Complications of Decompressive Craniectomy: A Case-Based Review

Sivaraman Kumarasamy[1] Kanwaljeet Garg[1] Hitesh Kumar Gurjar[1] Kokkula Praneeth[1] Rajesh Meena[1] Ramesh Doddamani[1] Amandeep Kumar[1] Shashwat Mishra[1] Vivek Tandon[1] Pankaj Singh[1] Deepak Agrawal[1]

[1] Department of Neurosurgery, All India Institute of Medical Sciences, New Delhi, India

Indian J Neurotrauma 2023;20:71–80.

Address for correspondence Kanwaljeet Garg, MCh, DNB, FACS, Department of Neurosurgery, Room no 720, CNC, All India Institute of Medical Sciences, New Delhi 110029, India (e-mail: kanwaljeet84@gmail.com).

Abstract

Background Decompressive craniectomy (DC) is a frequently performed procedure to treat intracranial hypertension following traumatic brain injury (TBI) and stroke. DC is a salvage procedure that reduces mortality at the expense of severe disability and compromises the quality of life. The procedure is not without serious complications.

Methods We describe the complications following DC and its management in a case-based review in this article.

Results Complications after DC are classified as early or late complications based on the time of occurrence. Early complication includes hemorrhage, external cerebral herniation, wound complications, CSF leak/fistula, and seizures/epilepsy. Contusion expansion, new contralateral epidural, and subdural hematoma in the immediate postoperative period mandate surgical intervention. It is necessary to repeat non-contrast CT head at 24 hours and 48 hours following DC. Late complication includes subdural hygroma, hydrocephalus, syndrome of the trephined, bone resorption, and falls on the unprotected cranium. An early cranioplasty is an effective strategy to mitigate most of the late complications.

Conclusions DC can be associated with a number of complications. One should be aware of the possible complications, and timely intervention is required.

Keywords

- complications
- cranioplasty
- decompressive craniectomy
- intracranial hypertension
- traumatic brain injury

Introduction

Decompressive craniectomy (DC) is a widely used neurosurgical procedure for raised intracranial pressure (ICP). It is a salvage procedure in patients with refractory intracranial hypertension and cerebral edema when the medical measures fail to control the ICP. An increase in ICP is due to delayed hematoma or brain swelling within the fixed volume of the skull. DC can lower raised ICP and aid in improving brain tissue oxygenation. DC has been proposed to improve survival and clinical outcomes. The indications of DC are varied and include traumatic brain injury (TBI), malignant ischemic stroke, aneurysmal subarachnoid hemorrhage, nontraumatic hypertensive intracranial hemorrhage, and cerebral venous thrombosis.[1–4]

Two randomized, multicenter prospective trials, RESCUEicp and DECRA, were conducted to determine whether decompressive craniectomy alters the outcome in the management of TBI.[5–7] The DECRA trial concluded that patients managed with DC for refractory raised ICP had

The manuscript was never presented as part at a meeting, the organisation, place, and exact date on which it was read.

article published online February 9, 2023

DOI https://doi.org/10.1055/s-0043-1760724.

ISSN 0973-0508.

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

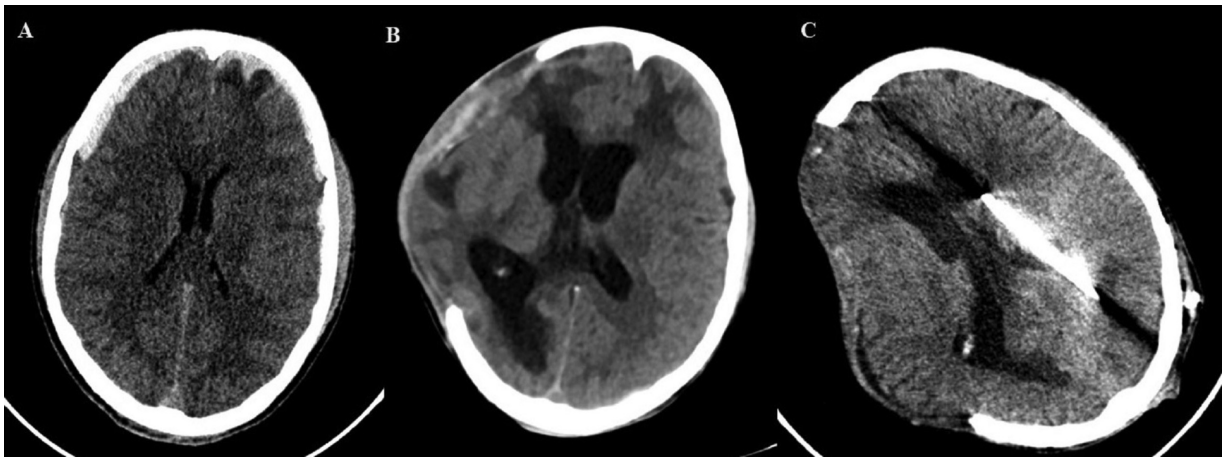

Fig. 1 Post decompressive craniectomy hydrocephalus. Noncontrast CT head showing (**A**) a bilateral frontotemporal hemorrhagic contusion with bilateral frontoparietal acute SDH with a thickness of 6.4 mm on right and 4.5 mm on left with a midline shift of 3.8 mm. (**B**) Postcraniectomy with features of hydrocephalus managed by CSF diversion (**C**).

decreased mean ICP and duration of ICU stay compared to standard therapy but with a significantly worse outcome at 6 months.[6] A meta-analysis showed the DC results in an improvement in the survival following TBI, while there was no change in the functional outcome.[8]

A multicenter, open, randomized trial, HAMLET, interpreted that surgical decompression for space-occupying cerebral infarction within 48 hours of stroke onset reduces case fatality and poor outcomes.[9]

DC has many known complications with rates as high as 53.9%.[10] Long-term deleterious neurocognitive and psychosocial effects leading to poor quality of life and social burden are well known following DC.[11] The possible complications of DC should be kept in mind, and it should be done only when absolutely indicated. In this case-based review, we aim to describe some of the common complications of DC done in patients with TBI that we encountered.

For ease of understanding, complications following DC are classified as early or late complications based on the time of occurrence following DC. Early complications are those that occur in the initial 4 weeks. Early complications, usually, occur in the same hospital admission as DC. Early complications include hemorrhagic complications (contralateral hematoma, hemorrhagic progression of contusion, and hemorrhagic transformation of infarction), external cerebral herniation, wound complications, CSF leak/fistulae, postoperative infection, and seizures. Late or delayed complications include altered CSF hydrodynamics (subdural hygroma and hydrocephalus) and the syndrome of the trephined (sunken flap syndrome).

