Article published online: 2023-01-18

Complications after Cranioplasty: A Pictorial Narrative with Techniques to Manage and Avoid the Same

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Indian J Neurotrauma 2023;20:124–132.

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Abstract

Background Cranioplasty following decompressive craniotomy is considered to be a "routine procedure" but several large series have documented a significant amount of both delayed and immediate complications and also a mortality rate of up to 3.6%.

Materials and Methods We went through some of the salient complications (excluding seizures) needing resurgery following interval cranioplasty over the past 18 years at our institution in over 300 cases and analyzed the literature that mention these complications and their treatment.

Results In addition to the commonly mentioned complications, we found some that had been rarely described or not mentioned hitherto in the literature which we have presented as a pictorial narrative. Based on our experience, we recommend some measures that may decrease the incidence or prevent the occurrence of the same.

Conclusions Attention to small but basic surgical techniques will go a long way in preventing unwanted postoperative events.

Keywords
- decompressive craniotomy
- cranioplasty
- complications
- infection
- bone flap resorption
- flap mobility
- epidural fluid collection
- extradural hemorrhage
- flap subsidence

Introduction

Cranioplasty (CP) following decompressive craniotomy (DC)[1] is performed after the intracranial pressure subsides with the aim of improving cosmesis and neurological function.[2,3] Conventionally done 3 to 6 months after DC, it is termed as "early" CP when done within 12 weeks of DC and "late" if done after this period.[3]

Though commonly considered to be a "simple operation"[1] and often done by residents in training it has numerous complications. In a series of 348 CPs, Zanaty et al[4] documented complications in 109 patients (31.32%) with a mortality rate of 3.16%, while in 62 patients who underwent CP, Gooch et al[5] noted postoperative complications in 34%, with 16 patients (25.8%) needing reoperation. In this article, we present a pictorial narrative of the complications (excluding seizures) we have encountered over the past 18 years in a series of over 300 CPs along with their management and suggested methods to avoid the same [►**Table 1**].

List of Complications, Management, and Preventive Measures

Skin Breakdown and Flap Exposure

Skin breakdown with exposure of the bone flap can occur at the incision line of the CP or even distant from it

article published online January 18, 2023

DOI https://doi.org/10.1055/s-0042-1760419.
ISSN 0973-0508.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

Table 1 List of complications (excluding seizures) following cranioplasty and preventive measures for the same

Complications	Preventive measures
Skin breakdown and flap exposure	Careful dissection Tension free closure Avoidance of excess use of cautery at the margins
Wound infection Subgaleal pus Intracranial empyema Pyogenic osteomyelitis	Avoiding excessive tissue dissection Eliminating dead space by placing a subgaleal drain, hitching the neo dura to the superior surface of the CP flap, tenting sutures of the scalp from galea to pericranium Strict maintenance of asepsis during surgery Frequent imaging in cases of unexplained or persistent subgaleal collection.
Hematoma below the replaced flap	Optimization of bleeding parameters before surgery Meticulous hemostasis Placement of dural tenting sutures through the bone to eliminate dead space and/or drain in the extradural space Keeping some space at the margins of the base of the replaced flap for escape of collection
Intraparenchymal hemorrhage	Careful and sharp dissection of the skin flap from the neodura after identifying the galeal-neodural plane avoiding dural breach Ensure the screws which fix the mini plates to the bone margins of the craniotomy defect do not violate the dura
Epidural fluid collection/ hygroma below the flap	Avoidance of opening dura during dissection of the skin flap Making small holes in the flap during CP to allow for egress of the collection into the subgaleal space with placement of a subgaleal suction drain
Flap resorption	Consider using artificial substitutes during initial CP in those who are at a higher risk of flap resorption – younger patients with larger, multifragmented flaps
Flap mobility - poor fixation - incompatible flap size due to absorption during storage	Fixing the flap firmly with a variety of commercially available devices rather than sutures Avoid replacing flaps that have decalcified and shrunk during storage in the body and use artificial substitutes like titanium mesh or PMMA bone cement
Flap subsidence	Rigid fixation of flap during CP to obviate any inward migration later
Implant extrusion	Avoid using fixation devices at sites where the overlying skin and soft tissue is thin Bicortical purchase of screws may prevent back out.

Abbreviations: CP, cranioplasty; PMMA, polymethyl methacrylate.

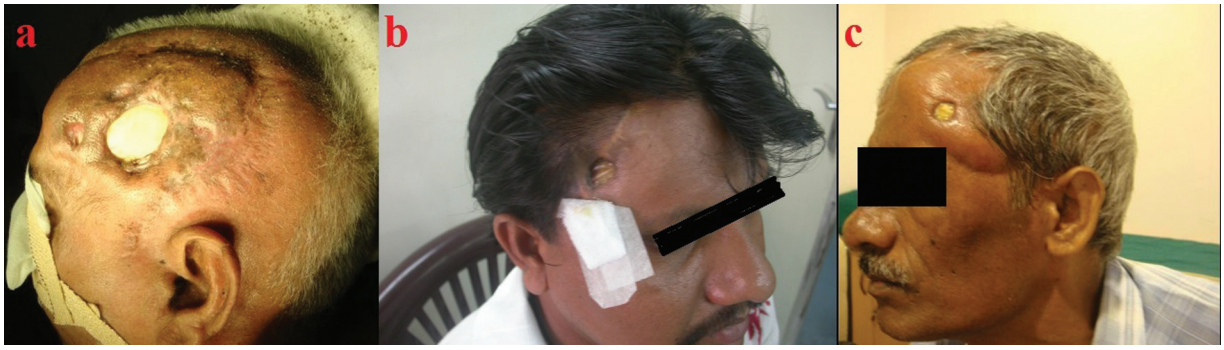

Fig. 1 Clinical images showing delayed wound complications with bony exposure both away from (**A, C**) and at the level of the incision (**B**).

