

Infratentorial and Supratentorial Strokes After a Cranioplasty

Rohan Chitale, MD, Stavropoula Tjoumakaris, MD, Fernando Gonzalez, MD, Aaron S. Dumont, MD, Robert H. Rosenwasser, MD, and Pascal Jabbour, MD

Introduction: Complications of cranioplasty are known to include infection, wound breakdown, intracerebral hemorrhage, bone resorption, and status epilepticus. Intracerebral hemorrhagic infarction after a cranioplasty is a very rare complication with only 2 reported cases to date. We present the first case in the literature of both supratentorial and infratentorial hemorrhagic infarctions after a cranioplasty.

Case Report: A 64-year-old male at an outside hospital suffered a right MCA hemorrhagic infarction requiring decompressive hemicraniectomy. One year later, the patient presents to our hospital for elective right-sided cranioplasty. The procedure was uneventful. However, postoperatively, the patient suffered a generalized tonic-clonic seizure and remained comatose. Electroencephalography showed no signs of status epilepticus, but imaging did reveal diffuse cerebral edema and both infratentorial and supratentorial hemorrhagic infarcts requiring placement of a ventriculostomy, removal of the cranioplasty plate, and suboccipital craniectomy. Postoperative tests revealed only the known right M1 occlusion, with no evidence of venous thrombosis, embolic source for new strokes, or new arterial dissection or occlusion. The patient remained with only brainstem reflexes and eventually expired.

Conclusions: This is the first in the literature to report the complication of both supratentorial and infratentorial strokes after a cranioplasty procedure. Reperfusion, vessel injury, and venous stasis after cranioplasty as evaluated by multiple neurological imaging modalities are examined as possible mechanisms for this unique complication. These factors must be considered when evaluating the safety of the procedure for a patient.

Key Words: decompressive hemicraniectomy, sunken skin flap syndrome, syndrome of the trephined, cranioplasty complications, stroke (*The Neurologist* 2013;19:17–21)

Cranioplasty complications are well described and include infection, wound breakdown, intracerebral hemorrhage, bone resorption, and status epilepticus.¹ Intracerebral hemorrhagic infarction after cranioplasty is a very rare complication with only 2 reported cases to date.^{2,3} We present the first case of both supratentorial and infratentorial strokes after a cranioplasty in a 64-year-old male who originally had a decompressive hemicraniectomy for a right MCA territory hemorrhagic infarct.

From the Department of Neurological Surgery, Division of Neurovascular Surgery and Endovascular Neurosurgery, Thomas Jefferson University Hospital, Philadelphia, PA.

The authors declare no conflict of interest.

Reprints: Pascal M. Jabbour, MD, Department of Neurological Surgery, Division of Neurovascular Surgery and Endovascular Neurosurgery, Thomas Jefferson University Hospital, 909 Walnut Street 2nd Floor, Philadelphia, PA 19107. E-mail: pascal.jabbour@jefferson.edu.

Copyright © 2013 by Lippincott Williams & Wilkins

ISSN: 1074-7931/13/1901-0017

DOI: 10.1097/NRL.0b013e31827c6bb6

Putative mechanisms to explain this rare complication will be discussed.

CASE REPORT

A 64-year-old man with no remarkable past medical history suffered a right MCA stroke with hemorrhagic conversion, requiring a decompressive hemicraniectomy at an outside hospital. At that time, he underwent an MRI which revealed a right M1 occlusion. His neurological examination revealed a left-sided hemiparesis. One year later, he presented electively to our hospital for a right-sided cranioplasty (Fig. 1). Implantation of a synthetic PEEK allograft was performed uneventfully. Immediately after the surgery, while waking up the patient experienced a generalized tonic-clonic seizure. A computed tomography (CT) scan showed expected postoperative changes with reexpansion of the right cerebral hemisphere (Fig. 2). Electroencephalography showed no evidence of status epilepticus. A follow-up CT scan was performed 5 hours later, which demonstrated diffuse brain edema, causing complete obliteration of the basal cisterns and early signs of hydrocephalus. In addition, there was a suggestion of developing posterior fossa infarcts (Figs. 3A, B). A left frontal ventriculostomy was placed with an opening pressure of 20 mm H₂O, and the patient was then taken to the operating room immediately for removal of the right cranioplasty flap. An MRI of the brain performed postoperatively revealed petechial hemorrhages in the midbrain, pons,

