ORIGINAL ARTICLE

Retrospective Comparison of Decompressive Hemicraniectomy and Hematoma Evacuation for Spontaneous Supratentorial Intracerebral Hematoma

Joarder MA[1], Karim AKMB[2], Kamal T[3], Sujon SI[4], Akhter N[5], Islam K[6], Hossain MZ[7], Mollik A[8], Sultana S[9], Shankar DRK[10], Jahangir SM[11], Chandy M[12]

Abstract

Objectives: The aim of this study was to test the hypothesis that decompressive hemicraniectomy (DHC), compared with craniotomy with evacuation of hematoma, and would improve clinical outcomes of patients with supratentorial intracerebral hemorrhage (SICH). Methods: We compared patients (November 2008–February 2014) with supratentorial ICH treated with DHC without hematoma evacuation and craniotomy with hematoma evacuation. DHC measured at least 150 mm and included opening of the dura. We analyzed clinical, radiological, and surgical characteristics. Outcome at 6 months was divided into good (modified Rankin Scale 0–4) and poor (modified Rankin Scale 5–6). Results: Fifteen patients (mean age 58 years) with ICH were treated by DHC. Median hematoma volume was 61 ml and mean preoperative Glasgow Coma Scale (GCS) was 7. Ten patients had good and five had poor outcomes. In hematoma evacuation group 29 patients were treated. Median hematoma volume was 55 ml and mean preoperative Glasgow Coma Scale (GCS) was 8. Seventeen patients had good and twelve had poor outcomes. Conclusions: DHC is more effective than hematoma evacuation in patients with SICH. Based on this small cohort, DHC may reduce mortality. Larger prospective study is warranted to assess safety and efficacy.

Introduction

Decompressive hemicraniectomy (DHC) is applied for space-occupying lesions such as major ischemic stroke, cerebral sinus venous thrombosis, aneurysmal subarachnoid hemorrhage, and traumatic brain injury.[1-4] DHC is performed to prevent raised intracranial pressure. Of the few published reports on DHC in intracerebral hemorrhage (ICH), most of the reports focus on a combined treatment of hematoma evacuation plus DHC.[5–9] Reports on DHC as the only treatment for ICH are rare, and the surgical decompression in these cases was smaller than a standardized DHC.[10] The rationale for evacuation of ICH is to prevent the toxic effects of hematoma degradation and the mechanical complication of mass effect.[11] Because DHC beneficially addresses mass effect, we evaluated whether DHC of sufficient size without clot evacuation is safe and feasible in patients with ICH.

Non traumatic SICH represents 10%-15% of all strokes and often carries a poor prognosis.[12,13,14] In general, we consider surgical intervention for patients who are relatively young; who have a large, symptomatic SICH; who have some preservation of brainstem function; and who can undergo surgery very

1. Specialist, Department of Neurosurgery, Apollo Hospitals Dhaka 2. Registrar, Department of Neurosurgery, Apollo Hospitals Dhaka 3-4. Clinical associate, Department of Neurosurgery, Apollo Hospitals Dhaka, 5. SMO, Department of Neurosurgery, Apollo Hospitals Dhaka 6-9. RMO, Department of Neurosurgery, Apollo Hospitals Dhaka, 10. Senior Consultant, Department of Neurosurgery, Apollo Hospitals Dhaka 11. Senior Consultant and coordinator, Neuro ICU and Department of Anesthesia. 12. Senior Consultant and coordinator, Department of Neurosurgery, Apollo Hospitals Dhaka.

soon (within a few hours at most) after their initial neurological deterioration. Under these circumstances, we operate on deep basal ganglia hemorrhages and on more superficial lobar hemorrhages. Unfortunately, even among this select group of patients, clinical outcomes remain poor.

Thus, the neurosurgical community continues to seek ways to improve outcomes for the subset of patients who meet the criteria for surgical evacuation. Clinical and laboratory research has demonstrated that SICH is followed by injury to the surrounding brain; this secondary injury might result from various mechanisms including excitotoxicity, direct toxicity of the hematoma, and inflammation.[15,16] These various forms of secondary injury cause cytotoxic edema, which can cause increased mass effect and tissue damage. On the basis of these studies and our own observations of the occasional need for further surgery after evacuation of SICH via small craniotomies, we hypothesized that surgical treatment for SICH might be optimized by larger DHCs, analogous to the treatment of mass effect caused by cytotoxic edema in the setting of head trauma and ischemic stroke. To test this hypothesis, we performed a retrospective chart review to compare outcomes after DHC and evacuation of SICH with craniotomy and replacement of the bone flap.