[►**Fig. 1**] and at varying intervals following the CP. Thin skin with lack of subcutaneous support, poor galeal closure, devitalized skin margins due to excessive use of cautery, and tight sutures causing necrosis of the margins are possible causes of incision line breakdown. Repaired lacerations in the middle of the skin flap that was raised during DC lack subcutaneous support, adhere to the "neo dura" and may be devascularized during dissection and elevation of the skin at CP and breakdown later. Most commonly, skin breakdown follows the infection of the operative site.

Di Rienzo et al[6] classified wound complications as being one of three types – ***dehiscence, ulceration, or necrosis***. Dehiscence, the commonest, is the separation of opposing margins along the suture line while ulceration is defined as the loss of substance occurring inside the skin flap distant

from the line of suture and necrosis is a large, discolored area of nonviable skin without exposure of the subjacent bone.[6] These need to be treated promptly as osteomyelitis and spreading infection may ensue. In the absence of infection and if detected, early dehiscence can be managed by debriding the margins until fresh bleeding occurs followed by secondary suturing but in cases who present late there is retraction of the margins and tension free closure is difficult. The conventional notion that an exposed CP ***must*** be removed (particularly in cases who present early) needs re-examination given the availability of better antibiotics and improved surgical techniques such as flap coverage.[7] As all our cases presented late, the CP flap was removed and closure with either pulley sutures or rotation flap was done.

Wound Infection

It is the most dreaded complication of CP and ranges from subgaleal pus, intracranial pus below the bone flap (empyema), frank osteomyelitis, or a combination of all of these [►**Figs. 2–4**]. Infections may be classified into early (occurring within 4 weeks of CP) or late (occurring after 4 weeks of CP).[8] Early infections usually present with fever, wound discharge, local swelling, redness and tenderness, and elevated total counts and C reactive protein (CRP) on blood investigations.[8] These may be absent[9] in delayed infections that can even occur several months after the CP.

On plain computed tomography (CT) scan imaging, subgaleal pus may be confused with a sterile subgaleal collection but the presence of air must be carefully looked for and if present this hypodensity is often an indicator of infection.[10] Contrast-enhanced CT or magnetic resonance imaging (MRI) scans showing enhancement of the walls of a single or multiseptate collection or restriction on diffusion-weighted imaging are confirmatory for the presence of infection.[8]

Though it is difficult to identify osteomyelitis unless the bone is grossly eroded because the avascular CP bone does not enhance on contrast we believe the treatment in all cases is re-exploration and removal of the bone flap, thorough debridement and closure to be followed by long-term antibiotics as per culture reports.

Hematoma Below the Replaced Flap

Collection of blood below the CP flap, which is thin and seen in only one to two cuts in a postoperative CT scan is of no clinical consequence. However, on occasion, a large collection may be seen below the replaced bone resulting

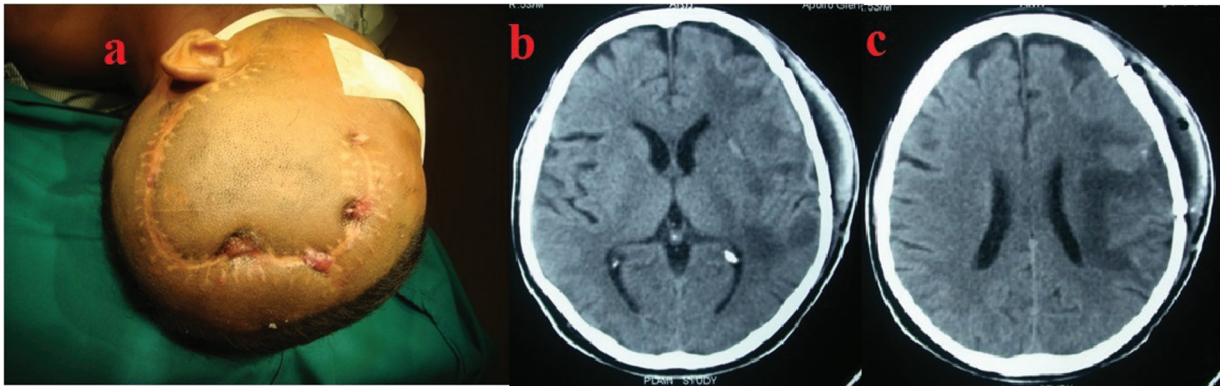

Fig. 2 Clinical image of a patient presenting with subgaleal collection (3 weeks after suture removal) due to infection with pouting granulation at the incision site (**A**); axial CT scans (**B, C**) showing collection in the subgaleal plane with hypodensity in the collection (**C**) that is a strong marker of infection.