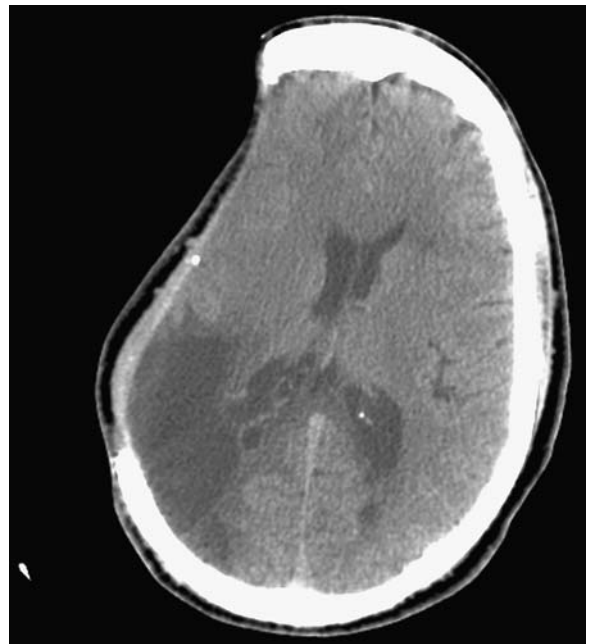


FIGURE 1. Preoperative head CT scan showing a sunken skin flap and old infarction of right MCA territory.

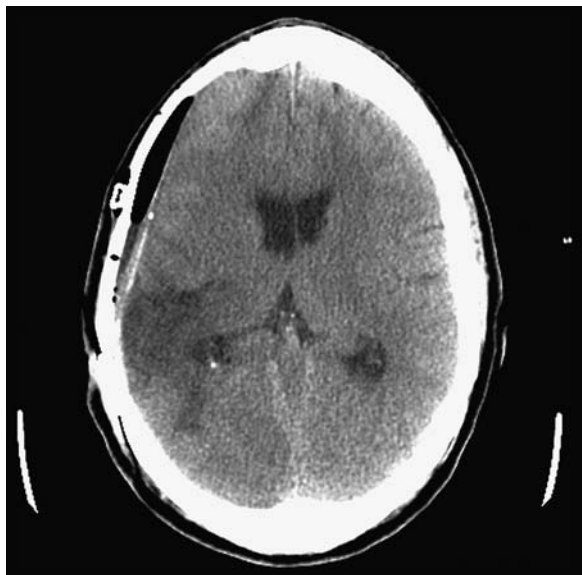


FIGURE 2. Immediate postoperative head CT scan showing reexpansion of brain and presence of allograft cranioplasty.

right frontal and occipital cortex along with restricted cortical diffusion in those areas, suggesting infarct. There was evidence of downward and upward herniation of the cerebellum (Figs. 4A–F). MRA of the head and neck showed no evidence of any new large vessel occlusion. Given the posterior fossa findings, the patient was taken directly back to the operating room for suboccipital decompressive craniectomy. Postoperatively, the patient never regained consciousness. Repeat CT scan of the head showed intraventricular blood with hemorrhagic conversion of both supratentorial and infratentorial strokes (Figs. 5A–E). No definitive evidence of any new arterial occlusion or venous thrombosis of intracranial vessels was evident on imaging. MRA revealed only the known right M1 occlusion. MRV also demonstrated decreased flow-related signal in the right transverse sinus possibly related to slow flow, but no sinus thrombosis (Figs. 6 A, B). Trans-thoracic and transesophageal echocardiogram showed no embolic source for stroke. The patient's neurological examination consisted

only of brainstem reflexes, and several weeks after these events the patient finally expired.

