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Case Report

Malignant bihemispheric cerebral edema after cranioplasty – An extension of the Monro-Kellie doctrine and predictive factors

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ABSTRACT

Background: Several changes in normal pressure dynamics on the brain occur with a decompressive craniectomy and subsequent cranioplasty. Dead space volume is an important factor contributing to intracranial volume postcranioplasty. A decrease in this volume due to negative suction drain along with relative negative pressure on the brain with the loss of external atmospheric pressure may lead to fatal cerebral edema.

Case Description: A 52-year-old gentleman with a 210 mL volume and middle cerebral artery territory infarction underwent an emergency craniectomy and 6 months later a titanium mold cranioplasty. Precranioplasty computed tomography (CT) scan evaluation revealed a sunken skin flap with a 9 mm contralateral midline shift. Immediately following an uneventful surgery, the patient had sudden fall in blood pressure to 60/40 mmHg and over a few min had dilated fixed pupils. CT revealed severe diffuse cerebral edema in bilateral hemispheres with microhemorrhages and expansion of the sunken right gliotic brain along with ipsilateral ventricular dilatation. Despite undergoing a contralateral decompressive craniectomy due to the midline shift toward the right, the outcome was fatal.

Conclusion: Careful preoperative risk assessment in cranioplasty and close monitoring postprocedure is crucial, especially in malnourished, poststroke cases, with a sinking skin flap syndrome, and a long interval between decompressive craniectomy and cranioplasty. Elective preventive measures and a low threshold for CT scanning and removal of the bone flap or titanium mold are recommended.

Keywords: Craniectomy, Complication, Cranioplasty, Edema, Malignant, Monro-Kellie, Trephination

INTRODUCTION

Cranioplasty in literature dates back to the 16th century where Fallopius makes mention of a gold plate used to reconstruct a skull defect, though there is a scattered evidence of similar procedures

being performed since the Incans.[18] With the advent of asepsis, antibiotics, and various autologous, allogeneic, and artificial graft materials, cranioplasty has evolved into a relatively low risk, straightforward procedure. It is, however, also subject to its share of complications with reported rates of up to 41%.[2] Table 1 enumerates the common complications that can occur postcranioplasty[2,4,11,17,29] and how they may be avoided.

Postcranioplasty rebound cerebral edema, initially described as "pseudohypoxic brain swelling" by Van Roost D *et al*.,[25] is a known, albeit extremely rare complication. Since being first described, only a handful of case reports and series exist in the literature.

We report a case of malignant cerebral edema with multiple ipsilateral hemorrhagic foci occurring within an hour of a titanium mold cranioplasty procedure. The aim of this manuscript is to bring to attention a rare, often under-rated complication of a seemingly straightforward procedure, elucidate possible pathophysiological mechanisms, and identify patients at a high risk for the same.

CASE REPORT

Initial evaluation and intervention

A 52-year-old gentleman had presented to the emergency room with sudden onset left hemiparesis, with a Glasgow Coma Scale (GCS) of E2M5Vt. He was diagnosed with a 210 mL volume, middle cerebral artery (MCA) territory infarction on magnetic resonance (MR) imaging with mass effect and a 12 mm midline shift. He was also diagnosed with a right brachial artery embolus on routine large vessel and neck MR angiograms. Carotid vasculature was noted to be normal with no narrowing or filling defect. An emergency right fronto-temporo-parietal decompressive with lax duraplasty was performed followed by a right brachial embolectomy by the vascular team. Further evaluation with a cardiac 2D echogram revealed no regional wall motion abnormality with mild concentric left ventricular hypertrophy (LVH), normally functioning valves and no clots or vegetations. Postoperatively, he had an uneventful recovery and over a week GCS improved to E4M6V5. He was discharged on therapeutic doses of low molecular weight heparin for the brachial artery embolus.

Cranioplasty

He presented 6 months later for a cranioplasty. He was conscious alert and oriented with modified Ashworth Grade II hypertonia and Medical Research Council Grade 3 power in the left upper and lower limbs. A computed tomography (CT) scan revealed a sunken skin flap with a 9 mm midline shift to the left along with gliotic changes in the right MCA territory [Figure 1]. The autologous bone preserved in a freezer was found to be unhealthy and he underwent a customized titanium mold cranioplasty. Intraoperatively, the brain was sunken. Urine output was maintained at 80–150 mL/h and vitals were stable with minimal fluctuations. The surgery was completed uneventfully.