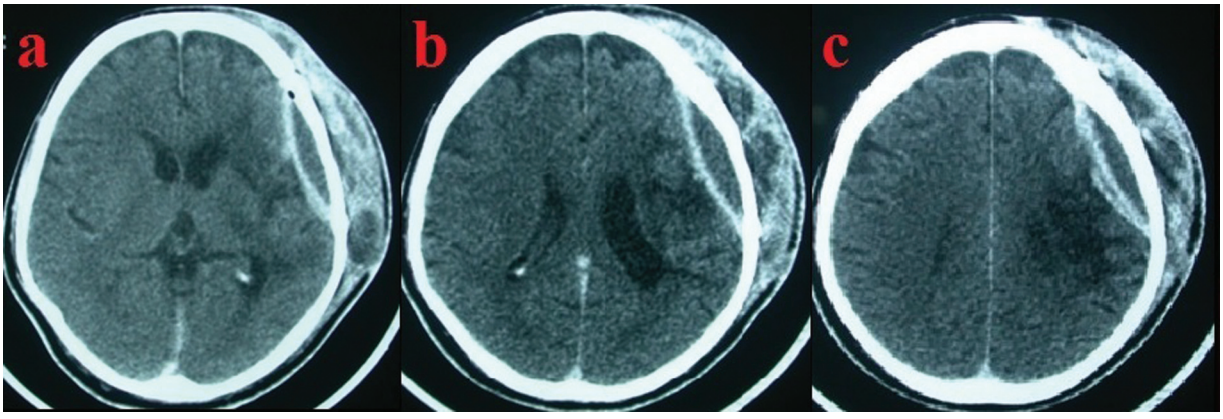

Fig. 3 Contrast-enhanced CT scans (**A–C**) showing abscess with enhancing walls both outside and inside the replaced bone flap.

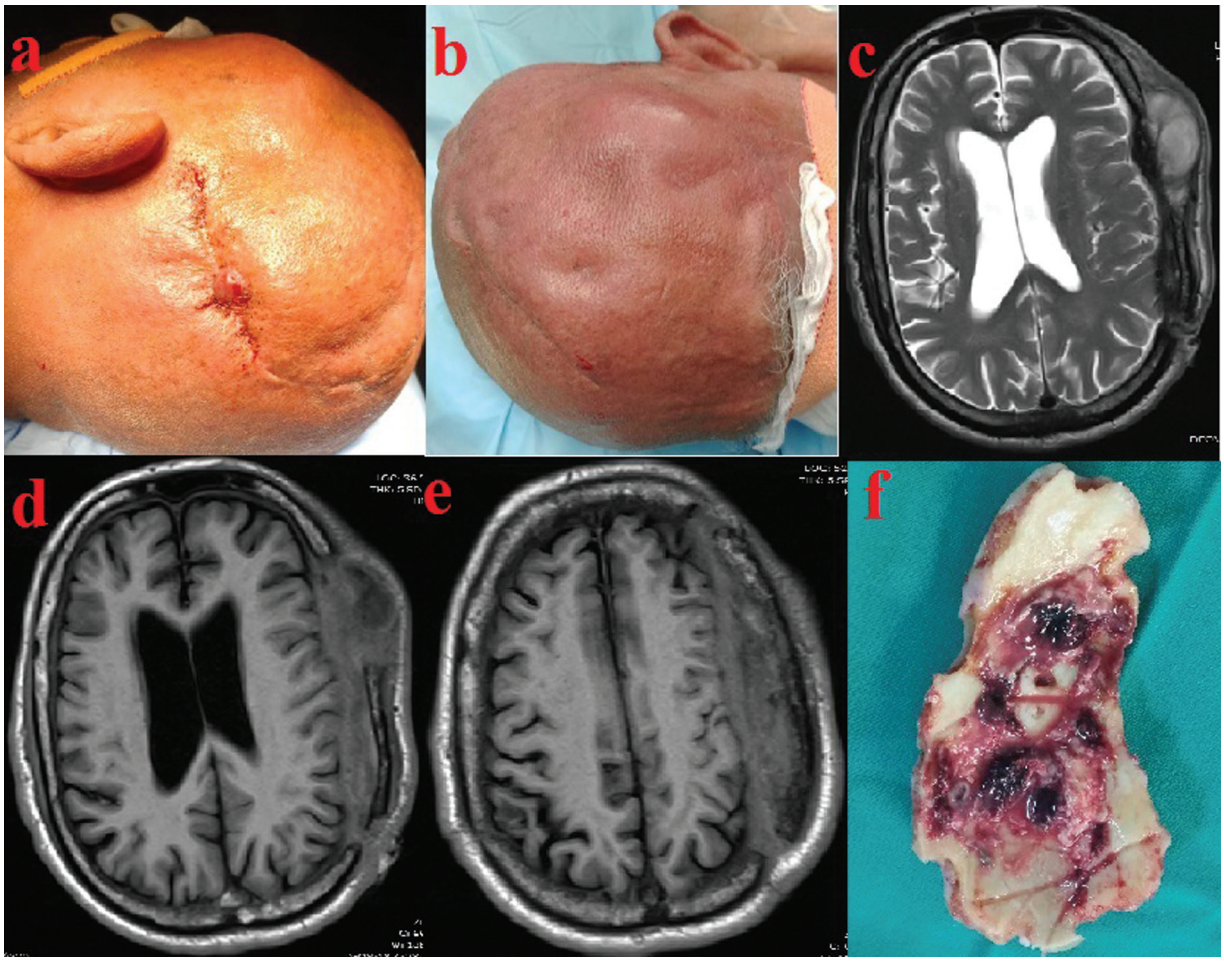

Fig. 4 Clinical photographs (**A, B**) of a patient with delayed wound infection 8 months after the cranioplasty showing granulation tissue with discharge at the incision site; noncontrast MR imaging (**C–E**) showing liquefied collection outside and granulation tissue inside the replaced bone flap and (**F**) clinical image of the osteomyelitic eroded bone flap.

in mass effect and midline shift of the underlying brain.[11] Causative factors include bleeding diathesis, improper operative site hemostasis, and a vascular "neo-dura" that is often found in early CPs.