Case 1–Post Decompressive Craniectomy Hydrocephalus

A 20-year-old male patient met with a road traffic accident where the two-wheeler was hit by a four-wheeler and sustained an injury to his head. His GCS was E1VTM5 on presentation. His noncontrast CT head showed a bilateral frontotemporal hemorrhagic contusion with bilateral frontoparietal SDH with a thickness of 6.4 mm on right and 4.5 mm on left with a midline shift of 3.8 mm (►**Fig. 1A**). The patient underwent right FTP DC. Patient GCS gradually improved to E4V5M6. On postoperative day 10, the patient developed deterioration of GCS to E3V1M5, and repeat noncontrast CT head showed features of hydrocephalus (►**Fig. 1B**). The patient underwent CSF diversion via left occipital horn with medium pressure ventriculoperitoneal shunt (►**Fig. 1C**). Patient's GCS gradually improved to E4V5M6.

Case 2–Ipsilateral New Onset Hematoma with Mass Effect

A 73-year-old male patient, a known case of diabetes mellitus, hypertension, and dementia on dual antiplatelets, was brought with an alleged history of fall from the bed. He was conscious since the fall. On presentation, his GCS was E1VTM4. His noncontrast CT head showed left temporoparietal acute SDH with a thickness of 1 cm and midline shift of 9 mm. The acute SDH extended along the falx cerebri up to the tentorium and had a maximum thickness of around ~ 1.6 cm (►**Fig. 2A**). The patient was transfused with blood products in view of deranged blood coagulation parameters. He underwent left FTP DC once his coagulation parameters improved. Intraoperatively, there was excessive bleeding, and all measures to achieve hemostasis were taken. The patient was managed in the ICU in the immediate postoperative period. Postoperative noncontrast CT head showed bleeding in the ipsilateral cerebral hemisphere with significant mass effect (►**Fig. 2B**). Patient's GCS gradually deteriorated to E1VtM1, and he died on postoperative day 3.

Case 3–Meningitis with Post Decompressive Craniectomy Hydrocephalus

A 22-year-old male patient met with a road traffic accident when his two-wheeler skid and fell where he was a helmeted pillion rider. His GCS was E1VTM1 on presentation to our

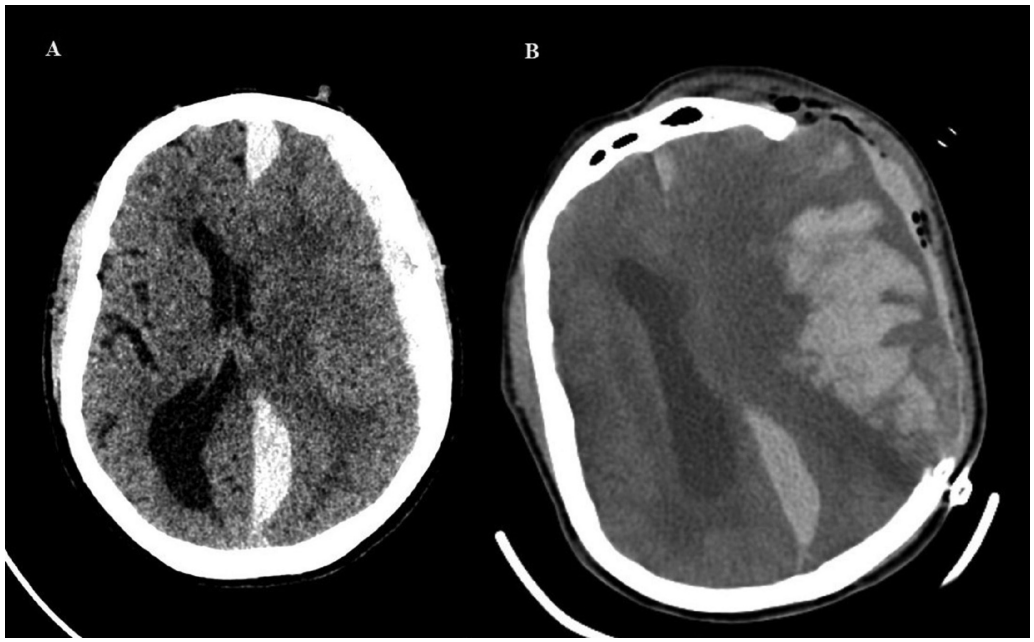

Fig. 2 New ipsilateral hematoma. Noncontrast CT head showing (**A**) left temporoparietal acute SDH with a thickness of 1 cm and a midline shift of 9 mm. (**B**) Postcraniectomy with bleed in the ipsilateral cerebral hemisphere with significant mass effect.

trauma center. His noncontrast CT head showed a large right FTP acute SDH with a midline shift of 1.3 cm and uncal herniation (►**Fig. 3A**). The patient underwent right FTP DC. The patient's GCS gradually improved to E2VtM5 in the postoperative period. The patient developed a persistent fever. Guarded lumbar puncture was done as his urine and blood cultures revealed no bacterial or fungal growth. CSF workup was found to be meningitic and the CSF culture grew *Acinetobacter baumannii*. The patient was started on intravenous antibiotics based on the culture sensitivity of the organism. His GCS deteriorated to E2VtM3. Repeat noncontrast CT head revealed the presence of hydrocephalus (►**Fig. 3B**). An urgent external ventricular draining catheter was inserted in the left frontal horn (►**Fig. 3C**) and it drained ~ 240 mL/day. Despite this, his GCS deteriorated to E1VTM1, and had persistent fever not responding to intravenous antibiotics. The patient was started on inotropic support for hemodynamic instability. The patient was hemodynamically unstable and died on postoperative day 18.