Table 1: Common complications and their avoidance in cranioplasty.

Complication	Avoidance
Intraoperative/Immediate	
Improper fitting of graft	Proper storage/early cranioplasty to minimize resorption Adequate contouring of titanium meshes/molds
Inadequate/high tension skin closure	Adequate subgaleal scalp release Avoid over-contouring molds
Hematoma (over or under prosthesis)	Passive (or under *mild* suction, as per institutional protocols) drain placement Absolute hemostasis Blood pressure control postoperatively
Early	
Facial/eyelid edema	Avoid excessively tight application of the crepe bandage
Infections	Performing cranioplasty within 3–6 months after craniectomy Strict asepsis and storage of autologous bone in the abdomen or cryopreserved at temperatures between−18°C and−83°C[4]
Wound dehiscence	Avoid tension at incision site Avoid excessive contouring of implant and stretching of skin over prominences Avoid projecting of any end of the implant/bone into the galea Asepsis
Late	
Bone resorption	Rigid fixation of bone flap to promote fusion and prevent pseudoarthrosis Custom implant in very large defects (>70 cm^2)

Postoperative period and outcome

The patient did not wake from anesthesia and had sudden fall in blood pressure (BP) to 60/40 mmHg a few minutes after shifting, on ventilator, to the intensive care unit. The hypotension lasted for less than a minute and he was stabilized with inotropes and noradrenaline boluses and later a drip infusion, with close titration owing to a very labile BP highly sensitive to minor adjustments in the inotropes. Blood gases, electrolytes, and postoperative hematocrit were within normal limits. A screening echocardiogram at the time showed an ejection fraction of 55% with freely moving valves and no clots or vegetations, ruling out a cardiac cause for the hypotension. During and immediately following stabilization, his pupils began dilating bilaterally from 2 mm to 8 mm over 30 min.

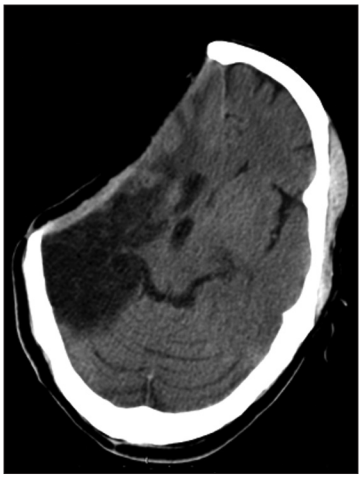

Figure 1: Precranioplasty computed tomography scan shows a sunken skin flap with a contralateral midline shift suggestive of a significant effect of the atmospheric pressure on the brain.

A CT scan showed diffuse severe cerebral edema in bilateral hemispheres with effaced basal cisterns, microhemorrhages, and expansion of the sunken right gliotic brain along with ipsilateral ventricular dilatation. There was poor grey-white matter differentiation in the contralateral (left) side with a midline shift of 5 mm toward the right (cranioplasty) side [Figure 2]. GCS remained E1M1Vt (No eye response or motor response, intubated on ventilator support) and pupils was now dilated fixed (8 mm bilaterally). Due to left-sided edema and risk of aggravation of midline shift by the right-sided titanium mold removal, a left-sided decompressive craniectomy and lax duraplasty were deemed suitable and performed.

A magnetic resonance imaging was performed the subsequent day that showed bilateral posterior cerebral artery territory and brainstem infarcts [Figure 3].

Despite all possible efforts and interventions, the patient did not improve, had absent brainstem signs with dilated fixed pupils and a GCS of E1M1Vt, and eventually a fatal outcome.

