Partial Temporal Decompression as Surgical Strategy for Function Preservation in Dominant Temporal Hemorrhagic Contusions

A M Ashour, M Helmy, M Nosseir

Citation

A M Ashour, M Helmy, M Nosseir. *Partial Temporal Decompression as Surgical Strategy for Function Preservation in Dominant Temporal Hemorrhagic Contusions* . The Internet Journal of Neurosurgery. 2022 Volume 17 Number 1.

DOI: 10.5580/IJNS.56521

Abstract

Background:

Hemorrhagic brain contusions are common sequelae of severe traumatic head injury occurring in 15–35% of severe head injuries. Patients with progressive deterioration related to the contusion causing uncontrolled increase in intracranial pressure, or signs of mass effect are managed by surgical intervention. Different surgical strategies have been adopted according to the location, type and severity of the contused brain. However, excision of the necrotic tissue carries a significant risk of loss of neurological function or deterioration. We try to identify this issue and improve surgical maneuver strategy for treating dominant side temporal contusions, targeting to preserve the neurological function and improve the final functional outcome.

Methods:

A retrospective study on post traumatic dominant temporal contusions were treated at Ain Shams University Hospitals between January 2018 and June 2021. We included patients with Glasgow Coma Scale scores less than or equal to 10, dominant side based on handiness, temporal hemorrhagic contusion greater than 20 cm3 in volume. The classic decompressive craniotomy was done, when the contusions were in the superior temporal gyrus 3-4 cm posterior than the temporal tip and posterior temporal part were left un-evacuated and the temporal pole was resected whether affected or not with average of 3 cm extension posteriorly, as well the lateral cortical part of the inferior temporal gyrus with depth of 2 cm from cortical surface exposing the middle cranial fossa. The middle temporal gyrus was partially excised when the superior temporal gyrus was severely contused posterior than 4 cm to temporal tip. While middle and inferior temporal contusion were evacuated varied from from simple superficial debridement of the contused brain to subtotal or total resection if necessary.

Results:

There were 14 patients included retrospectively, 57% were males, average 39 years (19-64 years). Pre-operative CT showed significant midline shift average 7 mm (5-15mm). The average contusion volume was 32.78 cm3. Average preoperative GCS score was 9 (7-10) while the postoperative GCS score 24 hours postoperative was 14 (5-15). Only one case of mortality. Complications were observed in 2 cases (14%), the location in relation to speech deficits postoperative; 60 % of superior and middle temporal contusions had, whole temporal lobe 100%, middle and inferior 25% and middle temporal contusion was 100%. The neurological deficits varied from aphasia to expressive dysphasia (57%), resolved completely after 1 year of follow up in 5 patients (62%), 2 cases till 1 year follow up had mild form of nominal dysphasia, while aphasia seen in one patient which improved later and his KPS back to normal.

Conclusions:

Such strategy was able to achieve the aim of controlling ICP, preserving cerebral perfusion and decreasing the mortality with the preservation of neurological function in this eloquent area. The strategy might achieve a better functional outcome, and can be recommended.

INTRODUCTION

Hemorrhagic brain contusions are common sequelae of severe traumatic head injury occurring in 15–35% of severe head injuries(1,2). Although most are small lesions do not need surgical intervention, only the patients with progressive deterioration related to the contusion causing uncontrolled increase in intracranial pressure, or signs of mass effect on CT scan are managed by surgical intervention(3–5). Both strategies of conservation and intervention are based on the control of intracranial tension. Different surgical strategies have been adopted according to the location, type and severity of the contused brain. The frequent used interventions to diminish the intracranial hypertension include; surgical excision of necrotic contused brain tissue, decompressive craniectomy (DC), or both(6–8). However, excision of the necrotic tissue carries a significant risk of loss of neurological function or deterioration(9,10). We try to identify this issue and improve surgical maneuver strategy for treating dominant side temporal contusions, targeting to preserve the neurological function and improve the final functional outcome.

METHODS

We retrospectively collected data on post traumatic temporal hemorrhagic contusions and radiological signs of raised intracranial pressure (ICP) who were treated at Ain Shams University Hospitals between January 2018 and June 2021.

