologies that condition cerebral edema, such as traumatic brain injury (TBI) or Stroke [11].

Cranioplasty is frequently underestimated by the neurosurgeon, however there are reports describing a complication rate of up to 34% [2]. Of all the complications of cranioplasty, malignant brain swelling after the surgery is the most serious complication and has an un-favorable prognosis, including death. [10] There are few cases reported and the pathophysiology has not been certainly established [3].

Clinical Case

It is an 18-year-old male patient with no significant chronic background. Begins his condition with Subarachnoid hemorrhage (SAH), Fisher III, Hunt and Hess 4, WFNS 4, an bifurcation of

left carotid artery aneurysm (Figure 1). With rebleeding and left middle cerebral artery infarction, a left fronto-temporo-parietal decompressive craniectomy was performed (Figure 2). After that develops hydrocephalus, we did a ventriculoperitoneal shunt, it is decided not to treat the aneurysm at this hospitalization. At external follow-up, it reached a 4 points of GOSE (Glasgow Outcome Scale Extended) and with trephine syndrome, paradoxical herniation, aneurysm clipping was performed and left cranioplasty with autologous graft, which remained in cryopreservation, 3 months after craniectomy. We corroborated the position and check adequate venous drainage. Closed subgaleal drainage with medium negative pressure. At 6 hours of the procedure a control skull tomography was performed (Figure 3), evidencing severe right hemispheric cerebral edema, right fronto-parietal acute subdural hematoma, midline deviation more than 10 mm. The proximal ventriculoperitoneal shunt partially slides with the tip at intraventricular space, immediately reoperated to perform a decompressive in situ hinge craniectomy and drainage of subdural hematoma.

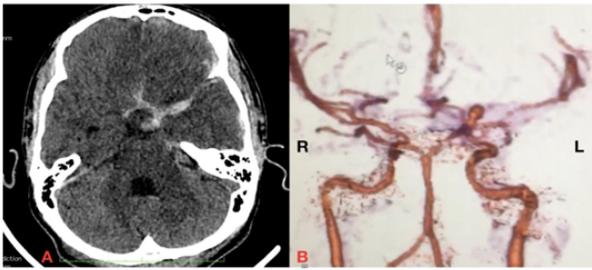


Figure 1: A, CT scan with subarachnoid hemorrhage. B, Cerebral aneurysm at bifurcation of left carotid artery.

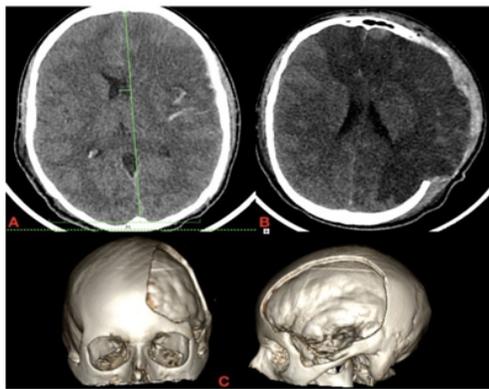


Figure 2: A, CT scan before decompressive craniectomy. B, CT scan after decompressive craniectomy. C, Post-surgical 3D CT scan.

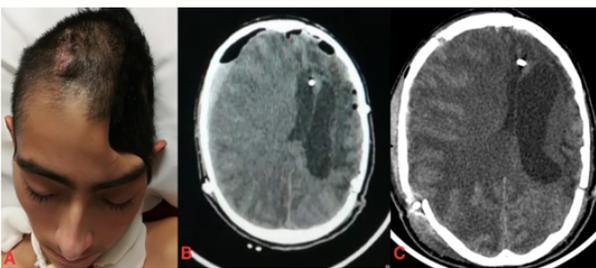


Figure 3: A, Preoperative photo with severe sinking skin flap. B, Post-cranioplasty CT scan. C, After in situ hinge craniectomy

Survived, and after was send home after 15 days of stay. In the follow-up after 5 months the patient shows discreet improvement and the shunt was relocated. Currently with 26 months of follow-up (Figure 4), the patient stays GOSE of 4 points.