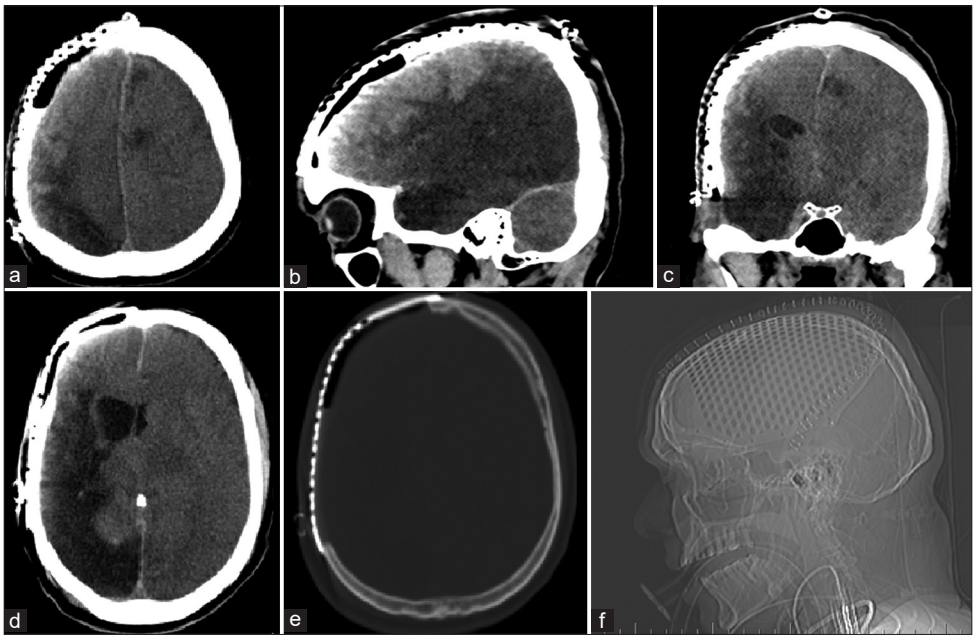

Figure 2: (a-c) Axial, sagittal, and coronal computed tomography scan images immediately after the procedure showing malignant bihemispheric cerebral edema with microhemorrhages. (d-f) Axial brain window, axial bone window, and a scout image showing the custom titanium mold *in situ*.

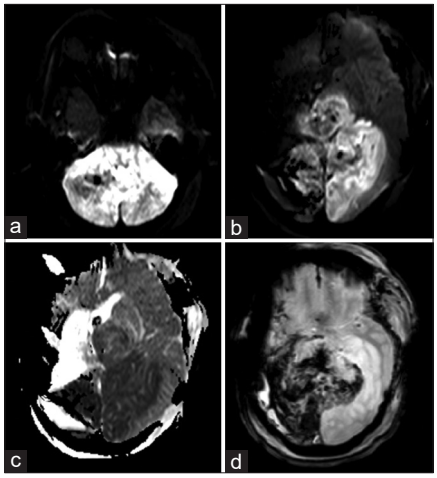

Figure 3: Post procedure magnetic resonance imaging revealed bilateral posterior circulation hemorrhagic infarcts and severe edema. Diffusion-weighted imaging showing diffusion restricting acute infarcts (a and b), decreased apparent diffusion coefficient (c), and blooming on gradient echo sequence (d).

Possible etiologies

In this case, other possibilities of malignant cerebral edema include fresh emboli from the heart causing fresh infarcts. Although fresh infarcts developing severe edema rapidly within an hour is rare, a screening cardiac 2D echogram was done at the time which showed normal findings apart from mild concentric LVH, and an ejection fraction of 55%, matching the evaluation done at the time of the initial infarct.

Hypoxia during surgery could theoretically cause bihemispheric edema; however, no such hypoxia or desaturation occurred intraoperatively or immediately postoperatively.

Hypotension due to an unrelated cause and subsequent hypoxic brain injury leading to malignant edema may be considered. However, no other systemic causes of hypotension could be identified. There was minimal blood loss during surgery and an 80–150 mL/h urine output throughout ruling out hypovolemia. Cardiac causes had been ruled out (as mentioned earlier) and there was no cause for septic shock immediate postoperatively. It was hence concluded that the hypotension was a consequence of severe brain edema and loss of autonomic control, further supported by the lability of the BP and extreme sensitivity to inotropic agents.

After excluding the above, the sequence of events suggests postcranioplasty malignant edema due to a change in pressure dynamics, as the most likely cause of the deterioration.

Table 2: Postulated mechanisms of rebound edema postcranioplasty.

Author	Year	Mechanisms postulated
Cecchi *et al*.[6]	2008	Reperfusion injury; Increased cerebral blood flow postcranioplasty
Eom *et al*.[8]	2010	Cerebral deep venous occlusion/ congestion leading to edema and venous hemorrhagic infarcts
Sviri[22]	2015	Intracranial hypotension caused by a negative suction drain, leading to brain shift, secondary ischemia, and edema
Honeybul[13]	2016	Failure of cerebral autoregulation leading to fluctuations in cerebral blood flow with changes in blood pressure and intracranial pressure dynamics

DISCUSSION

Postcranioplasty cerebral edema is, fortunately, an exceedingly rare complication, with twenty odd cases reported throughout world literature till date,[5,7-10,12,14,15,19,22,25] with a 90% of mortality rate.