We included patients with Glasgow Coma Scale (GCS) scores less than or equal to 10, dominant side based on handiness, temporal hemorrhagic contusion greater than 20 cm3 in volume, midline shift of at least 5 mm, and/or cisternal compression on CT scan. The inclusion and exclusion criteria are summarized in Table 1.

The volume and location of all contusion hemorrhages were recorded. Other intracranial pathologies were noted, including extradural hematoma (EDH), subdural hematoma (SDH), subarachnoid hemorrhage.

Inclusion criteria were; adults from 18 – 65 years, with Glasgow Coma Scale (GCS) scores less than or equal to 10. Post traumatic Temporal cerebral contusions greater than 20 cm3. Midline shift equal or more than 5 mm and/or cisternal compression in CT scan. Surgical intervention was done within the first 48 hours. No pre-existing neurologic condition with residual disability or requiring ongoing medical attention

Exclusion criteria included; Patients with diffuse contusions injury pattern, penetrating, brain stem contusions and with hemodynamic instability. Non dominant temporal lobe.

Description of surgical procedures

The choice of surgical approach was at the decided by of the consultant neurosurgeon. This surgical strategy was mainly applied to brain contusion of the dominant superior and posterior temporal as being eloquent regions of the brain.

Decompression with duraplasty

The classic decompressive craniotomy was done by making a reverse question mark skin incision, with the following extensions: frontal to the midpupillary line, posterior 3 cm posterior to the tragus; superior, 2 cm lateral to midline “away from edge of the superior sagittal sinus” and inferior exposing floor of the middle cranial fossa at the with partial temporal craniectomy; removal of a portion of the inferior part temporal bone (greater then 3 cm cranio-caudal and 5 cm antro-posterior)(6,9,11–14). Durotomy was done over the entire of the middle frontal and temporal lobe. When subdural hematoma seen were evacuated if present with widening of durotomy.

When the intra-parenchymal dominant temporal hemorrhagic contusions were in the superior temporal gyrus 3-4 cm posterior than the temporal tip and posterior temporal part were left un-evacuated and the temporal pole was resected whether affected or not with average of 3 cm extension posteriorly, as well the lateral cortical part of the inferior temporal gyrus with depth of 2 cm from cortical surface exposing the middle cranial fossa, also the middle temporal gyrus was partially excised when the superior temporal gyrus were severely contused posterior than 4 cm to temporal tip. To provide adequate room in middle fossa for contused temporal lobe decompression. While middle and inferior temporal contusion were evacuated varied from from simple superficial debridement of the contused brain to subtotal or total resection if necessary. Meticulous hemostasis was done using different hemostatic agents. Duroplasty was achieved using a pericranium graft. The portion of cranium was placed back without tight fixation. Cranioplasty was performed approximately 3 months after discharge.

Postoperative management

The postoperative care was concordance with the principles

of the AANS guidelines for management of severe head injury regardless of their surgical intervention(15,16). All severe patients were kept sedated and intubated postoperatively and ventilated. Extubation was done if patient GCS improved enough. Head CT was performed after the patient was surgery 8 hours post. Central venous and Foley catheters were inserted into all patients for fluid balance monitoring. Gastroprotective and anticonvulsants were prophylactically given. Mannitol infusion (1 g/kg every 6–12 h) and furosemide (20–40 mg every 6–12 h) were used to decrease the ICP for 5 days in average. Cerebral perfusion pressure was maintained by keeping mean blood pressure around 90Hg. Postoperative complications such as delayed hematoma, hematoma enlargement, cerebral infarction, intracranial infection, hydrocephalus, brain herniations and CSF leakage were observed.

Data collection

In a standard fashion, we retrospectively identified patients and collected data on initial GCS score, acute hospital course, preoperative deficits, post-operative deficits, length of hospital stay, mortality and long-term outcomes for 12 months. Radiographic data were obtained from the pre-operative CT scans. The contusion volume was calculated using semiautomated tools for volumetric assessment by Synapse PACS Fujifilm Global. While midline shift was defined as midline shift greater than or equal to 5 mm.

Complications and Prognosis Evaluation

Neurological outcome was assessed for each patient before discharge , at 1,3, 6 and 12 months follow-up based on the KPS score. Either by face-to-face interviews with patients or caregivers.