DISCUSSION

Decompressive hemicraniectomy is a life-saving procedure that can be performed to alleviate intractable intracranial pressures related to malignant cerebral infarctions. Removal of the bone flap allows edematous brain tissue to herniate through the created defect, and thus improve intracranial pressure and mass effect upon midline structures. After the intractable raised intracranial pressures have resolved, cranioplasty is often performed in a delayed manner to improve cosmetic appearance, restore protection to the exposed brain, and improve function. In some craniectomy patients, the atmospheric pressure and forces of gravity can be greater than the intracranial pressure, resulting in a paradoxical herniation called sinking skin flap syndrome or syndrome of the trephined.⁴ This syndrome can resolve with cranioplasty secondary to reequilibration of intracranial and extracranial forces. Several studies have been performed studying the perfusion effects of cranioplasty. One study used transcranial Doppler studies and 18-FDG positron emission tomography to show that cerebrovascular reserve capacity and glucose metabolism improved after cranioplasty.⁵ Xenon CT and P31 magnetic resonance spectroscopy have shown improved brain perfusion and cerebral metabolism after cranioplasty as well.⁶ CT perfusion after cranioplasty has demonstrated increased cerebral blood flow not only in the injured hemisphere but also in the contralateral hemisphere.⁷ Recently, MR perfusion has also shown increased cerebral blood flow and blood volume along with decreased mean transit time after a cranioplasty.⁸

Hemorrhagic infarctions have been reported as a complication of cranioplasty procedures.^{2,3} Cecchi et al² propose that the mechanism for ipsilateral hemorrhagic infarction is related to the increased cerebral blood flow on the side of the cranioplasty, as described above, where the brain was already prone to hemorrhage from a previous stroke. Possible mechanisms for diffuse hemorrhagic infarctions, both supratentorially and infratentorially as in our case, are less clear. Deep venous occlusion can lead to diffuse cerebral edema, infarcts, and hemorrhagic conversions with a clinical picture

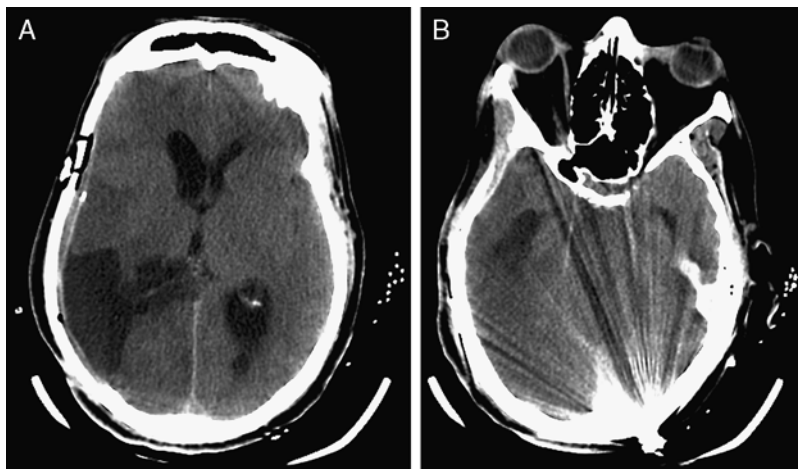


FIGURE 3. A and B, Postcranioplasty swelling and effacement of cisterns with suspicion of posterior fossa infarcts.