Pathophysiology

Table 2 summarizes the various pathophysiological mechanisms proposed.[6,8,13,22]

When a decompressive craniectomy is performed, the Monro-Kellie doctrine[16] no longer applies and over time, a new homeostasis is achieved with the cerebral compliance and intracerebral pressure reaching an equilibrium with the external atmospheric pressure. Cranioplasty re-establishes a closed intracranial compartment. We posit that an extended Monro-Kellie doctrine comes into play where:

k (*constant*) = *Brain parenchymal volume + Cerebral blood volume + Cerebrospinal fluid volume + Dead space volume*

This dead space has a natural relative negative pressure[25] over the brain parenchyma due to the now absent positive atmospheric pressure, which is further accentuated by a negative suction subgaleal drain.[20] Therefore, the dead space has a natural tendency to reduce in volume and collapse on itself.

In a gliotic brain, the parenchymal volume is already considerably reduced, and with cerebrospinal fluid (CSF) volume being relatively constant in a hyperacute setting, cerebral blood volume increases with decreasing dead space volume. An already dysfunctional cerebral microcirculation (due to trauma/infarction etc.) gives way to increased cerebral blood flow, leading to malignant edema and hemorrhages.

This is supported by the largest reported case series by Sviri[22] in 2015, who reported four patients with cerebral edema

Table 3: Current case reports in literature of postcranioplasty malignant edema.

Author/Year	Age/Sex	Reason for craniectomy	Time interval before cranioplasty	Skin flap before cranioplasty	Type of cranioplasty	Drain	Intervention	Outcome
Cecchi *et al.* (2008)	77/M	Right frontoparietal hemorrhagic infarct	2 months	Significantly sunken flap	Autologous bone flap	-	-	Fatal
Eom *et al.* (2009)	63/M	Ischemic infarct	9 months	Significantly sunken flap	Autologous bone flap	-	-	Fatal
Santana-Cabrera *et al.* (2012)	17/M	Traumatic brain injury – left subdural hematoma and generalized edema	2 months	Significantly sunken flap	Autologous bone flap	-	Removal of bone graft and bifrontal decompressive lobectomy	Fatal
Chitale *et al.* (2013)	64/M	Right MCA infarct with hemorrhagic transformation	12 months	Significantly sunken flap	PEEK allograft	-	Removal of Graft	Fatal
Lee *et al.* (2015)	50/F	Spontaneous right temporal lobe intracranial hemorrhage with right subdural hematoma	2 months	Significantly sunken flap	Autologous bone flap	Closed suction drain	Medical management with anti-edema measures	Good
Sviri (2015)	22/M	Traumatic right fronto-temporo-parietal subdural hematoma and frontal brain contusions	9 months	Full craniectomy site flap	Right Autologous bone flap (frozen – 72°C)	Closed suction drain	Immediate contralateral decompression	Fatal
	14/M	Traumatic left fronto-temporo-parietal subdural hematoma	10 months	Significantly sunken flap	Autologous bone flap (frozen – 72°C)	Closed suction drain	Removal of mesh and ipsilateral decompression	Fatal
	28/M	Penetrating gunshot injury into right frontal bone and lobe with intracerebral hematoma	17 months	Significantly sunken flap	PMMA cement	Closed suction drain	Immediate bilateral decompressive craniectomy	Fatal
	24/M	Traumatic left fronto-temporo-parietal subdural hematoma	3 months	Full craniectomy site flap	Autologous bone flap (frozen – 72°C)	Closed suction drain	Immediate bilateral decompressive craniectomy	Fatal
Mangubat and Sani (2015)	14/F	Traumatic left subdural hematoma and cerebral edema	3 months	Significantly sunken flap	PEEK allograft	-	-	Fatal
Hassaneen *et al.* (2015)	50/F	Right MCA territory infarct	4 months	Significantly sunken flap	Autologous bone flap	-	-	Fatal
Honeybul (2016)	25/F	Acute cerebral infarction	11 months	Significantly sunken flap	Titanium mesh	-	Removal of titanium mesh	Fatal
	74/F	Right MCA infarct	2 months	Significantly sunken flap	Autologous cranioplasty	-	Removal of titanium mesh	Fatal

(Contd...)