Statistical analysis

All data were analyzed by SPSS22 (IBM SPSS Statistics Version 22, International Business Machines Corp., Armonk, NY, USA). Results are expressed as mean ± standard deviation. All normally distributed continuous data were analyzed using unpaired t-tests and expressed as means and standard deviation. $P < 0.05$ were considered statistically significant.

RESULTS

Demographic data and preoperative head CT characteristics

There were 14 patients included retrospectively, whom underwent the described surgical procedure, 57% were males, average 39 years (19-64 years). Pre-operative CT showed significant midline shift average 7 mm (5-15mm) in most of the patients. The average contusion volume was 32.78 cm3. Associated SDH or SAH presented in 35% or 14% of patients, respectively. Average preoperative GCS score was 9 (7-10) while the postoperative GCS score 24 hours postoperative was 14 (5-15). Only one case of mortality which involved the whole temporal lobe and preoperative GCS 7 and postoperative was GCS 5. Length of stay 9 days in average (6-18), complications were observed in 2 cases (14%) chest infection and wound infection Table 1. Regarding the location in relation to speech deficits postoperative; 60 % of superior and middle temporal contusions had, whole temporal lobe 100%, middle and inferior 25% and middle temporal contusion was 100%.

Regarding functional KPS score and location shown in Table 2. KPS in relation to location Figure 1 and contusion preoperative volume Figure 2. Regarding the neurological deficits varied from aphasia to expressive dysphasia (57%) , resolved completely after 1 year of follow up in 5 patients (62%), 2 cases till 1 year follow up had mild form of nominal dysphasia, while aphasia seen in one patient which improved later and his KPS back to normal Table 3.

Figure 1

KPS compared to location.

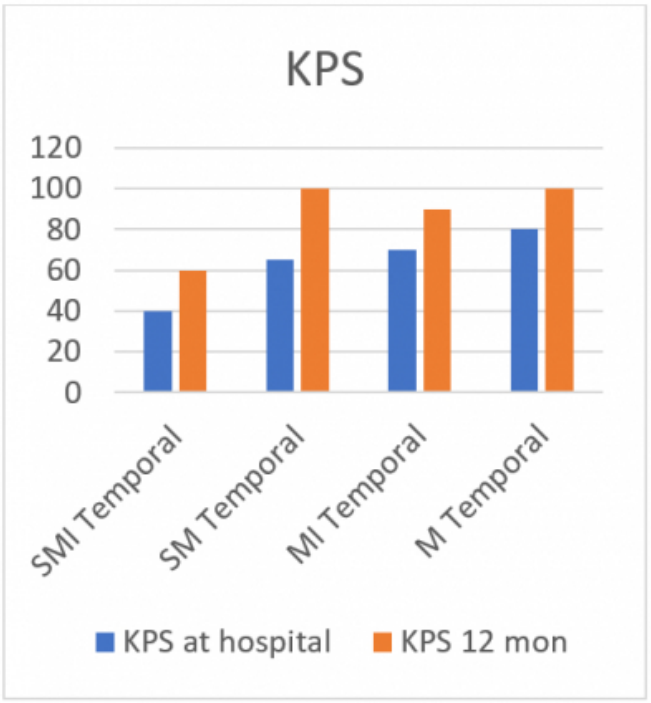

Figure 2

KPS compared to contusion volume through 12 months.