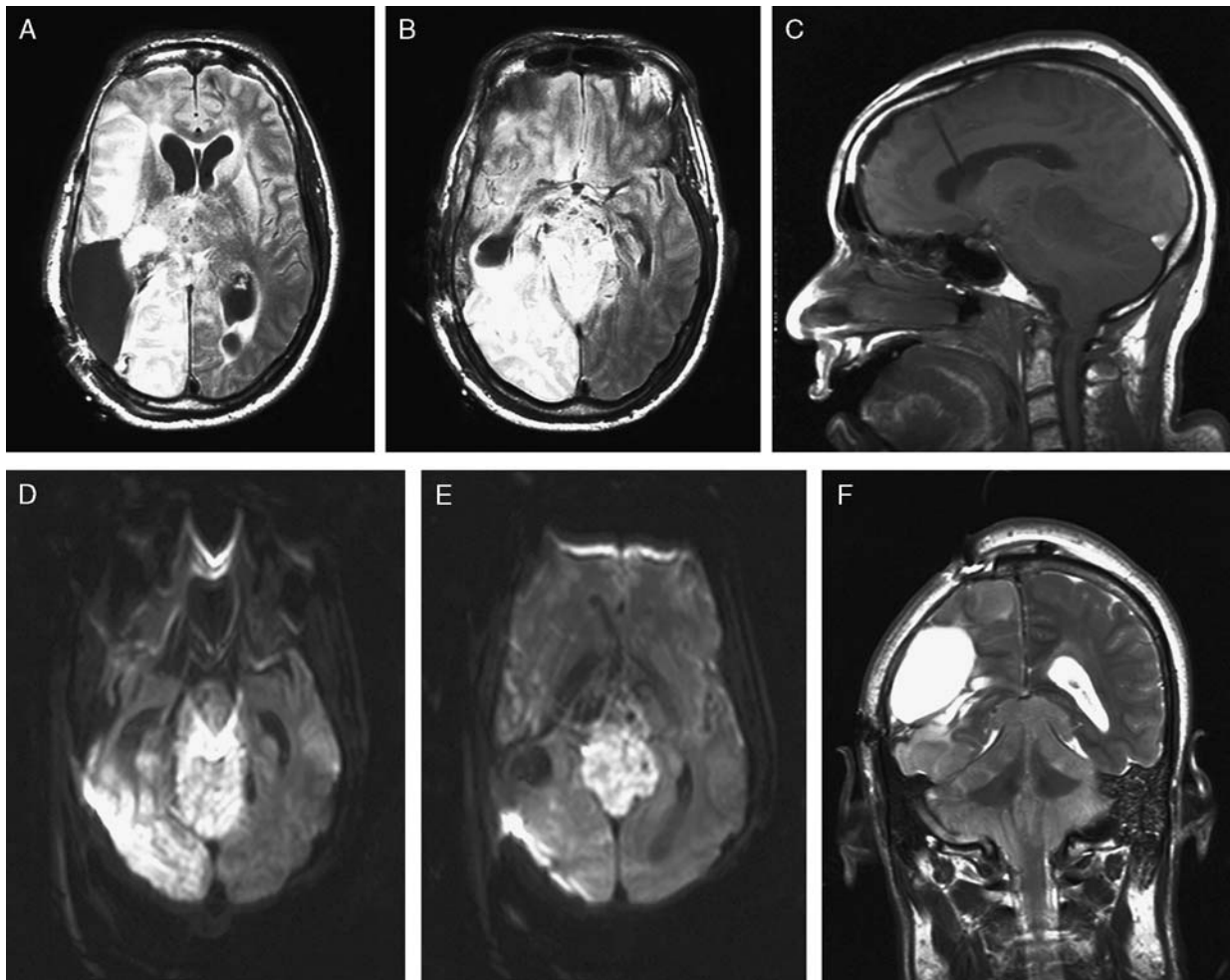


FIGURE 4. A–F, Postcranioplasty MRI showing multiple hemorrhagic infarctions and petechial hemorrhages in brainstem, with upward and downward herniation of cerebellum.

similar to that seen in our patient. Eom et al³ propose that the rapid increase in bilateral cerebral blood flow and volume in the chronic dysfunctional brain results in venous stasis and congestion that can lead to thrombosis and diffuse hemorrhagic infarcts as seen in our patient. Although MRV showed no definitive occlusion or thrombosis, it did demonstrate slower flow in the venous sinuses.

Reequilibration of the brain pressure vectors after the cranioplasty coupled with perfusion increases may suggest another causal mechanism. Removal of the atmospheric pressure vector on the brain results in a reexpansion of the brain inside the cranial vault. Such movement may result in the stretch of intracranial vessels. Stretch of supratentorial, infratentorial, and perforator vessels may result in a transient decrease in perfusion to target tissue as well as microvascular injury, which increases propensity for hemorrhage. The reexpansion may also lead to compression of PCA vessels on the incisura, leading to large territory infarctions as seen in our patient. In addition, the increase in cerebral perfusion after the cranioplasty may have increased the patient's risk of

hemorrhage into already weak brain tissue that lost the ability of autoregulation. This combination of mechanisms may have resulted in the diffuse hemorrhagic infarctions seen in our patient.