These hematomas are usually extradural while subdural bleed post CP is rare due to the tenuous but well-formed arachnoidal connections traversing the subdural space between the brain and the overlying "neo-dura" that develop following DC essentially obliterating the subdural space.

We have found extradural hematomas to be more frequent in cases where there is an indwelling shunt

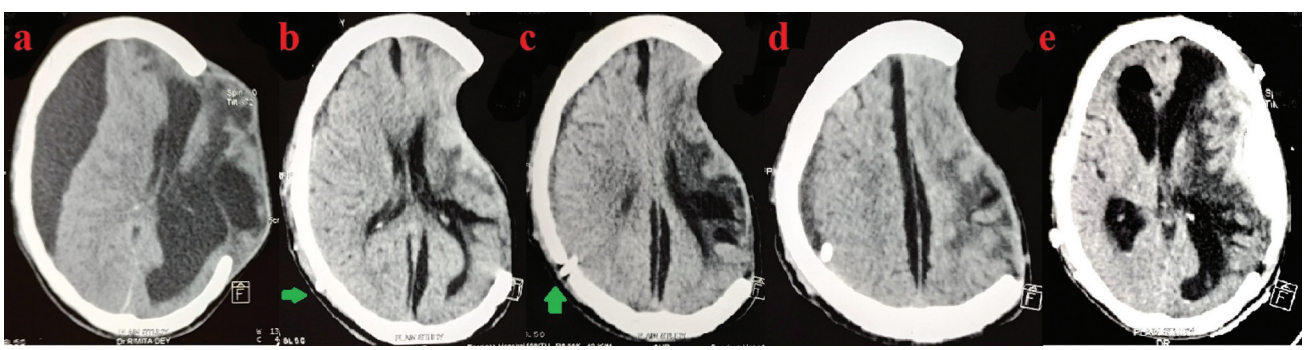

Fig. 5 Pre-CP axial CT scan image (**A**) of a patient operated for left-sided acute subdural hematoma and contusions showing contralateral (right sided) hygroma with external cerebral herniation and midline shift; Axial images (**B–D**) showing concave flap after placement of a subduroperitoneal shunt and (**E**) extradural hemorrhage below the replaced flap after CP.

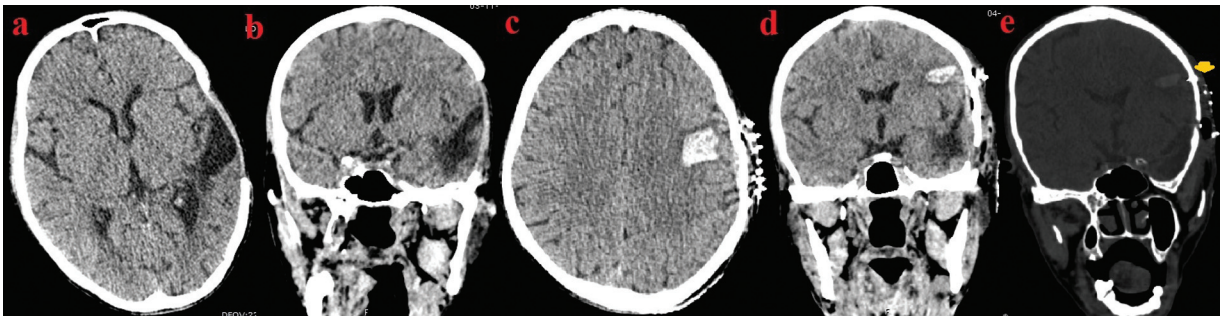

Fig. 6 Pre-CP CT scan images (**A, B**) of a patient operated for left temporal intracerebral hemorrhage showing gliosis at the site of surgery; post CP CT images (**C, D**) showing an intraparenchymal hemorrhage below the replaced flap but away from the gliotic region and (**E**) coronal bone windows showing a screw used for fixation of the bone (orange arrow) penetrating the full thickness and causing brain injury.

[►**Fig. 5**] or if a lumbar puncture (LP) is done preoperatively to slacken the brain prior to CP as this decreases the natural tamponading effect the latter has on capillary ooze from the dissected neo-dura and also in cases when bone cement used to fashion an artificial bone flap obliterates the temporo-basal gap through which any collection can egress to the subgaleal space.

A "wait and watch" policy can be followed if the patient is asymptomatic but significant neurological deficits mandates re-exploration, evacuation of the blood and replacement of the bone flap after drilling a few small holes in it along with drains (in the subgaleal and extradural planes).

Intraparenchymal Hemorrhage

Intraparenchymal hemorrhage after CP can occur due to iatrogenic injury to fragile blood vessels formed following posttraumatic angiogenesis[12] while lifting the skin and galeal flap off the neodura dural cover during exposure or due to screws used to fix the bone [►**Fig. 6**]. The former etiology is likely to occur more frequently in cases where adequate duraplasty is not done during the initial surgery.[13] Also, negative pressure suction drains might cause traction injury to these vessels.