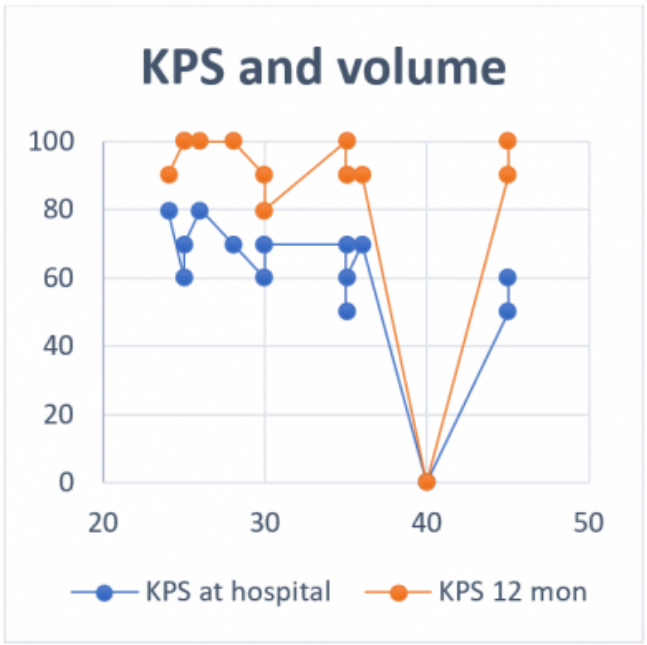

Table 1

Demographics

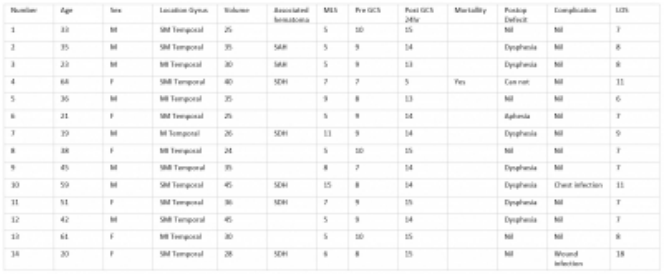

Table 2

Contusion location, deficits and outcome.

Location Gyrus	Volume	Postop Defecit	Deficit resolution 1 year	KPS at hospital	KPS 12 mon
SM Temporal	25	Nil	Nil	60	100
SM Temporal	35	Dysphesia	Resolution	70	100
MI Temporal	30	Dysphesia	Resolution	60	90
SMI Temporal	40	Can not	Nil	0	0
MI Temporal	35	Nil	Nil	50	90
SM Temporal	25	Aphesia	Moderate dysphasia	70	100
M Temporal	26	Dysphesia	Resolution	80	100
MI Temporal	24	Nil	Nil	80	90
SMI Temporal	35	Dysphesia	Resolution	60	90
SM Temporal	45	Dysphesia	Mild nominal dysphasia	50	90
SM Temporal	36	Dysphesia	Resolution	70	90
SMI Temporal	45	Dysphesia	Mild nominal dysphasia	60	100
MI Temporal	30	Nil	Nil	70	80
SM Temporal	28	Nil	Nil	70	100

Table 3

KPS during study duration

Patient	KPS at hospital	KPS 1 mon	KPS 3 mon	KPS 12 mon
1	60	90	90	100
2	70	80	100	100
3	60	80	90	90
4	0	0	0	0
5	50	80	80	90
6	70	80	80	100
7	80	90	80	100
8	80	90	80	90
9	60	90	90	90
10	50	80	90	90
11	70	90	80	90
12	60	70	80	100
13	70	70	70	80
14	70	80	100	100
Average	60	80	80	90

DISCUSSION

Severe brain hemorrhagic contusions are often associated with mass effect not related to hemorrhage mass effect which increase within 12 to 48 hours after the first insult. The mechanisms of the rapid progress of the mass effect is not fully explained by the theories of vasogenic and cytotoxic edema (17). The damage of membranes and

cytoplasmic particles lead to high osmolality within the damaged brain. The elevated osmosis forces across the periphery of the brain tissues cause accumulation of water in the contused brain tissue, which is a main cause of rapid deterioration(18). As well as the elevated intracranial pressure and the diminished perfusion may result in brain ischemia, up to mortality(19–21). Cases with hemorrhagic brain contusions represent 40.5 to 43% of the whole population of TBI, and the mortality ranges from 12.5 - 28.0%. In common practice its known that surgical intervention should be done as early as possible, when criteria are fulfilled (20,22–24). The surgical maneuvers differ from institution to another. However, DC when done early considered to help in decreasing the possibility of secondary cerebellar damage, decreasing brain swelling, and improve the functional and survival outcome (25–30). Kawamata et al. reported that massive brain welling is caused solely by hemorrhagic contusion, and surgical excision of the damaged tissue help in manipulation of the increased ICP and the final outcome(31).

Most of the studies in literature indicated that excision of contused tissues with DC was the most appropriate strategy for contusions located in non eloquent brain area. However the mortality diminished applying this strategy, the excised parts were usually manifested by different degree of deficits, from mild symptoms as memory impairments, abnormal behavior, abnormal cognition up to manifesting deficits as hemiplegia and aphasia(19,32–34). It was considered not the best strategy choice for all the conditions. On the other side managing large contusion without removal of the necrotic tissue has been accepted as skull bones decompression can significantly diminish the intracranial pressure nd improve functional and survival outcome(21,24,35–38).