Table 3: *(Continued).*

Author/Year	Age/Sex	Reason for craniectomy	Time interval before cranioplasty	Skin flap before cranioplasty	Type of cranioplasty	Drain	Intervention	Outcome
	41/F	Left basal ganglia intracerebral hemorrhage	12 months	Significantly sunken flap	Titanium mesh	External ventricular drain (10 mL drained)	-	Fatal
Shen *et al.* (2017)	51/M	Traumatic subdural and intracerebral hematoma	6 months	Significantly sunken flap	Titanium mesh	Closed suction drain (500 mL drained)	Removal of mesh and ipsilateral decompression	Fatal
Wang *et al.* (2017)	24/M	Traumatic brain infarction	4.5 months	Significantly sunken flap	Titanium mesh	-	Removal of titanium mesh and decompression	Good
Urano *et al.* (2017)	64/M	Massive left intracerebral hemorrhage with intraventricular hemorrhage	1 month	Significantly sunken flap	Titanium mesh	Closed suction drain	Removal of titanium mesh	Fatal
Zhang *et al.* (2019)	40/M	Right basal ganglia bleed and brain herniation	2 months	Bulging skin flap	Titanium mesh	Lumbar CSF Drainage (350 mL -preoperatively; 400 mL -postoperatively)	Removal of titanium mesh and decompression	Fatal
Wen *et al.* (2019)	42/M	Massive cerebral infarction in the left MCA territory	1 year	Significantly sunken flap	Titanium mesh	Closed suction drain (150 mL)	-	Fatal
Shimizu *et al.* (2020)	34/M	Traumatic right subdural hematoma	38 days	Partly sunken flap with dural dystrophic calcification	Autologous bone flap (frozen – 20°C)	-	-	Fatal
Wang *et al.* (2023)	45/M	Traumatic brain injury	3 months	Significantly sunken flap	-	-	-	-

PMMA: Polymethylmethacrylate, MCA: Middle cerebral artery, CSF: Cerebrospinal fluid, PEEK: Polyetheretherketone, M: Male, F: Female

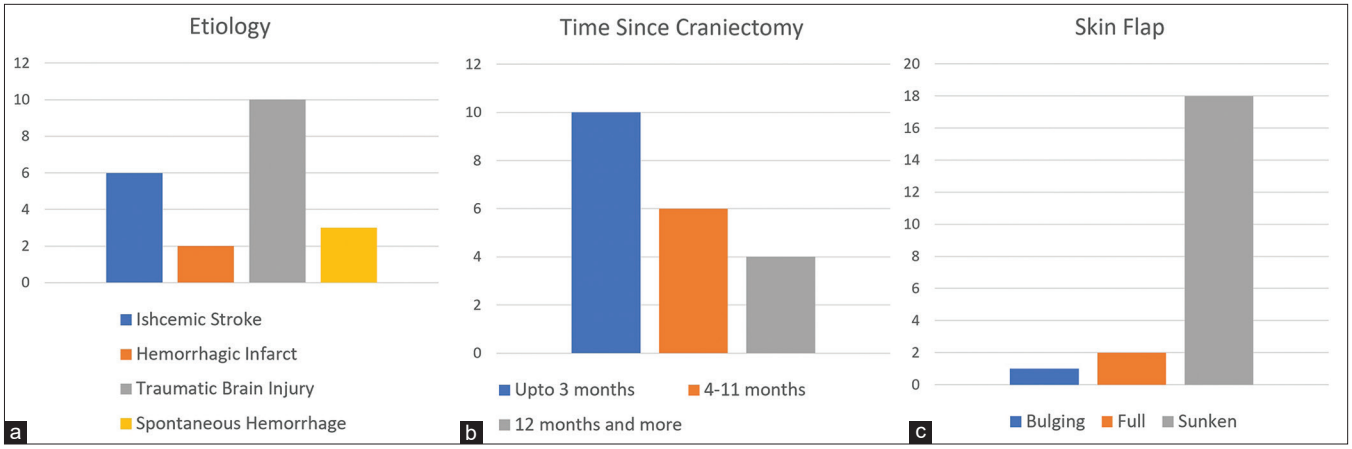

Figure 4: (a-c) Frequency of various risk factors in literature.