Materials and methods

We identified 44 patients with supratentorial SICH who had undergone DHC (15 patients) and hematoma evacuation (29 patients) during 2008-2014. According to review of preoperative CT images, patients were classified as having had basal ganglia or lobar hemorrhage. Review of medical records and CT images provided preoperative and intraoperative clinical data and postoperative outcome data. To determine the hematoma volume and maximal midline shift, we reviewed radiologists' reports, when the midline shift was not specified in a report, it was measured at the foramen of Monro by using digital measuring tools available on our viewing software.

Surgical Procedures

For craniotomy and evacuation case, the goal was evacuation of the hemorrhage, usually assisted by microscope. Endoscopic procedures and stereotactic guided hematoma aspiration were not performed during the study period. For all cases of basal ganglia hemorrhages were approached by using the operating microscope. The small craniotomies were generally standard pterional craniotomies intended to facilitate exposure of the sylvian fissure; Patients who underwent DHC only, without evacuation of the hematoma, the DHCs were generally trauma-type flap intended to maximize decompression of the cerebral hemisphere. The opening of the dura was performed in a stellate fashion, and the exposed brain was covered by augmentive duraplasty with Galea Aponeurotica.

Results

During the study period, 15 patients were treated with DHC without clot evacuation for spontaneous supratentorial ICH. Mean age was 58 years, and 11 patients were male and 4 patients were female. The hemorrhage lateralized to the left in 10 patients and to the right in 5 patients, The ICH was lobar in 7

Table I: Mean distribution of volume in different location between two groups

Location	Decompressive hemicraniectomy Mean±SD	Haematoma evacuation Mean±SD
Basal ganglia (BG)	58.75±10.31	56.26±10.79
Lobar	65.42±23.15	50.66±50.66

Table II: Mean variables

	Decompressive hemicraniectomy Mean±SD	Haematoma evacuation Mean±SD
Volume	61.86±17.17	55.68±10.43
Midline shift	10.80±3.16	10.79±1.82
Admission GCS	7.93±3.12	8.10±2.51
Discharge GCS	5.13±3.06	5.13±4.20
6 month FU mRS	3.26±1.90	4.10±1.89

Table III : Outcome between two groups

		Group		P value
		Decompressive hemicraniectomy	Haematoma evacuation	
mRS	0-4	10	12	0.112
	5-6	5	17	
Total		15	29	

Table IV: Summary of literature on hematoma evacuation and DHC in SICH.

Authors & year	No. of cases	Dominant side	>60ml	mortality	Term	Good outcome	Term
Hematoma evacuation							
Dierssen et al., 1983	73	53%	unknown	33%	2 yrs	45%	2yrs
Ma et al., 2010	38	unknown	unknown	32%	1 month	55%	6 months
Maira et al., 2002	15	unknown	unknown	20%	1 yr	73%	1 yr
Murthy et al., 2005	12	8%	67%	8%	discharge	50%	17 months
Present series	29	44%	32%	41%	6 months	41%	6 months
DHC							
Ramnarayan et al., 2009	23	43%	30%	13%	3 months	56%	3 months
Fung et al., 2012	12	58%	50%	25%	6 months	50%	6 months
Present series	10	66%	60%	26%	6 months	33%	6 months

patients and basal ganglia in 8 patients. On admission mean GCS score was 7 (range 5-13), mean hematoma volume was 61 ml (range 35-90 ml), mean midline shift was 10 mm (range 6-16mm). 29 patients were treated with craniotomy and clot evacuation for spontaneous supratentorial ICH. Mean age was 57 years, and 18 patients were male and 11 patients were female. The hemorrhage lateralized to the left in 13 patients and to the right in 16 patients. The ICH was lobar in 3 patients and basal ganglia in 26 patients. On admission, the mean GCS score was 8 (range 5–13), and the mean ICH volume was 55 ml (range 42–82 ml), the mean midline shift was 10 mm (range 8–16 mm).