In our research we tried to perform the classical DC in patients, where contusions involve or surround the temporal dominant eloquent areas, without excision of the contused tissue, to avoid as possible the postoperative manifesting neurological deficits as aphasia. A satisfactory outcome was accomplished in decreasing the mortality and avoidance the functional burden of aphasia and decreasing dysphasia. Binghui et al., reported almost the same surgical strategy for the sever localized cerebral hemorrhagic contusion in eloquent areas, their prognosis reached 68.2%, and mortality diminished to 6.8%. They also reported that in some cases the classical DC alone could not treat the intracranial hypertension then the excision of the safe tissues was done.

Our study focused on group of cases with dominant temporal contusions and achieved a satisfactory functional outcome and a lower death rate as observed in this study. The excision of the possible temporal lobe parts not only decreased the intracranial hypertension in our report, but also give a room for the non-excised contused tissues, to avoid further deterioration of function which was confirmed in many studies as well(24).

The most important finding in our study is that the morbidity and no-need for reoperation rate were lower compared to cases series in literature; mortality rate of temporal contusion was 32–56% (31,39). While, mortality rate in ours was only 13.2%, which is significantly less.

There were 8 patients with speech deficits varied from aphasia to expressive dysphasia (57%), the location in relation to speech deficits postoperative; 60 % of superior and middle temporal contusions, whole temporal lobe 100%, middle and inferior 25% and middle temporal contusion was 100%. Which resolved completely after 1 year of follow up in 5 patients (62%), 2 cases till 1 year follow up had mild form of nominal dysphasia, while aphasia seen in one patient which improved later and his KPS went back to normal.

The better functional recovery as assessed by KPS was observed, probably due to the preservation of the surrounding contusion tissue areas and creating a room for contused tissues keeping the perfusion penumbra, maximizing the possible recovery of the surviving cases Figure 3-4.

STUDY LIMITATIONS

Our study had several limitations. First, the study included cases enrolled from a single institute. Also, a large number of cases with multiple brain contusions, were excluded to diminish the potential associated factors. Finally, the retrospective manner and the small number of patients in the study, decreased our ability to reach statistically significant results.

CONCLUSIONS

The aim of surgery in brain contusion is to control increased intracranial hypertension, and to preserve the neurological functions . Such strategy was able to achieve the aim of controlling ICP, protecting cerebral perfusion and diminishing the mortality with the preservation of neurological function in this eloquent area. The strategy might achieve a better functional outcome, and can be

recommended.