Case 4–Subdural Hygroma

A 2-year-old child presented to our trauma center following a fall from height (approximately 20 feet). On presentation, the GCS was $E1V_{cry}$ $M6_{SPONT}$. His non-contrast CT head showed elevated right frontal fracture extending to the base of the skull with right frontal lobe hemorrhagic contusion with significant mass effect and midline shift of 8 mm (►**Fig. 4A**). Patient underwent right FTP DC and excision of the contusion. The patient's postoperative period was uneventful. Later, the patient developed flap site swelling

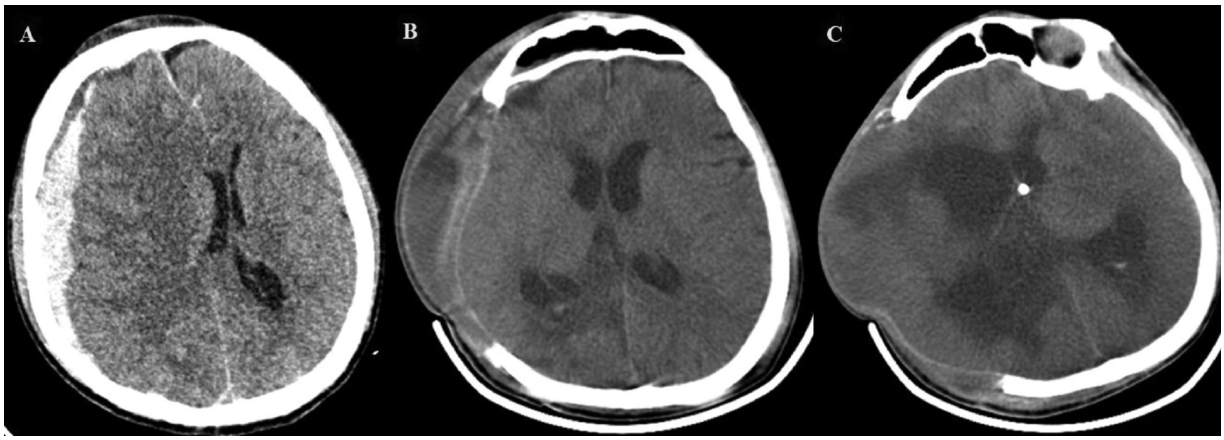

Fig. 3 Meningitis with post-DC hydrocephalus. Noncontrast CT head showing (**A**) a large right FTP SDH with mass effect, a midline shift of 1.3 cm, and uncal herniation. (**B**) Postcraniectomy with the presence of hydrocephalus. (**C**) External cerebral herniation with hydrocephalus with left frontal external ventricular drainage in situ.

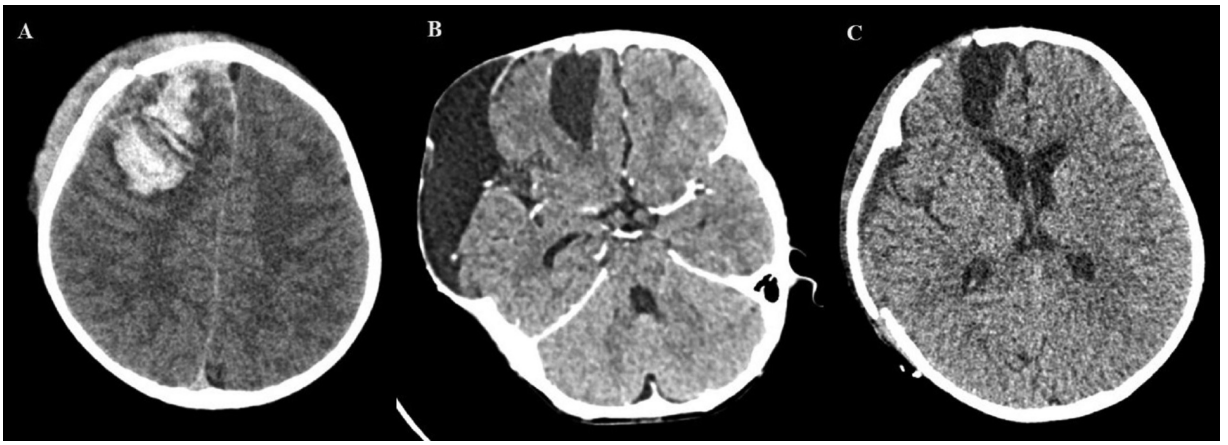

Fig. 4 Subdural hygroma. Noncontrast CT head showing (**A**) elevated right frontal fracture extending to the base of the skull with right frontal lobe hemorrhagic contusion with significant mass effect and a midline shift of 8 mm. (**B**) Postcraniectomy with right subdural hygroma with no mass effect. (**C**) Subdural hygroma treated by early autologous cranioplasty.

in the right frontal region. On examination, there was a bulge that was cystic in nature. Postoperative noncontrast CT head showed right subdural hygroma with no mass effect (►**Fig. 4B**). The patient underwent early autologous cranioplasty following drainage of the hygroma (►**Fig. 4C**), 5 weeks after DC.

Case 5–Contralateral New EDH

A 17-year-old male patient met with a road traffic accident of unknown mechanism. His GCS was E2VTM5 on presentation to our trauma center. His noncontrast CT head showed left FTP SDH and falcine acute SDH with mass effect and midline shift of 8 mm with effacement of cisterns (►**Fig. 5A**). The patient underwent left FTP DC. Immediate postoperative noncontrast CT head showed a right parietal EDH with mass effect, which was not there in the earlier scan (►**Fig. 5B**). He underwent right parietal craniotomy and EDH evacuation (►**Fig. 5C**). His GCS gradually improved to E4VtM5 at the last follow-up of 10 days following trauma.

Case 6–Wound Necrosis with CSF Leak

A 60-year-old female patient met with a road traffic accident. Her GCS was E1VtM4 on presentation to our trauma center. Her noncontrast CT head showed left FTP acute SDH with midline shift of 1.1 cm with left frontotemporal contusion (►**Fig. 6A**). The Patient underwent left FTP DC (►**Fig. 6B**). The patient developed wound infection in the postoperative period. On postoperative day 6, she developed wound necrosis with dehiscence followed by a CSF leak/fistula (►**Fig. 7A**). The patient underwent a triple rhombic flap cover (►**Fig. 7B**).

Case 7–External Cerebral Herniation

A 55-year-old male patient met with a road traffic accident where he was a non-helmeted two-wheeler driver. His vehicle hit another two-wheeler head-on, and he sustained an injury to his head. His GCS was E1VtM4 on presentation. His noncontrast CT head showed left FTP acute SDH of 12 mm thickness with a midline shift of 1.6 cm and left