Outcome

Outcome was assessed by the modified Rankin Scale, when patients returned to our outpatient clinic after 6 months. Good outcome was defined as modified Rankin Scale of 0 to 4 and bad outcome as modified Rankin Scale of 5-6. Ten patients (66%) in the DHC group had good and 5 (34%) had poor outcome. In hematoma evacuation group, 12 patients (41%) had good and 17 patients (59%) had poor outcome. Mortality in DHC group was 26% and in evacuation group was 41%.

Discussion

ICH is devastating with mortality rates up to 44% at 30 days.[17,18] Despite the International Surgical Trial in Intracerebral Haemorrhage

(STICH), surgical treatment in ICH remains a matter of debate and attempts to improve outcome using surgical therapy are ongoing. Trauma of open craniotomy and especially trauma to the brain parenchyma for hematoma evacuation were considered to outweigh the benefits of surgery.[19] Therefore, many efforts were made to minimize the invasiveness of operative procedures related to clot evacuation.[20-22] Driven by recent promising results of DHC in ischemic stroke, DHC could also be promising for treatment of space-occupying ICH. Advantages of DHC include no trauma to brain, ease and speed of the procedure and no issues of brain parenchymal hemostasis.

Complications

Complications in DHC group were observed in 5 of 15 patients. Among 5 patients, 2 patients had subdural hygroma, 2 of them had increased hematoma and perihemorrhagic edema and 1 developed hydrocephalus. Similar complications have been reported after DHC and are probably not specifically related to DHC in ICH.[23,24] Complications in hematoma evacuation group were observed in 8 patients. 4 of them rebled with increased size of hematoma, 2 developed hydrocephalus and 2 developed brain swelling following hematoma evacuation. In addition, 15 medical complications occurred in both group (6 in DHC and 9 in evacuation). However, such medical complications are common in a neurointensive care unit. Our results show that DHC after ICH is feasible and may also be safe. There were no deaths related directly to surgery and all complications were manageable without long-term sequelae. Besides hematoma volume, perihemorrhagic edema may cause secondary deterioration of ICH patients. Kollmar et al showed that mild hypothermia prevented the increase of edema[25] and Zazulia et al[26] reported an increase in edema formation measured by an increasing midline shift. Although the direct effect of DHC on perihemorrhagic edema remains unknown, our data show that DHC significantly reduced midline shift, thereby possibly counteracting the space-occupying effects of the hematoma and edema formation.

The limitations of our study are its retrospective design, the small sample size, and the heterogeneity of the patient with respect to the origin of ICH. Nevertheless, our preliminary results are encouraging and justify the initiation of a prospective study.

References

1. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, et al. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol. 2007;6:215–22.

2. Weiner GM, Lacey MR, Mackenzie L, Shah DP, Frangos SG, Grady MS et al. Decompressive craniectomy for elevated intracranial pressure and its effect on the cumulative ischemic burden and therapeutic intensity levels after severe traumatic brain injury. Neurosurgery. 2010;66:1111–8; discussion 1118.

3. Nagel A, Graetz D, Vajkoczy P, Sarrafzadeh AS. Decompressive craniectomy in aneurysmal subarachnoid hemorrhage: relation to cerebral perfusion pressure and metabolism. Neurocrit Care. 2009;11:384–94.

4. Ferro JM, Crassard I, Coutinho JM, Canhão P, Barinagarrementeria F, Cucchiara B et al. Second international study on cerebral vein and dural sinus

thrombosis (ISCVT 2) investigators. Decompressive surgery in cerebrovenous thrombosis: a multicenter registry and a systematic review of individual patient data. Stroke. 2011;42:2825–31.

5. Ma L, Liu WG, Sheng HS, Fan J, Hu WW, Chen JS. Decompressive craniectomy in addition to hematoma evacuation improves mortality of patients with spontaneous basal ganglia hemorrhage. J Stroke Cerebrovasc Dis. 2010;19:294–98.