List of abbreviations

DC Decompressive Craniotomy

GCS Glasgow Coma Scale

ICP Intra Cranial Pressure

KPS Karnofsky Performance Scale

SAH Sub Arachnoid Hematoma

SDH Sub Dural Hematoma

TBI Traumatic Brain Injury

References

1. Lobato RD, Cordobes F, Rivas JJ, de la Fuente M, Montero A, Barcena A, et al. Outcome from severe head injury related to the type of intracranial lesion. J Neurosurg. 1983 Nov;59(5):762–74.
2. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. J Neurol Neurosurg Psychiatry. 1981 Apr;44(4):285–93.
3. Miller JD, Butterworth JF, Gudeman SK, Faulkner JE, Choi SC, Selhorst JB, et al. Further experience in the management of severe head injury. J Neurosurg. 1981 Mar;54(3):289–99.
4. Gennarelli TA, Spielman GM, Langfitt TW, Gildenberg PL, Harrington T, Jane JA, et al. Influence of the type of intracranial lesion on outcome from severe head injury. J Neurosurg. 1982 Jan;56(1):26–32.
5. Teasdale G, Jennett B. {ASSESSMENT} {OF} {COMA} {AND} {IMPAIRED} {CONSCIOUSNESS}. The Lancet. 1974 Jul;304(7872):81–4.
6. Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. J Neurosurg. 2006 Apr;104(4):469–79.
7. Coplin WM, Cullen NK, Policherla PN, Vinas FC, Wilseck JM, Zafonte RD, et al. Safety and Feasibility of Craniectomy with Duraplasty as the Initial Surgical Intervention for Severe Traumatic Brain Injury. J Trauma Inj Infect Crit Care. 2001 Jun;50(6):1050–9.
8. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, et al. Decompressive Bifrontal Craniectomy in the Treatment of Severe Refractory Posttraumatic Cerebral Edema. Neurosurgery. 1997 Jul;41(1):84–94.
9. Chibbaro S, Tacconi L. Role of decompressive craniectomy in the management of severe head injury with refractory cerebral edema and intractable intracranial pressure. Our experience with 48 cases. Surg Neurol. 2007 Dec;68(6):632–8.
10. Yatsushige H, Takasato Y, Masaoka H, Hayakawa T, Otani N, Yoshino Y, et al. Prognosis for Severe Traumatic Brain Injury Patients Treated with Bilateral Decompressive Craniectomy. In: Brain Edema {XIV}. Springer Vienna; 2009. p. 265–70.
11. YAMAKAMI I, YAMAURA A. Effects of Decompressive Craniectomy on Regional Cerebral Blood Flow in Severe Head Trauma Patients. Neurol Med Chir (Tokyo). 1993;33(9):616–20.
12. Geyik AM. Role of Early Decompressive Craniectomy in Traumatic Brain Injury$\mathsemicolon$ Our Clinical Experience. Turk J Trauma Emerg Surg. 2021;
13. Williams RF, Magnotti LJ, Croce MA, Hargraves BB, Fischer PE, Schroeppel TJ, et al. Impact of Decompressive Craniectomy on Functional Outcome After Severe Traumatic Brain Injury. J Trauma Inj Infect Crit Care. 2009 Jun;66(6):1570–6.
14. Hilton G. Guidelines for the Management of Severe Head Injury: A Joint Initiative of the American Association of Neurological Surgeons and the Brain Trauma Foundation. J Neurosci Nurs. 1996 Oct;28(5):342.
15. Warden DL, Gordon B, McAllister TW, Silver JM, Barth JT, Bruns J, et al. Guidelines for the Pharmacologic Treatment of Neurobehavioral Sequelae of Traumatic Brain Injury. J Neurotrauma. 2006 Oct;23(10):1468–501.
16. Rusnak M, Janciak I, Majdan M, Wilbacher I, Mauritz W. Severe Traumatic Brain Injury in Austria {VI}: Effects of guideline-based management. Wien Klin Wochenschr. 2007 Feb;119(1–2):64–71.
17. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. J Neurol Neurosurg Ampmathsemicolon Psychiatry. 1981 Apr;44(4):285–93.
18. Jiang JY, Xu W, Li WP, Xu WH, Zhang J, Bao YH, et al. Efficacy of Standard Trauma Craniectomy for Refractory Intracranial Hypertension with Severe Traumatic Brain Injury: A Multicenter, Prospective, Randomized Controlled Study. J Neurotrauma. 2005 Jun;22(6):623–8.
19. Elwatidy S. Bifrontal decompressive craniectomy is a life-saving procedure for patients with nontraumatic refractory brain edema. Br J Neurosurg. 2009 Jan;23(1):56–62.
20. Lobato RD, Cordobes F, Rivas JJ, Fuente M de la, Montero A, Barcena A, et al. Outcome from severe head injury related to the type of intracranial lesion. J Neurosurg. 1983 Nov;59(5):762–74.
21. Hendam H, Taha A. Surgical Outcome of Traumatic Intracranial Hematoma. Open J Mod Neurosurg. 2020;10(01):51–62.
22. Traumatic brain injury: Patterns of failure of nonoperative management. Am J Emerg Med. 2001 Sep;19(5):442.
23. Chibbaro S, Tacconi L. Role of decompressive craniectomy in the management of severe head injury with refractory cerebral edema and intractable intracranial pressure. Our experience with 48 cases. Surg Neurol. 2007 Dec;68(6):632–8.
24. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical Management of Traumatic Parenchymal Lesions. Neurosurgery. 2006 Mar;58(suppl_3):S2-25-S2-46.
25. Yatsushige, Hiroshi, Yoshio Takasato, Hiroyuki Masaoka, Takanori Hayakawa, Naoki Otani, Yoshikazu Yoshino, Kyoko Sumiyoshi et al. "Prognosis for severe traumatic brain injury patients treated with bilateral decompressive craniectomy." In Brain Edema XIV, pp. 265-270. Springer, Vienna, 2010.
26. Coplin WM, Cullen NK, Policherla PN, Vinas FC, Wilseck JM, Zafonte RD, et al. Safety and Feasibility of Craniectomy with Duraplasty as the Initial Surgical Intervention for Severe Traumatic Brain Injury. J Trauma Inj Infect Crit Care. 2001 Jun;50(6):1050–9.
27. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: observations on the use of the Glasgow