Hyperperfusion of underlying brain can cause sudden increase in cerebral blood flow once atmospheric pressure is removed following CP and may lead to intraparenchymal bleed particularly in patients with a sunken flap.[14] If the injury is small with no mass effect, midline shift or cisternal effacement on CT scan, conservative treatment may be continued. Surgical evacuation is warranted if the hemorrhage is large and life-threatening and then the bone flap should again not be replaced.

Epidural Fluid Collection/Sub-flap Hygroma

The incidence of epidural fluid collections seen on CT scan below the replaced flap varies from 6.1% to 37.3%.[15] The vast majority are asymptomatic resolving over time.[15,16] They are typically hypodense as opposed to extradural hematomas. Factors such as dural stiffening preventing brain expansion after CP,[15] inflammatory response to artificial bone substitutes,[15] and intraoperative cerebrospinal fluid (CSF) leak have been implicated in its causation.[15,16]

When they are large enough to cause mass effect on the brain with fresh deficits [►**Fig. 7**], treatment options include making a burr hole through the flap to let the fluid out, or craniotomy and evacuation of the collection with placement of subgaleal drain and dural tenting sutures.

Flap Resorption

It is a delayed complication of CP, previously called aseptic necrosis of the bone flap and is of two types[17]–Type 1

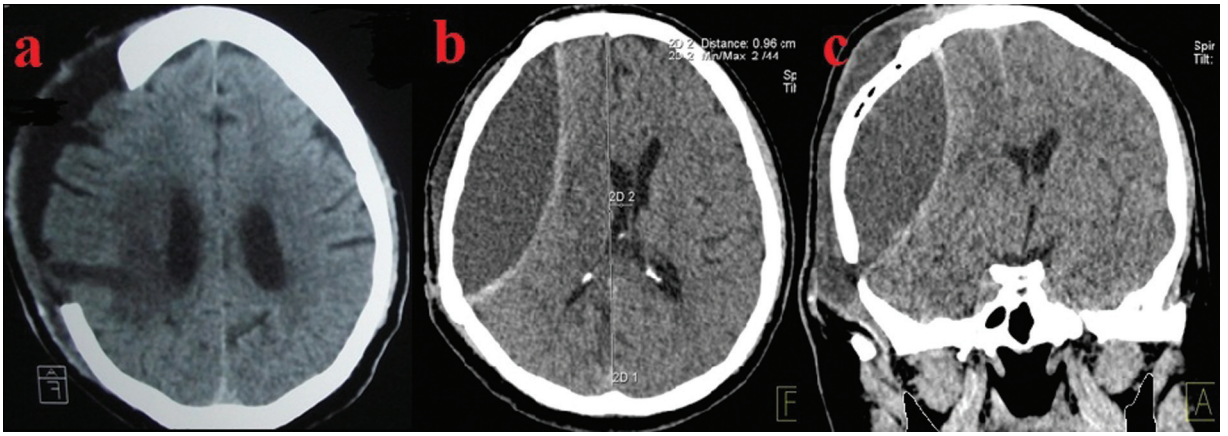

Fig. 7 Pre-CP CT scan images (**A**) showing a "full" flap that necessitated LP to make space and seat the bone flap; postoperative CT scans (**B, C**) showing sub-flap epidural fluid collection causing mass effect and midline shift.