6. Shimamura N, Munakata A, Naraoka M, Nakano T, Ohkuma H. Decompressive hemi-craniectomy is not necessary to rescue supratentorial hypertensive intracerebral hemorrhage patients: consecutive single-center experience. Acta Neurochir Suppl. 2011;111:415-19.

7. Dierssen G, Carda R, Coca JM. The influence of large decompressive craniectomy on the outcome of surgical treatment in spontaneous intracerebral haematomas. Acta Neurochir (Wien). 1983;69:53-60.

8. Murthy JM, Chowdary GV, Murthy TV, Bhasha PS, Naryanan TJ. Decompressive craniectomy with clot evacuation in large hemispheric hypertensive intracerebral hemorrhage. Neurocrit Care. 2005;2:258-62.

9. Kim KT, Park JK, Kang SG, Cho KS, Yoo DS, Jang DK et al. Comparison of the effect of decompressive craniectomy on different neurosurgical diseases. Acta Neurochir(Wien). 2009;151:21–30.

10. Ramnarayan R, Anto D, Anilkumar TV, Nayar R. Decompressive hemicraniectomy in large putaminal hematomas: an Indian experience. J Stroke Cerebrovasc Dis. 2009;18:1–10.

11. Broderick JP. The STICH trial: what does it tell us and where do we go from here? Stroke. 2005;36:1619–20.

12. Caplan LR.Intracerebral haemorrhage. Lancet 1992;339:656–58.

13. Rincon F, Mayer SA. Intracerebral hemorrhage: getting ready for effective treatments. CurrOpin Neurol.2010;23:59–64.

14. Woo D, Broderick JP. Spontaneous intracerebral hemorrhage: epidemiology and clinical presentation. Neurosurg Clin N Am. 2002;13:265–79.

15. Aronowski J, Zhao X. Molecular pathophysiology of cerebral hemorrhage: secondary brain injury. Stroke. 2011;42:1781–86.

16. Wang J. Preclinical and clinical research on inflammation after intracerebral hemorrhage. Prog Neurobiol. 2010;92:463-77.

17. Broderick JP, Brott T, Tomsick T, Miller R, Huster G. Intracerebral hemorrhage more than twice as common as subarachnoid hemorrhage. J Neurosurg. 1993;78:188-91.

18. Anderson CS, Chakera TM, Stewart-Wynne EG, Jamrozik KD. Spectrum of primary intracerebral haemorrhage in Perth, Western Australia, 1989-90: incidence and outcome. J Neurol Neurosurg Psychiatr. 1994;57:936–40.

19. Mendelow AD, Gregson BA, Fernandes HM, Murray GD, Teasdale GM, Hope DT et al. Early surgery versus initial conservative treatment in patients with spontaneous supratentorialintracerebralhaematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): a randomised trial. Lancet. 2005;365:387-397.

20. Teernstra OP, Evers SM, Lodder J,Leffers P, Franke CL, Blaauw G. Multicenter randomized controlled trial (SICHPA). Stereotactic treatment of intracerebral hematoma by means of a plasminogen activator: a multicenter randomized controlled trial (SICHPA). Stroke. 2003;34:968-74.

21. Nishihara T, Nagata K, Tanaka S, Suzuki Y, Izumi M, Mochizuki Y et al. Newly developed endoscopic instruments for the removal of intracerebral hematoma. Neurocrit Care. 2005;2:67-74.

22. Newell DW, Shah MM, Wilcox R, Hansmann DR, Melnychuk E, Muschelli J et al. Minimally invasive evacuation of spontaneous intracerebral hemorrhage using sonothrombolysis. J Neurosurg. 2011;115:592-601.

23. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A et al. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol. 2007;6:215-222.

24. Güresir E, Vatter H, Schuss P, Oszvald A, Raabe A, Seifert V et al. Rapid closure technique in decompressive craniectomy. J Neurosurg. 2011;114:954-60.

25. Kollmar R, Staykov D, Dörfler A, Schellinger PD, Schwab S, Bardutzky J. Hypothermia reduces perihemorrhagic edema after intracerebral hemorrhage. Stroke. 2010;41:1684-89.

26. Zazulia AR, Diringer MN, Derdeyn CP, Powers WJ. Progression of mass effect after intracerebral hemorrhage. Stroke. 1999;30:1167–73.