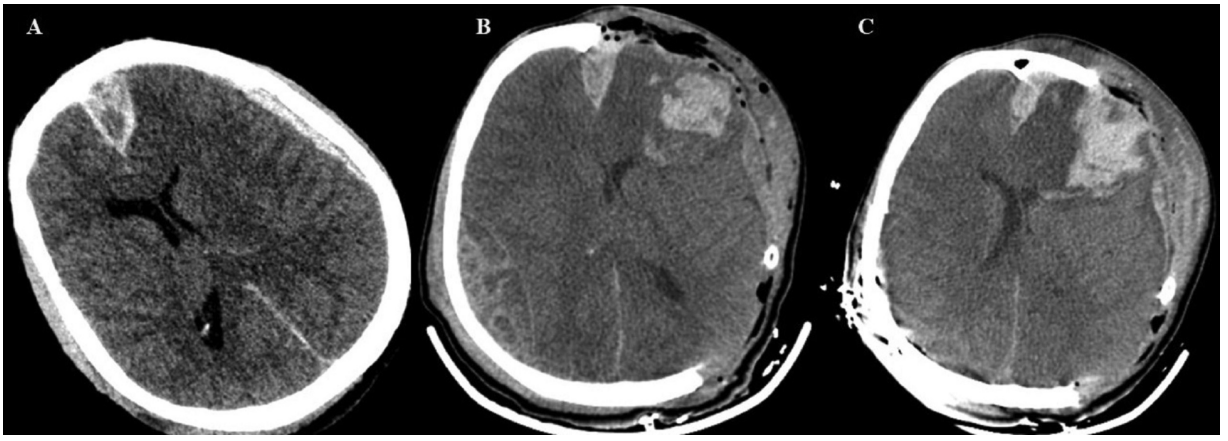

Fig. 5 New contralateral hematoma. Noncontrast CT head showing (**A**) left FTP SDH and falx SDH with mass effect and a midline shift of 8 mm with effacement of cisterns. (**B**) Postcraniectomy showed new ipsilateral hematoma with contralateral right parietal EDH. (**C**) Postright parietal craniotomy and EDH evacuation.

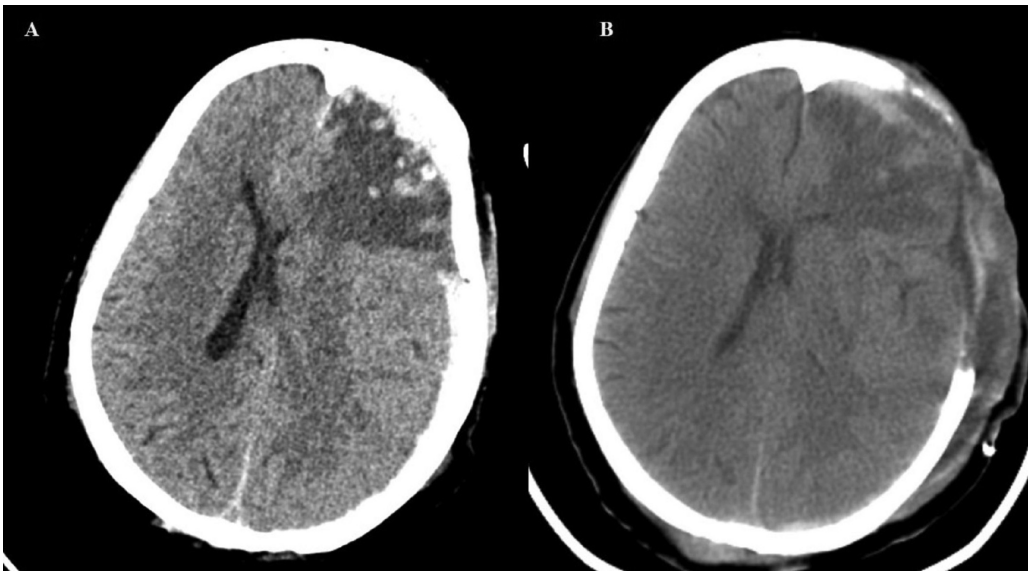

Fig. 6 Non-contrast CT head showing (**A**) left FTP SDH with a midline shift of 1.1 cm with left frontotemporal contusion who underwent (**B**) left FTP DC.

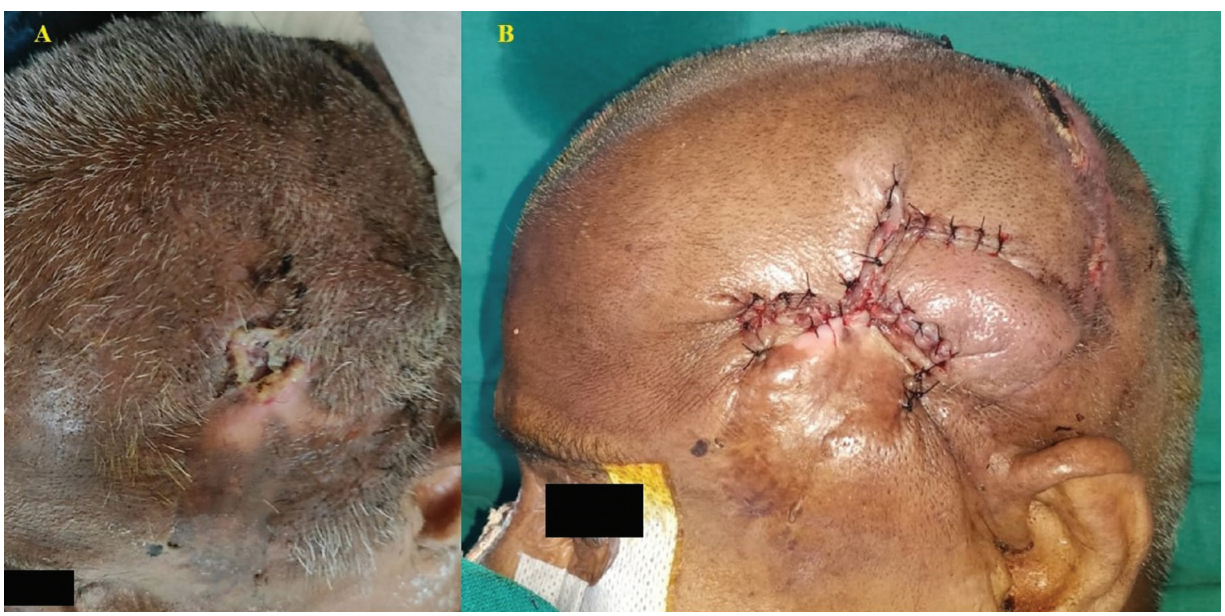

Fig. 7 Wound necrosis. Postoperative wound infection with necrosis and CSF leak/fistula (**A**) which was managed by triple rhombic flap cover (**B**).

frontotemporal hemorrhagic contusion (►**Fig. 8A**). He underwent left FTP DC. His GCS gradually improved to E3VtM5. In the postoperative period, the patient developed left external cerebral herniation (►**Fig. 8B**) with 1.61 cm of herniated brain tissue through the craniectomy site and left subdural hygroma. The patient was managed conservatively with decongestants and his GCS was E4VtM5 at the last follow-up 12 days following trauma.