Table 4: Postcranioplasty cerebral edema risk assessment.

Risk factor	Score
Craniectomy site	
Full/Bulging	Low risk
Asymptomatic sunken skin flap	Mod risk
Sinking skin flap syndrome (symptomatic)	High risk
Time since craniectomy	
>12 months	Low risk
3–12 months	Mod risk
0–3 months	High risk
Pathology	
Spontaneous intracranial hemorrhage	Low risk
Ischemic/Hemorrhagic infarct	Mod risk
Traumatic brain injury	High risk

after cranioplasty, all of whom had a significantly sunken craniotomy site preoperatively and a large craniectomy defect, thereby potentially leading to a considerable relative negative pressure in the dead space and postcranioplasty. This creates a controversy in the management of sinking skin flap syndrome where cranioplasty has been accepted as the definitive management.[3,23] We agree with this line of management, with emphasis on early identification of patients in whom acute decompression of the atmospheric pressure over the brain may lead to malignant edema, elaborated further in the manuscript.

Sviri also noted all patients had a closed suction drainage.[22] A case reported by Shen *et al*. also involved a 300 mL collection in the drain,[20] further supporting the negative pressure theory. Table 3 collates the risk factors in various case reports in current literature [Table3].[21,24,26-28,30]

Risk factors and predictors

This complication occurred over all age groups and no particular age group could be identified as being at a high risk. Based on the reported cases in the literature till date, the following high-risk factors have been identified – Sinking skin flap syndrome, the time since craniectomy, and the primary pathology [Figure 4]. The risk factors are based on underlying pathophysiological mechanisms of (1) increase in relative negative pressure with increased effects of precranioplasty atmospheric pressure, (2) increased cerebral compliance with time and time-sensitive microcirculatory changes to adapt to the external atmospheric pressure, and (3) observed frequency of primary pathologies in reported cases of postcranioplasty cerebral edema [Table 4]. A patient with more than one high-risk factor, we postulate, warrants extra caution, and postoperative monitoring in an intensive care unit for the first 48-h postprocedure. Nonrecovery from anesthesia, inadequate spontaneous breathing, generalized seizures,[20] and a low/drop in GCS postoperatively are all red flags that must prompt an immediate CT scan.

The above factors need validation and testing in large numbers of cranioplasty patients and must be suitably extended/modified and condensed into a risk scoring system, before being conclusively used to identify patients at a high risk of developing cerebral edema postcranioplasty.

Possible prevention in high-risk patients postoperatively

As per the extended Monro-Kellie doctrine described above, we believe that minimization of either the dead space or the negative pressure in it[25] is crucial to prevent increased cerebral blood flow and parenchymal shear, and thereby avoid edema/hemorrhage formation in patients at high risk for the same. The authors would like to emphasize that the following are pathophysiology-based suggestions and need to be clinically evaluated with randomized controlled trials before being incorporated into institutional protocols.

1. Generous hydration preoperatively to promote CSF formation and decrease shear on the brain parenchyma

in accordance with the extended Monro-Kellie doctrine
2. A slow spinal infusion of normal saline through a lumbar puncture to expand the compressed cerebral hemisphere[1] and thereby reduce paradoxical herniation before the cranioplasty surgery
3. Moderate irrigation and partly filling saline in the dead space – As saline is slowly absorbed/drained, it may potentially help in preventing sudden brain shifts. Caution must be exercised in not filling excess saline that may exert pressure on the brain and accentuate the preexisting midline shift
4. Dead space may be reduced by dependent positioning of the patient with the ipsilateral side gravity dependent (like in the management for sinking skin flap syndrome) after the procedure
5. Passive subgaleal drain – without suction.

CONCLUSION

Decompressive craniectomy changes the intracranial milieu and adds external atmospheric pressure to the myriad of forces at play on the brain. The modified Monro-Kellie doctrine as described emphasizes the role of dead space volume after cranioplasty, which we believe plays a critical role in malignant cerebral edema after cranioplasty. It is crucial to assess the risk of this fatal complication preprocedure and take steps to prevent it in the high-risk subgroup. Trephination syndrome, an etiology of traumatic brain injury and cranioplasty within 3 months after craniectomy, is high-risk factors. In these patients, close monitoring and measures to prevent cerebral edema are recommended.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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