Outcome Scale. J Neurol Neurosurg Psychiatry. 1981 Apr;44(4):285–93.
28. Hilton G. Guidelines for the Management of Severe Head Injury: A Joint Initiative of the American Association of Neurological Surgeons and the Brain Trauma Foundation. J Neurosci Nurs. 1996 Oct;28(5):342.
29. Warden DL, Gordon B, McAllister TW, Silver JM, Barth JT, Bruns J, et al. Guidelines for the Pharmacologic Treatment of Neurobehavioral Sequelae of Traumatic Brain Injury. J Neurotrauma. 2006 Oct;23(10):1468–501.
30. Williams RF, Magnotti LJ, Croce MA, Hargraves BB, Fischer PE, Schroeppel TJ, et al. Impact of Decompressive Craniectomy on Functional Outcome After Severe Traumatic Brain Injury. J Trauma Inj Infect Crit Care. 2009 Jun;66(6):1570–6.
31. Katayama Y, Kawamata T. Edema fluid accumulation within necrotic brain tissue as a cause of the mass effect of cerebral contusion in head trauma patients. In: Kuroiwa T, Baethmann A, Czernicki Z, Hoff JT, Ito U, Katayama Y, et al., editors. Brain Edema XII. Vienna: Springer Vienna; 2003. p. 323–7.
32. Mani SS, Fine EJ, Mayberry Z. Alexia without agraphia: Localization of the lesion by computerized tomography. Comput Tomogr. 1981 Jan;5(1):95–7.
33. Chieregato A, Fainardi E, Servadei F, Tanfani A, Pugliese G, Pascarella R, et al. Centrifugal Distribution of Regional Cerebral Blood Flow and its Time Course in Traumatic Intracerebral Hematomas. J Neurotrauma. 2004 Jun;21(6):655–66.
34. YAMAKAMI I, YAMAURA A. Effects of Decompressive Craniectomy on Regional Cerebral Blood Flow in Severe Head Trauma Patients. Neurol Med Chir (Tokyo). 1993;33(9):616–20.
35. Yamakami I, Yamaura A, Murai H, Isobe K. Effects of Decompressive Craniectomy on Regional Cerebral Blood Flow in Severe Head Trauma Patients Assessed by HMPAO SPECT. In: Recent Advances in Neurotraumatology [Internet]. Springer Japan; 1993. p. 204–7. Available from: https://doi.org/10.1007%2F978-4-431-68231-8_46
36. Narayan RK, Greenberg RP, Miller JD, Enas GG, Choi SC, Kishore PRS, et al. IMPROVED CONFIDENCE OF OUTCOME PREDICTION IN SEVERE HEAD INJURY. J Comput Assist Tomogr. 1981 Dec;5(6):947.
37. Butterworth JF, Selhorst JB, Greenberg RP, Miller D, Gudeman SK. Flaccidity after Head Injury. Neurosurgery. 1981 Sep;9(3):242–8.
38. WILSON JTL, PETTIGREW LEL, TEASDALE GM. Structured Interviews for the Glasgow Outcome Scale and the Extended Glasgow Outcome Scale: Guidelines for Their Use. J Neurotrauma. 1998 Aug;15(8):573–85.
39. Edna TH. Risk factors in traumatic head injury. Acta Neurochir (Wien). 1983 Aug 1;69(1):15–21.

Author Information

Ahmed M Ashour, MD. PhD.
Lecturer and consultant; Neurosurgery department, Ain Shams University
Cairo, Egypt

Mohamed Helmy, MD. PhD.
Lecturer and consultant; Neurosurgery department, Ain Shams University
Cairo, Egypt

Mohamed Nosseir, MD. PhD.
Assistant Professor and consultant; Neurosurgery department, Ain Shams University
Cairo, Egypt