Intraoperative vascular insufficiency may have also been a factor caused by positional stenosis or dissection of vessels related to positioning in the operating room. However, this is unlikely given MRA and CTA evidence that the patient had minimal atherosclerotic disease and no evidence of arterial dissection in the carotid or vertebral arteries and the fact that his head was turned only 30 degrees during surgery. It should be noted, as well, that anesthesia records show no evidence of perioperative systemic hypotension as an attributable factor.

Analysis of best current evidence shows that timing of cranioplasty after decompression does not have an effect on the rate of cranioplasty complication.⁹ Although a full year had elapsed before this patient presented to our hospital for treatment, no technical difficulty was encountered regarding scalp dissection or scar formation to explain the witnessed complications. In addition, systemic variables including the

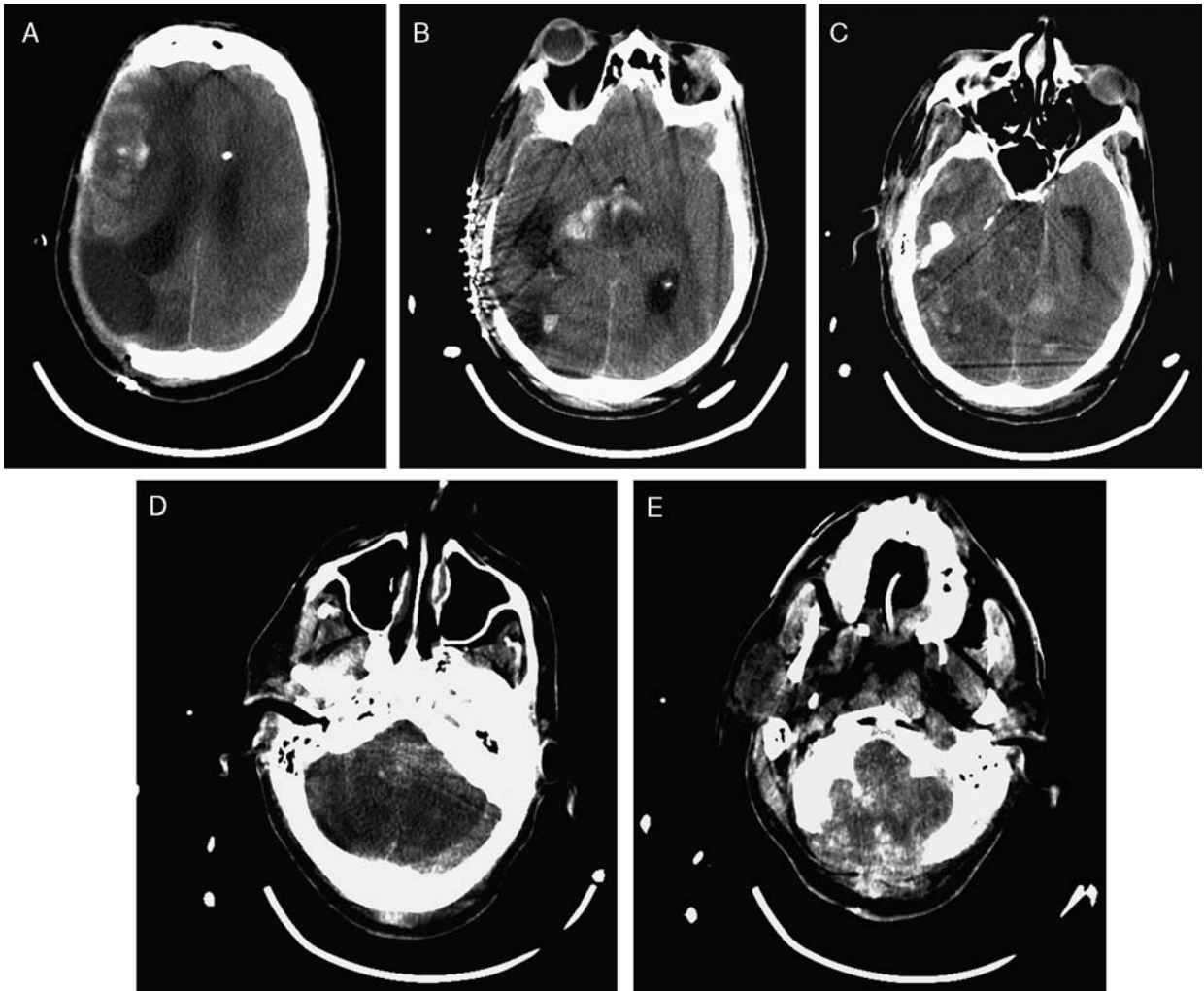


FIGURE 5. A–E, Postoperative head CT scan showing diffuse edema, new infarcts, and hemorrhagic conversion.

coagulation profile, platelet count, and nutritional status, which may predispose to complications, were all within normal limits.

This is the first reported case of supratentorial and infratentorial hemorrhagic infarctions after cranioplasty.

Although this complication of cranioplasty is extremely rare, the concepts of reperfusion, vessel injury, and venous stasis after cranioplasty should be considered when evaluating the risk of this procedure for the patient.

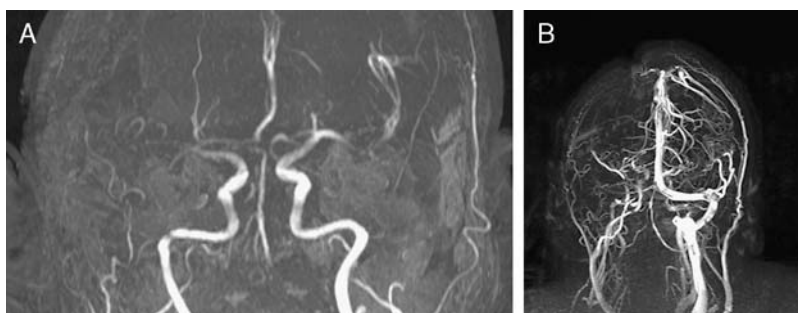


FIGURE 6. A and B, MRA and MRV demonstrating no new definitive arterial occlusion or venous sinus thrombosis.