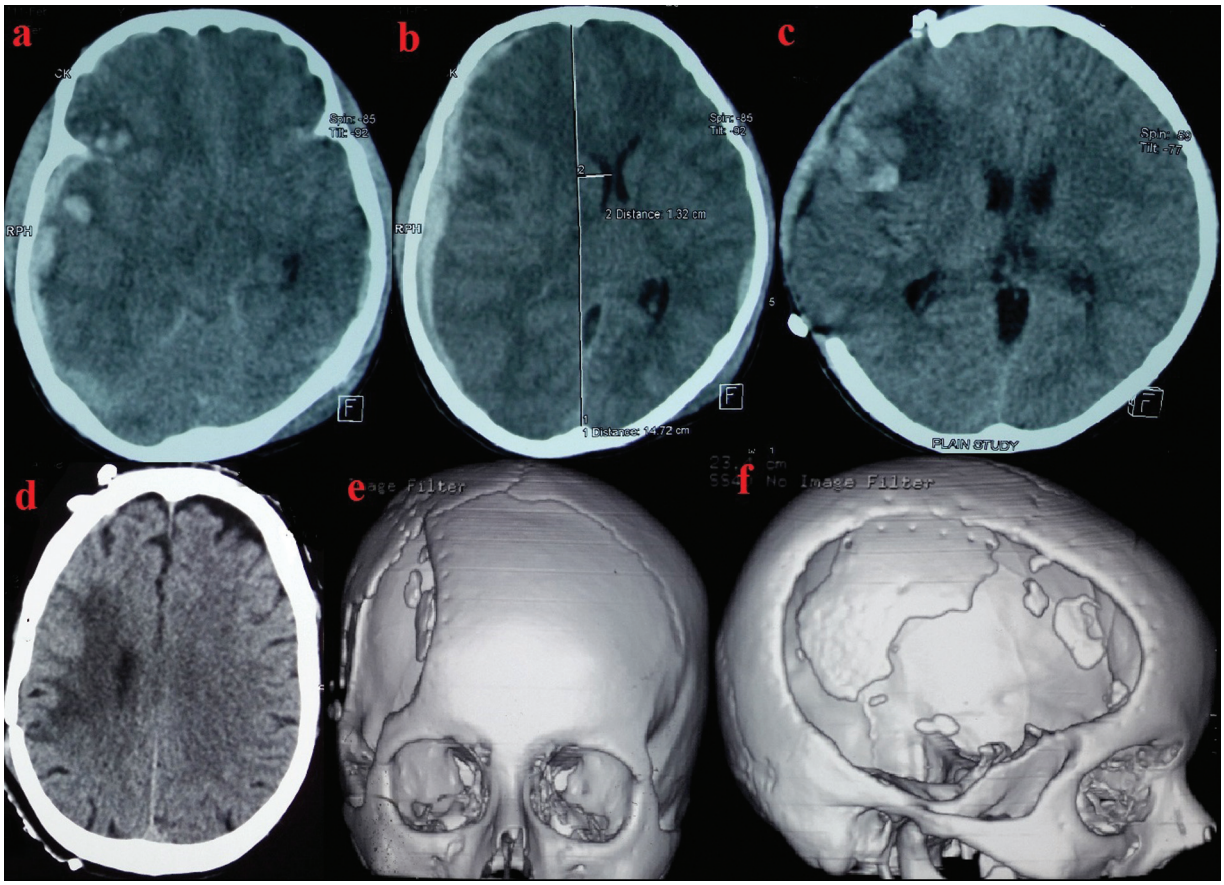

Fig. 8 Preoperative CT scans (**A, B**) showing a right-sided acute subdural hematoma with contusions causing mass effect and midline shift; post DC CT image (**C**) showing decrease of mass effect; immediate post CP image (**D**) showing replaced bone flap and 3D reconstructed CT scan images after 2 years (**E, F**) showing significant resorption of the bone flap with only patchy islands remaining intact.

where there is thinning of the bone or erosion at the margins of the flap and Type 2 where there is complete lysis of both tables within the flap. The incidence of this complication depends on the length of the follow-up and the attention with which it is sought for and varies from 7.2% to 50%.[17]

Multifragmented bone flaps, late CP, larger flaps, younger age of the patient, CP done for trauma, and the presence of a VP shunt have been implicated as predisposing factors for the same.[17,18] Patients present with progressive softening of the operative site and rarely pain and CT scans (with 3D reconstructions) show the extent of resorption [►**Fig. 8**]. Management options include re-CP with cement or mesh or an expectant "wait and watch" policy in children as re-calcification may occur.[19]

Flap Mobility

A flap that is not firmly fixed (as with sutures instead of miniplates and screws) may on occasion move with variations in intracranial pressure (occurring with coughing/sneezing) or with posture too. While most patients will have non-serious complaints such as a subjective fullness of the operative site on getting up from sleep which sinks inward somewhat as the day progresses, abnormal mobility of the implanted bone may cause symptoms and called the "Sinking Bone Syndrome."[20] We have documented a case of reversible postural hemiparesis[21] where there was variation in MCA flow with flap movement [►**Fig. 9**].

Flap mobility can also occur due to incompatible flap size due to its absorption during storage. We have encountered it in cases where we had placed the flap in a subcutaneous abdominal pocket and when the patient underwent a delayed CP [►**Fig. 10**]. It is probably due to absorption and decalcification of the bone during storage in a metabolically active location.[22]

Flap Subsidence

A flap that is inadequately fixed during CP may sink into the brain if LP is done to rule out meningitis [►**Fig. 11**] or after ventriculoperitoneal shunting. Neurological deterioration may occur due to disturbances in cerebral metabolism (documented using flurodeoxy glucose positron emission tomography studies) due to decreased cerebral blood flow by the pressure of the flap.[21]

Extrusion of Implants Used for Fixation

We encountered a previously unreported complication that occurred due to back out of the miniplates and screws used to fix the bone flap during CP with erosion through the skin [►**Fig. 12**]. Treatment entails removal of the extruded screws