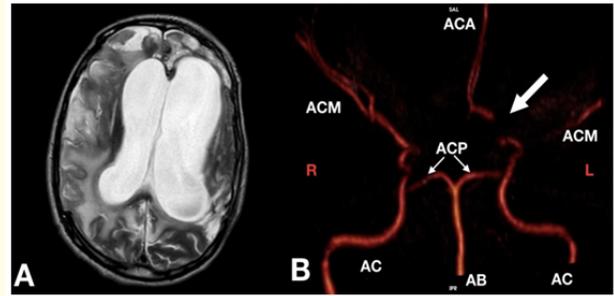


Figure 4: A, 6 month follow up MR, T2 sequence whit severe injury at white and gray substance. B, Angio-MR, vascular flow modified by vasospasm and infarctions, whit no evidence of aneurysm (white arrow). (ACA, Anterior cerebral artery; AC, Carotid artery; ACM, Middle cerebral artery; ACP, Posterior cerebral artery; AB, Basilar artery; R, Right; L, Left).

Discussion

The cranioplasty is performed not only for aesthetic purposes, but also to improve the neurological condition of patients with trephined syndrome who had a decompressive craniectomy. The cranioplasty is generally considered safe and the mortality rate is low. Consequently, cerebral swelling as a complication of cranioplasty is rare 1% [10]. The precised mechanism underlying this complication has not yet been fully understood. Lee., et al. re-ported a case of unexpected and severe postoperative cerebral edema after an autologous cranioplasty [5]. The authors in this previous report speculated that the placement of an epi-dural drainage system with active negative pressure may have caused an acute decrease in intracranial pressure and the subsequent rapid expansion of the brain, which led to a miss self-regulation and injury by reperfusion [4].

A negative pressure drainage will cause a sudden decrease in intracranial pressure and in-crease the intracranial pressure gradient. The trephined syndrome or “syndrome of the sinking skin flap” (SSSF) can occur if the brain gravity and atmospheric pressure exceed the intracranial pressure and subsequently produce a cavity when a cranioplasty is performed, the large cavity between the severely collapsed brain and the graft is filled with brain, which immediately facilitates the pressure differentials. Hoenybul., et al. supported this theory of negative pressure that produces malignant brain inflammation [3]. The rapid increase in cerebral blood flow will lead to venous congestion and stasis in case of miss self-regulation. Chitale., et al. stated that a differential of pressure induces venous stasis at venous sinuses and can lead to a venous

thrombosis and cerebral venous infarction [1]. Furthermore, we think that a prolonged collapsed brain increases the permeability of the tissues and causes a massive cerebral edema if the brain tissue expands in a short time [7]. Van Roost., *et al.* estimated that a negative pressure difference may occur in 1 of 7000 neurosurgical operations for supratentorial lesions [9]. The incidence of this pressure difference after cranioplasty may be higher, especially when the skull dome defect is large and the brain is atrophied and depressed. On the basis of the literature review, fatal massive brain swelling immediately after an uncomplicated cranioplasty may be a distinct clinical syndrome [8]. Robles., *et al.*, made a systematic review and even propose a flowchart to reverse SSSF to prevent complications like brain swelling after cranioplasty [6]. In conclusion, for patients with malignant edema after a cranioplasty, the SSSF and vacuum suction drainage are the main risks; In addition, the prognosis is extremely bad, with death reported for almost all patients [6,7].

Conclusions

Cerebral swelling after a cranioplasty is a complication that is probably reported less frequently than what happens. The cascade of events that precede this complication is not yet well defined; however it is worth taking into consideration what has already been described in this regard, to try to avoid this situation. In our case, we believe that the presence of a previous ventriculoperitoneal shunt, a paradoxical hernia and the immediate re expansion of abnormal brain tissue due to previous cerebral infarction conditioned this complication. Let's hope that the brief review helps us to understand a little more about the mechanisms that condition post-cranioplasty cerebral edema.

Conflict of Interest

The author has no financial or personal relationships with people or organizations that could influence this report. There is not a conflict of interest.

Founding

None

Ethical Consent

The informed consent was obtained from the family and patient for publication.

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