Case 8–Syndrome of the Trephined

A 35-year-old male patient was brought following a fall from a tree. His GCS was E1VtM5 on presentation. His noncontrast CT head showed bilateral basifrontal contusion with acute SDH with a midline shift of 8 mm (►**Fig. 9A**). He underwent bifrontal decompressive craniectomy and placement of bone flap in the abdominal pouch. His GCS gradually improved to E4VtM6. The Patient was lost to follow-up during the COVID-19 pandemic and presented to us 2 years later with a bifrontal calvarial defect with sunken flap syndrome (►**Fig. 9B**). He was conscious and fully alert and oriented, though his family members complained that he had some memory disturbance. His autologous bone flap in the abdomen got partially resorbed. The patient then underwent patient-specific implant cranioplasty (►**Fig. 9C**). He had no neurological deficits at the last follow-up 12 days following cranioplasty.

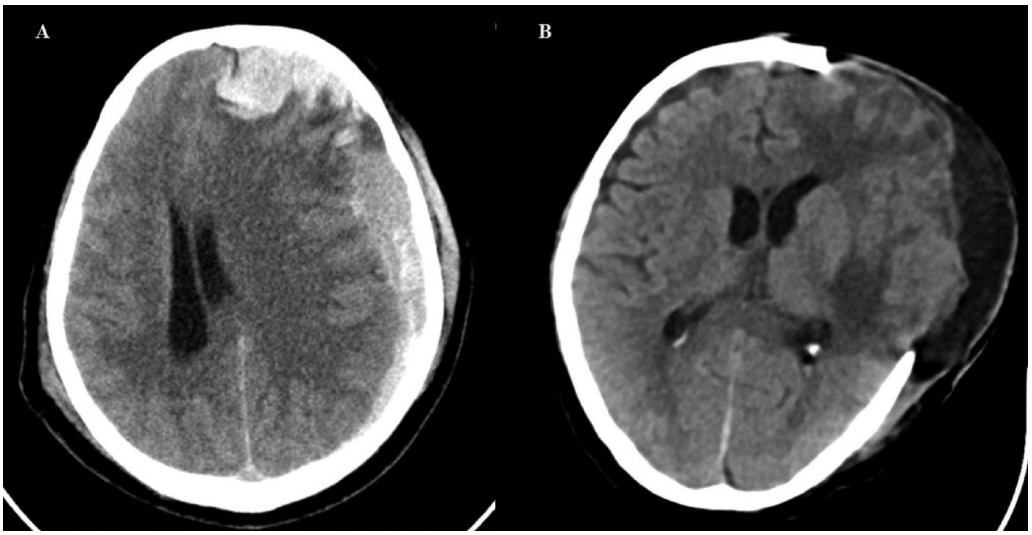

Fig. 8 External cerebral herniation. Noncontrast CT head showing (**A**) left FTP SDH of 12 mm thickness with a midline shift of 1.6 cm and left frontotemporal hemorrhagic contusion. (**B**) Postcraniectomy with external cerebral herniation with 1.61 cm of herniated brain tissue through the craniectomy site.

Case 9–Subdural Hygroma

A 26-year-old male patient met with a road traffic accident of unknown mechanism. His GCS was E1VtM2 on presentation to our trauma center. His noncontrast CT head showed left frontoparietal acute SDH with a midline shift of 5 mm with bilateral frontotemporoparietal sulcal SAH (►**Fig. 10A**). He underwent left FTP DC (►**Fig. 10B**). His GCS gradually improved to E2VtM3. In the postoperative period, the patient developed contralateral frontoparietal subdural hygroma (►**Fig. 10C**). He underwent burrhole and drainage of hygroma. His GCS was E2VtM3 on last follow-up 3 weeks following trauma.

Case 10–Bone Resorption

An 8-year-old male patient met with a road traffic accident where he was a pedestrian hit by a two-wheeler. His GCS was E1VtM4 on presentation to our trauma center. His noncontrast CT head showed left frontotemporoparietal EDH with fracture of the left temporal bone (►**Fig. 11A**). He underwent left FTP craniotomy and evacuation of EDH. In ICU, he underwent ICP monitoring in the postoperative period and it showed raised ICP. He then underwent left FTP DC on postoperative day 2 (►**Fig. 11B**). His GCS gradually improved to E4V5M6. He underwent autologous cranioplasty 94 days following trauma (►**Fig. 12A**). His postoperative period was uneventful. On follow-up, the left FTP bone flap got resorbed within 20 months of cranioplasty (►**Fig. 12B**). The patient then underwent patient-specific implant cranioplasty (►**Fig. 12C**). He had no neurological deficits following PSI cranioplasty.

Discussion

There is no doubt that DC has become a valuable tool in the management of intractable intracranial hypertension due to various pathologies. Most papers have focused on the risk

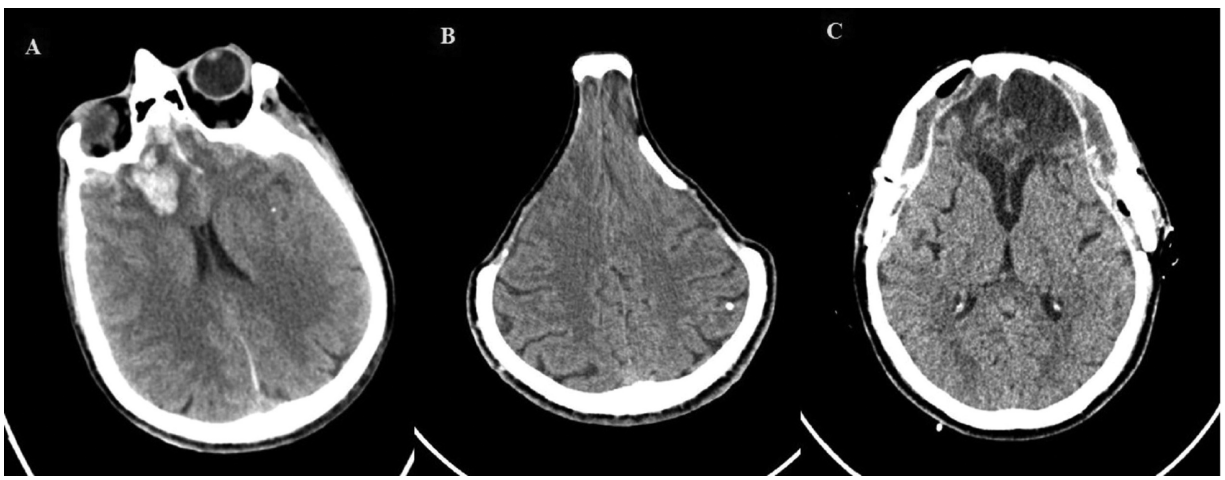

Fig. 9 Syndrome of the trephined. Noncontrast CT head showing (**A**) bilateral basifrontal contusion with SDH with a midline shift ~ 8 mm. (**B**) Sunken flap syndrome that was managed by patient-specific implant cranioplasty (**C**).