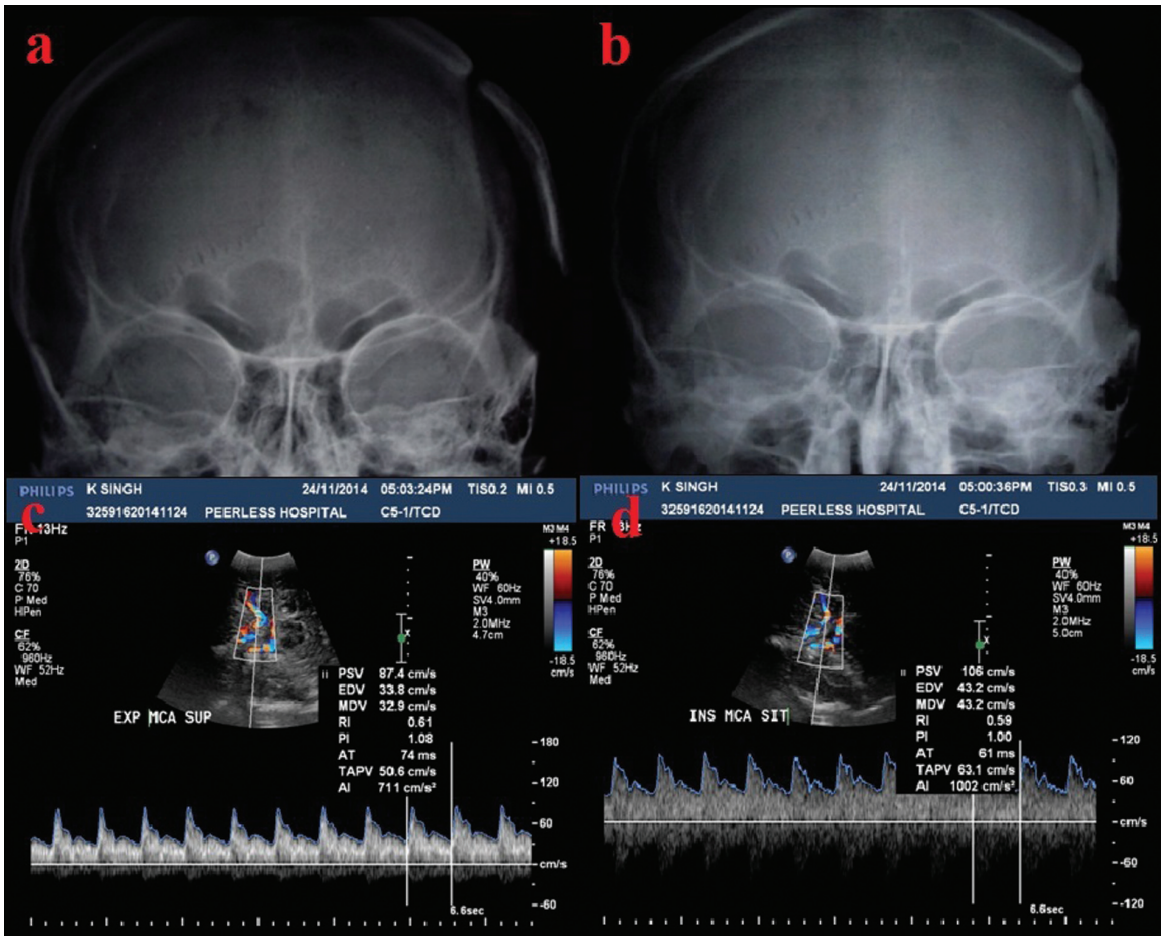

Fig. 9 Anteroposterior plain skiagrams of a post CP mobile flap in supine position with full expiration (**A**) and sitting position with full inspiration (**B**) showing outward and inward mobility respectively. Transcranial doppler (**C, D**) showing increase in peak systolic velocity in the middle cerebral artery when the flap drifts inwards compared with that in supine position.

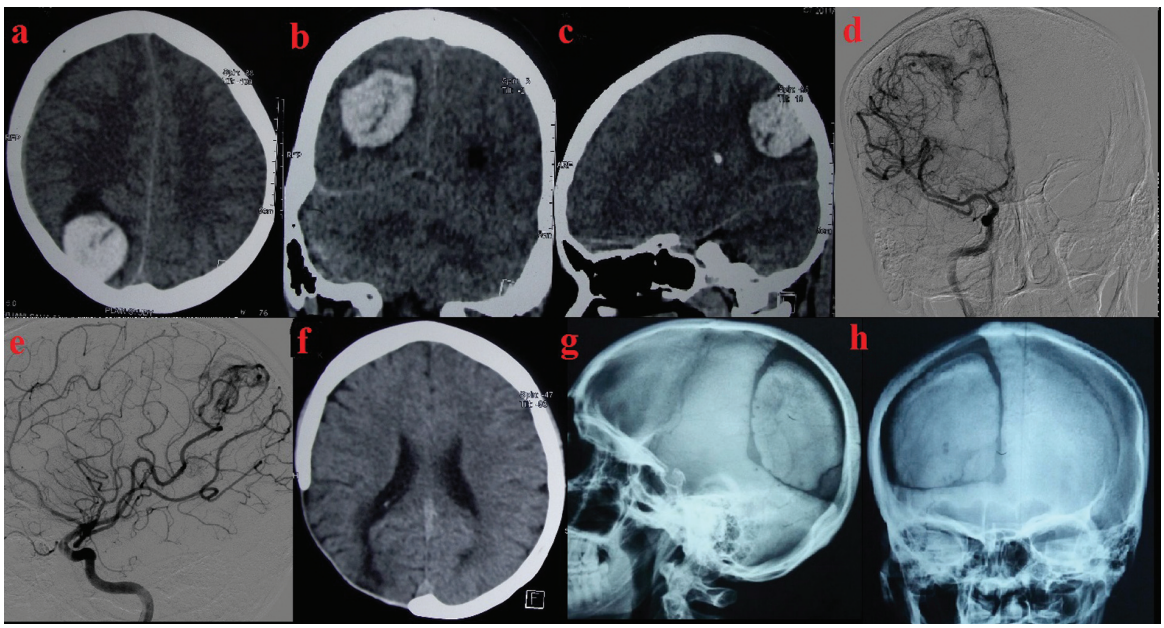

Fig. 10 Preoperative CT scan images (**A–C**) showing a spontaneous right sided parietal intracerebral hemorrhage; preoperative digital subtraction angiography showed an arteriovenous malformation fed by branches of the middle cerebral artery following excision of which bone flap was placed in a subcutaneous abdominal pocket; pre CP CT scan image (**D**) and postoperative skiagrams after the patient complained of postural flap mobility (**E, F**) showing incompatible flap size due to absorption in storage.