REFERENCES

1. Gooch MR, Gin GE, Kenning TJ, et al. Complications of cranioplasty following decompressive craniectomy: analysis of 62 cases. *Neurosurg Focus*. 2009;26:E9.
2. Cecchi PC, Rizzo P, Campello M, et al. Haemorrhagic infarction after autologous cranioplasty in a patient with sinking flap syndrome. *Acta Neurochir (Wien)*. 2008;150:409–410; discussion 411.
3. Eom KS, Kim DW, Kang SD. Bilateral diffuse intracerebral hemorrhagic infarction after cranioplasty with autologous bone graft. *Clin Neurol Neurosurg*. 2010;112:336–340.
4. Akins PT, Guppy KH. Sinking skin flaps, paradoxical herniation, and external brain tamponade: a review of decompressive craniectomy management. *Neurocrit Care*. 2008;9:269–276.
5. Winkler PA, Stummer W, Linke R, et al. The influence of cranioplasty on postural blood flow regulation, cerebrovascular reserve capacity, and cerebral glucose metabolism. *Neurosurg Focus*. 2000;8:e9.
6. Yoshida K, Furuse M, Izawa A, et al. Dynamics of cerebral blood flow and metabolism in patients with cranioplasty as evaluated by ¹³³Xe CT and ³¹P magnetic resonance spectroscopy. *J Neurol Neurosurg Psychiatry*. 1996;61:166–171.
7. Sakamoto S, Eguchi K, Kiura Y, et al. CT perfusion imaging in the syndrome of the sinking skin flap before and after cranioplasty. *Clin Neurol Neurosurg*. 2006;108:583–585.
8. Kemmling A, Duning T, Lemcke L, et al. Case report of MR perfusion imaging in sinking skin flap syndrome: growing evidence for hemodynamic impairment. *BMC Neurol*. 2010;10:80.
9. Yadla S, Campbell PG, Chitale R, et al. Effect of early surgery, material, and method of flap preservation on cranioplasty infections: a systematic review. *Neurosurgery*. 2011;68:1124–1129, discussion 1130.