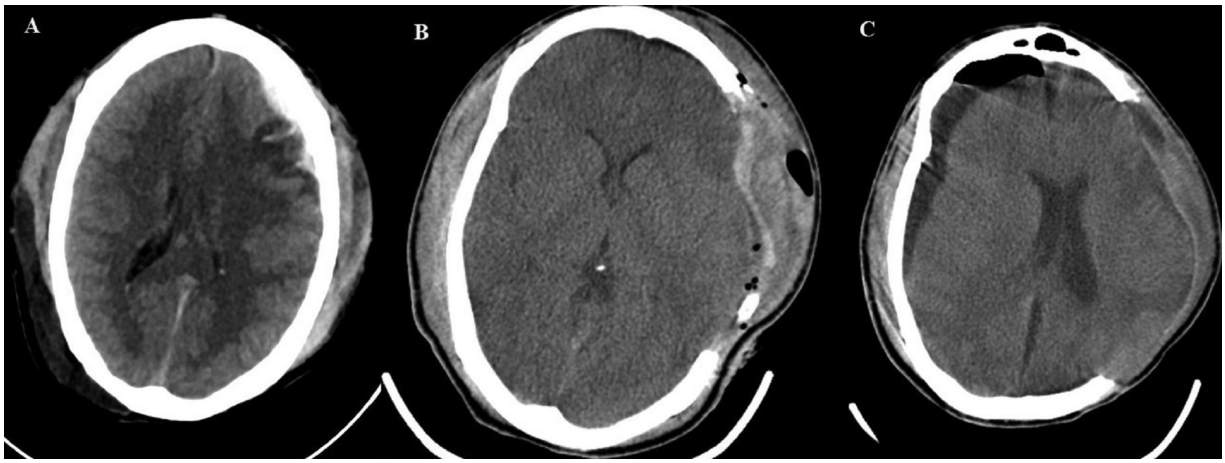

Fig. 10 Contralateral subdural hygroma. Noncontrast CT head showing (**A**) left frontoparietal acute SDH with a midline shift of 5 mm with bilateral frontotemporoparietal sulcal SAH. (**B**) Left FTP DC. (**C**) Contralateral frontoparietal subdural hygroma.

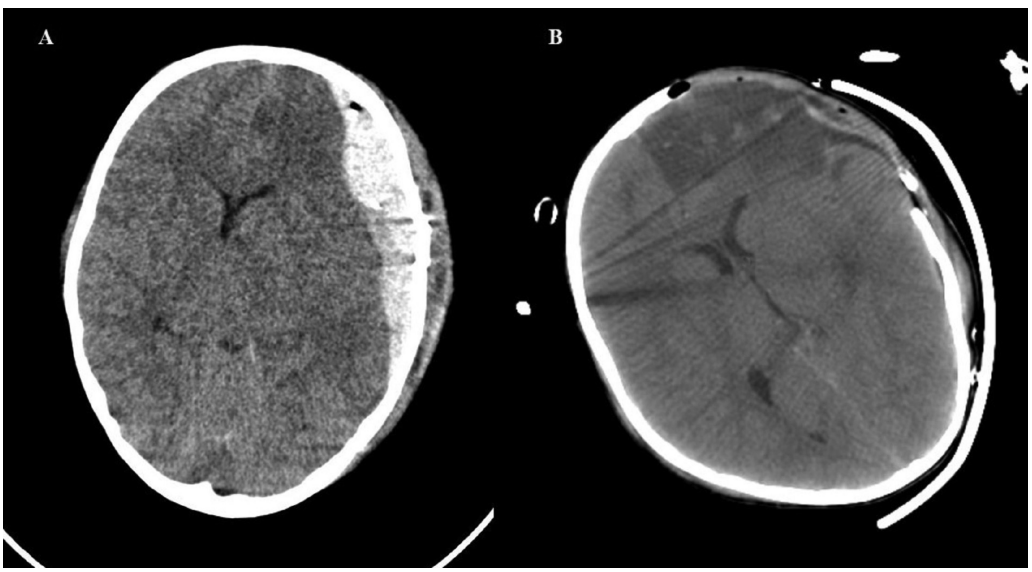

Fig. 11 Noncontrast CT head showing (**A**) left frontotemporoparietal EDH with fracture of the left temporal bone. (**B**) Left FTP DC.

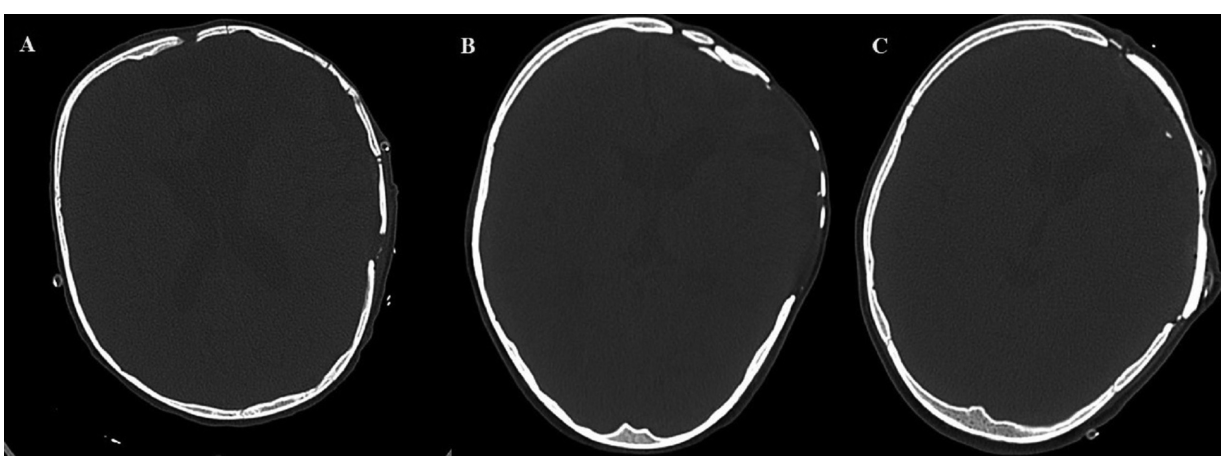

Fig. 12 Bone resorption. (**A**) Post autologous cranioplasty with bone resorption (**B**) which was managed by patient-specific implant cranioplasty (**C**).

factors, techniques, and outcomes but relatively a few have comprehensively discussed the complications of the DC.[12]

Early Complications of DC

Hemorrhage

Hemorrhagic progression of contusion which is conservatively managed, contralateral hematoma, and other hemorrhagic complications usually occur early following DC (►**Figs. 2B** and **5B**). Most expansion occurs in the immediate postoperative period, which may lead to neurological deterioration, prolonged ICU stays, and may even be fatal. It is due to the loss of tamponade effect after the removal of the bone, along with a fall in ICP which leads to expansion of the hematoma.