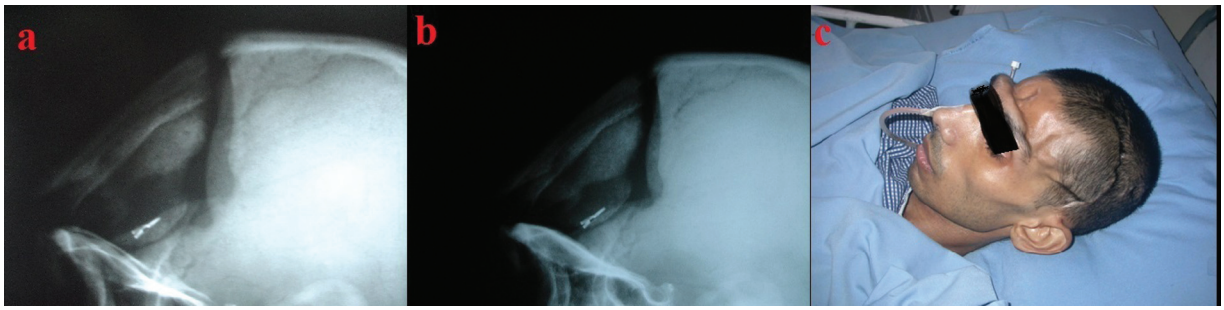

Fig. 11 Plain skiagrams showing a replaced bifrontal CP flap initially created for hematoma evacuation and clipping of a distal anterior cerebral artery aneurysm at first follow up (**A**) and immediately following lumbar puncture 6 months later for meningitis (**B**) following which he became drowsy instantaneously and clinical photograph (**C**) after lumbar puncture before reinstallation of saline in the thecal sac.

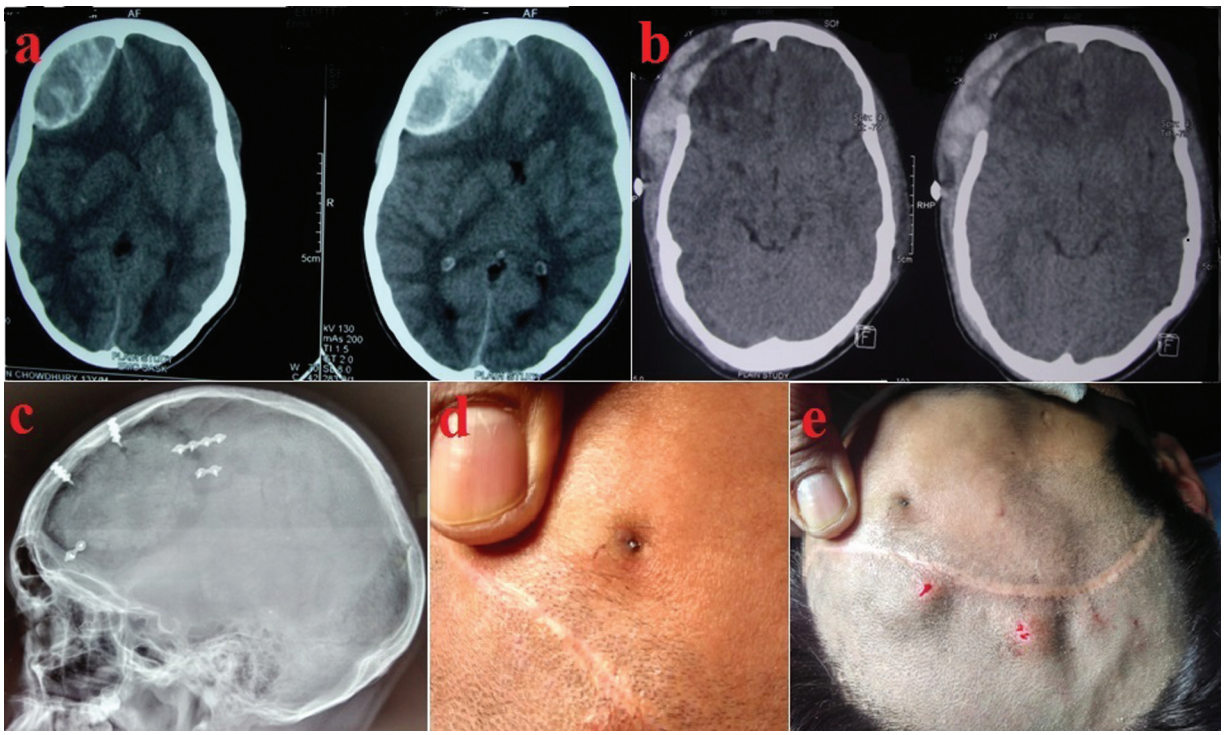

Fig. 12 Preoperative CT scans (**A**) of a patient with right sided extradural hematoma; postoperative CT scans (**B**) after DC and clot evacuation; post CP skiagram (**C**) showing the implants used to anchor the bone flap and clinical photographs (**D**,**E**) showing erosion of implant through the skin at one place and elevation of the wound at two other places due to implant back out.

and loosened plates and re-fixation at another site if multiple fixation points are compromised or removal of just the offending implant if localized to one level.

Conclusions

CP is a common but far from uncomplicated surgical procedure. Though we have not analyzed the reason for every complication in each patient, it must be stressed that post-CP complications are myriad and at times may be serious enough to threaten life or warrant resurgery. Attention to small but basic surgical techniques will go a long way in preventing unwanted postoperative events.

Conflict of Interest
None declared.

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