A new hematoma mandated a second craniotomy in 50% of TBI patients in one study.[13] Repeat CT head in the immediate postoperative period and 48 hours later help in the timely diagnosis of these bleeds and early treatment. We routinely get an early postoperative noncontrast CT scan in all patients undergoing DC for TBI.

External Cerebral Herniation

An external cerebral herniation is defined as more than 1.5 cm of herniated brain tissue through the center of the craniectomy defect[14] (►**Fig. 8B**). It is due to altered hydrostatic gradients from the capillaries after decompression and edema from cerebral reperfusion. It can be avoided by large craniectomies and augmentative duraplasty. The Brain Trauma Foundation recommends a large FTP DC (12×15 cm diameter) in severe TBI patients for reducing mortality and improving neurologic outcomes.[15]

Wound Complications

Wound complications after DC are dehiscence, ulceration, or necrosis. The factors that predispose to ischemic flap breakdown are the large size of the scalp flap and the injury to the superficial temporal artery during DC. This can be prevented by preserving the superficial temporal artery and limiting the posterior extension of the skin flap to less than 5 cm posterior to the pinna.[16] Wound complications are more following DC in TBI due to nonclean injuries and the presence of scalp lacerations.

Superficial wound infections following DC include wound breakdown, surgical site infections, and wound necrosis (►**Fig. 7A**). Deep infections include epidural abscesses and subdural empyema. Surgical site infections are particularly common following cranioplasty. Wound breakdown and infection can also occur in abdominal wounds where the bone flap is preserved.

CSF Leak/Fistulae

CSF leak refers to the escape of CSF into the extradural space. These terms are used interchangeably. Augmentative duraplasty and watertight scalp closure would prevent CSF leaks from the wound. A randomized control trial comparing watertight duraplasty with rapid-closure DC without watertight duraplasty showed no statistically significant difference in the development of CSF leak in the postoperative period.[17]

Seizures/Epilepsy

Postoperative epilepsy is due to reduced epileptogenic threshold and increased hyperexcitability. The incidence of postoperative epilepsy is less following DC for TBI when compared to malignant stroke.[18] Increasing the dosage or addition of another anticonvulsant is a reasonable strategy to follow in the first 2 weeks post-injury. Early cranioplasty has been proposed to mitigate the risk of seizures but lacks conclusive evidence.[19]

Late Complications of DC

Subdural Hygroma

A subdural hygroma is a frequent complication following DC. It is due to CSF flow abnormalities following decompression due to surgical disruption of the subarachnoid pathway of cerebrospinal fluid or increased cerebral perfusion pressure. It is commonly located in the subdural, subgaleal, or interhemispheric areas (►**Fig. 4B**). It can be reduced by early pressure dressing during the 7th to 10th postoperative day following DC[20] (►**Fig. 4C**). If there is a tense collection of fluid, by ball valve effect, it can cause pressure on the brain. This condition requires drainage of the collection and repair of the dural rent. Cranioplasty can be done in the same sitting if the brain is lax.

Hydrocephalus

Communicating type of hydrocephalus following DC is due to altered CSF flow dynamics (►**Figs. 1B** and **3B**). The development of hydrocephalus is associated with unfavorable outcomes.[21] It should be managed by CSF diversions such as external ventricular drainage or shunt procedure (►**Figs. 1C** and **3C**). Recently, endoscopic third ventriculostomy has been tried in post-traumatic hydrocephalus with limited success.[22] Early cranioplasty can mitigate the risk of hydrocephalus following DC.[23] Craniectomy without early cranioplasty is associated with an increased risk of post-traumatic hydrocephalus.[19] The risk factors for the development of post-traumatic hydrocephalus include advanced age, a higher score on the Fisher grading system, low post-resuscitation GCS, CSF infection, and delayed cranioplasty.[24]

Syndrome of the Trephined

Syndrome of the trephined or sunken flap syndrome was first reported by Grant and Norcross in 1939.[25] The sunken flap lacks bony support that causes altered cerebral blood flow and underlying cortical dysfunction (►**Fig. 9B**). Early cranioplasty, within 8 weeks after DC can mitigate this complication (►**Fig. 9C**). Delayed cranioplasty and bone flap resorption following cranioplasty can cause depression of the flap, which produces an unappealing cosmetic defect (►**Fig. 13A**). This requires reconstruction with mesh or bone cement (►**Fig. 13B**).

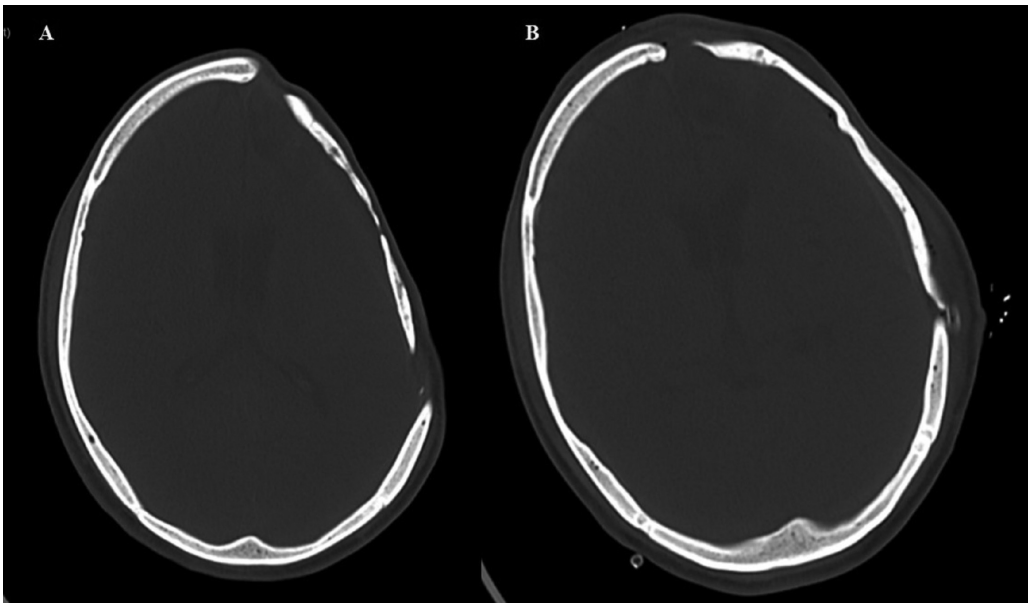

Fig. 13 Bone resorption. (**A**) Postcranioplasty with bone resorption and sunken flap, which was managed by replacement cranioplasty (**B**).

Falls on Unprotected Cranium

Accidental falls following DC can produce a higher chance of injury even with trivial trauma due to unprotected cranial contents. This can be prevented by early cranioplasty, close monitoring, and being cautious.

Conclusion

DC is a proven treatment for refractory raised ICP due to ischemic stroke or severe TBI. However, DC may be associated with a wide spectrum of complications that can negatively impact the functional outcome of the patient. These complications need to be anticipated early and treated accordingly.

Funding
None.

Conflict of Interest
None declared.

References

1 Hutchinson PJ, Kolias AG, Timofeev IS, et al; RESCUEicp Trial Collaborators. Trial of decompressive craniectomy for traumatic intracranial hypertension. N Engl J Med 2016;375(12): 1119–1130
2 Vahedi K, Hofmeijer J, Juettler E, et al; DECIMAL, DESTINY, and HAMLET investigators. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol 2007;6(03):215–222
3 Goedemans T, Verbaan D, Coert BA, et al. Decompressive craniectomy in aneurysmal subarachnoid haemorrhage for hematoma or oedema versus secondary infarction. Br J Neurosurg 2018;32(02):149–156
4 Zuurbier SM, Coutinho JM, Majoie CB, Coert BA, van den Munckhof P, Stam J. Decompressive hemicraniectomy in severe cerebral venous thrombosis: a prospective case series. J Neurol 2012;259(06):1099–1105
5 Hutchinson P, Corteen E, Czosnyka M, et al. Decompressive Craniectomy in Traumatic Brain Injury: the Randomized Multicenter RESCUEicp Study (www. RESCUEicp. com). Brain edema XIII: Springer; 2006:17–20
6 Cooper DJ, Rosenfeld JV, Murray L, et al; DECRA Trial Investigators Australian and New Zealand Intensive Care Society Clinical Trials Group. Decompressive craniectomy in diffuse traumatic brain injury. N Engl J Med 2011;364(16):1493–1502
7 Sharma R, Garg K. Role of decompressive craniectomy in traumatic brain injury—how much wiser are we after randomized evaluation of surgery with craniectomy for uncontrollable elevation of intracranial pressure trial? Neurosurgery 2017;81(05):E58–E60
8 Garg K, Singh PM, Singla R, et al. Role of decompressive craniectomy in traumatic brain injury–a meta-analysis of randomized controlled trials. Neurol India 2019;67(05): 1225–1232
9 Hofmeijer J, Kappelle LJ, Algra A, Amelink GJ, van Gijn J, van der Worp HBHAMLET investigators. Surgical decompression for space-occupying cerebral infarction (the hemicraniectomy after middle cerebral artery infarction with Life-threatening Edema Trial [HAMLET]): a multicentre, open, randomised trial. Lancet Neurol 2009;8(04):326–333
10 Ban SP, Son YJ, Yang HJ, Chung YS, Lee SH, Han DH. Analysis of complications following decompressive craniectomy for traumatic brain injury. J Korean Neurosurg Soc 2010;48(03): 244–250
11 van Middelaar T, Nederkoorn PJ, van der Worp HB, Stam J, Richard E. Quality of life after surgical decompression for space-occupying middle cerebral artery infarction: systematic review. Int J Stroke 2015;10(02):170–176
12 Hanko M, Soršák J, Snopko P, Opšenák R, Zeleňák K, Kolarovszki B. Incidence and risk factors of early postoperative complications in patients after decompressive craniectomy: a 5-year experience. Eur J Trauma Emerg Surg 2021;47(05):1635–1647
13 Qiu W, Guo C, Shen H, et al. Effects of unilateral decompressive craniectomy on patients with unilateral acute post-traumatic brain swelling after severe traumatic brain injury. Crit Care 2009;13(06):R185

14 Yang XF, Wen L, Shen F, et al. Surgical complications secondary to decompressive craniectomy in patients with a head injury: a series of 108 consecutive cases. Acta Neurochir (Wien) 2008;150 (12):1241–1247, discussion 1248
15 Carney N, Totten AM, O'Reilly C, et al. Guidelines for the management of severe traumatic brain injury. Neurosurgery 2017;80(01):6–15
16 Kurland DB, Khaladj-Ghom A, Stokum JA, et al. Complications associated with decompressive craniectomy: a systematic review. Neurocrit Care 2015;23(02):292–304
17 Vieira E, Guimarães TC, Faquini IV, et al. Randomized controlled study comparing 2 surgical techniques for decompressive craniectomy: with watertight duraplasty and without watertight duraplasty. J Neurosurg 2018;129(04):1017–1023
18 Brondani R, Garcia de Almeida A, Abrahim Cherubini P, et al. High risk of seizures and epilepsy after decompressive hemicraniectomy for malignant middle cerebral artery stroke. Cerebrovasc Dis Extra 2017;7(01):51–61
19 Gopalakrishnan MS, Shanbhag NC, Shukla DP, Konar SK, Bhat DI, Devi BI. Complications of decompressive craniectomy. Front Neurol 2018;9:977
20 Xu GZ, Li W, Liu KG, et al. Early pressure dressing for the prevention of subdural effusion secondary to decompressive craniectomy in patients with severe traumatic brain injury. J Craniofac Surg 2014;25(05):1836–1839
21 Honeybul S, Ho KM. Decompressive craniectomy for severe traumatic brain injury: the relationship between surgical complications and the prediction of an unfavourable outcome. Injury 2014;45(09):1332–1339
22 Sharma R, Sharma R, Tandon V, et al. Is endoscopic third ventriculostomy a feasible option or ventriculoperitoneal shunt a safer bet for the treatment of posttraumatic hydrocephalus? A gap time model-based algorithm. Neurol India 2020;68(05):1125–1132
23 Bonow RH, Oron AP, Hanak BW, et al. Post-traumatic hydrocephalus in children: a retrospective study in 42 pediatric hospitals using the pediatric health information system. Neurosurgery 2018;83(04):732–739
24 De Bonis P, Pompucci A, Mangiola A, Rigante L, Anile C. Post-traumatic hydrocephalus after decompressive craniectomy: an underestimated risk factor. J Neurotrauma 2010;27(11):1965–1970
25 Grant FC, Norcross NC. Repair of cranial defects by cranioplasty. Ann Surg 1939;110(04):488–512

