Original Article

Clinical Profile and Predictors of Functional Outcome After Decompressive Craniectomy Across Major Stroke Subtypes: A Retrospective Cohort Study

Keerthiraj DB[1], Pooja Tripathi[1], Dinkar Kulshrestha[1], Ajai Kumar Singh[1], Pradeep Kumar Maurya[1], Adbul Qavi[1]

[1]Department of Neurology, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh, India

Abstract

Background: Decompressive craniectomy is used in severe stroke with malignant cerebral oedema or mass effect, but long-term functional outcome varies considerably. **Material and Methods:** This retrospective cohort study included 287 patients undergoing decompressive craniectomy for ischemic stroke, intracerebral haemorrhage, or cerebral venous sinus thrombosis between 2019 and 2024. Clinical, radiological, perioperative, and follow-up data were analysed. Poor 1-year outcome was defined as modified Rankin Scale (mRS) 5–6. Multivariable logistic regression identified independent baseline predictors. **Results:** Ischemic stroke accounted for 62.4% of cases, ICH for 30.0%, and CVST for 7.7%. In-hospital mortality was 15.3%. At 1 year, 149/264 (56.4%) had favourable outcome and 115 (43.6%) had poor outcome. Older age (aOR 1.92 per 10 years), higher admission NIHSS (aOR 1.63 per 5 points), and lower GCS (aOR 1.29 per point decrease) independently predicted poor outcome. **Conclusion:** Long-term outcome after decompressive craniectomy was more strongly related to age and neurological severity than to stroke subtype or surgical timing.

Keywords: Decompressive craniectomy; Stroke; Functional outcome; Modified Rankin Scale; Prognostic factors.

Received: 21 July 2026 **Revised: 02 August 2026** **Accepted: 18 August 2026** **Published: 11 September 2026**

Introduction

Decompressive craniectomy is a potentially lifesaving intervention in patients with severe stroke complicated by space-occupying cerebral oedema, raised intracranial pressure, and impending herniation. Decompression can help to decrease secondary tissue injury and prevent brainstem compression, which can be fatal, by allowing the enlarged brain to expand beyond the confines of the skull. It has been most clearly demonstrated to be beneficial in malignant middle cerebral artery (MCA) infarction, where early randomized trials have demonstrated a significant mortality benefit and improved chances of survival with acceptable functional outcome.[1-4]

The benefit of decompressive surgery, however, needs to be considered alongside the degree of disability among survivors. When counselling patients and families, long-term outcome is therefore of particular importance, and the pooled analysis of the DECIMAL, DESTINY and HAMLET trials showed that surgery within 48 hours of stroke onset in younger patients with malignant MCA infarction was associated with a clear survival benefit, but a significant proportion of patients remained functionally dependent after surgery.[1] This survival advantage was confirmed in patients older than 60 years in the DESTINY II study, but a significant number of survivors were functionally dependent.[5]

The evidence for decompressive craniectomy in other types of stroke is less clear. The prognosis for patients with decompression for stroke is variable and depends on the underlying vascular pathology and the severity of cerebral injury; decompression is life-saving in selected patients with large haematomas, cerebral oedema and mass effect, and in severe cerebral venous sinus thrombosis with haemorrhagic venous infarction, malignant oedema or impending herniation, where meaningful neurological recovery may follow.[6-7]

Therefore, it is important to identify factors associated with functional recovery for surgical decision making and prognostication. Real world outcomes in different stroke subtypes may thus be helpful in patient selection, family counselling and rehabilitation planning, and may be useful in the evaluation of outcome after hemicraniectomy, as previous studies have suggested that age, neurological severity, level of consciousness, radiological mass effect and midline shift may influence outcome after decompressive surgery.[8] Indian experience has also shown that functional improvement can continue over months after hemicraniectomy, but long term data are relatively limited and most published studies have focused predominantly on malignant MCA infarction.[9]

Address for correspondence: Dr. Keerthiraj D B, Assistant Professor, Department of Neurology, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.
E-mail: dbkeerthiraj@gmail.com

DOI:
10.21276/amit.2026.v13.i3.1028

How to cite this article: Keerthiraj DB, Tripathi P, Kulshrestha D, Singh AK, Maurya PK, Qavi A. Clinical Profile and Predictors of Functional Outcome After Decompressive Craniectomy Across Major Stroke Subtypes: A Retrospective Cohort Study. Acta Med Int. 2026;13(3):114-121.

The aim of the present study was to describe the demographic, clinical, radiological, peri-operative and outcome profile of patients undergoing decompressive craniectomy for ischemic stroke, intracerebral haemorrhage and cerebral venous sinus thrombosis in a tertiary care centre. It also sought to determine clinical and radiological factors associated with functional outcome at around 1 year after surgery.

Materials and Methods

Study design and setting: This retrospective observational cohort study was conducted at Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow, a tertiary care referral centre in northern India. Hospital records of all patients who underwent decompressive craniectomy or hemicraniectomy for stroke between 1 January 2019 and 31 December 2024 were reviewed. Follow-up outcomes were obtained from available hospital records and, where permitted, through telephone, video, or in-person assessment.

Study population: Patients were included if decompressive surgery was performed for acute ischemic stroke with malignant cerebral oedema, spontaneous intracerebral haemorrhage (ICH), or cerebral venous sinus thrombosis (CVST) with venous infarction, haemorrhage, oedema, or mass effect. Patients with primary subarachnoid haemorrhage, bilateral strokes not attributable to a unilateral decompressive procedure, isolated external ventricular drainage or ventriculoperitoneal shunting, decompression for non-stroke conditions, or inadequate records to confirm the diagnosis or surgical procedure were excluded. Complete enumeration of all eligible patients during the study period was performed.

Data collection: Data were extracted using a structured study proforma. Baseline variables included age, sex, body mass index, vascular risk factors, premorbid modified Rankin Scale (mRS), stroke subtype, admission National Institutes of Health Stroke Scale (NIHSS), Glasgow Coma Scale (GCS), pupillary abnormalities, clinical evidence of herniation, and neurological deterioration before surgery.

Laboratory investigations and baseline neuroimaging findings were recorded where available. Radiological variables included lesion location, infarct or haematoma size, midline shift, ventricular compression, basal cistern effacement, radiological herniation, intraventricular haemorrhage, haemorrhagic transformation, and other relevant findings. Surgical and perioperative information included time from stroke onset and hospital presentation to surgery, type of decompressive procedure, haematoma evacuation or infarct resection, mechanical ventilation, vasopressor requirement, intensive care unit stay, and total hospital stay. Postoperative complications, including sepsis, pneumonia, seizures, acute kidney injury, thromboembolic events, wound complications, and repeat neurosurgical intervention, were recorded separately from baseline predictors.

Outcome assessment: Functional outcome was assessed using the mRS, with the original 0–6 score retained at discharge, approximately 3 months, approximately 1 year, and the latest available follow-up beyond 1 year. The primary outcome was functional status at approximately 1 year. An mRS score of 0–4 was classified as favourable outcome and mRS 5–6 as poor outcome; death was coded as mRS 6. Patients without an assessable 1-year outcome were not assigned to either outcome category.

NIHSS scores were recorded at admission, discharge, and follow-up where available. In-hospital mortality and major postoperative complications were analysed as secondary outcomes.

Statistical analysis: Continuous variables were summarized as mean ± standard deviation or median with interquartile range, and categorical variables as n (%). Comparisons across ischemic stroke, ICH, and CVST used one-way analysis of variance or the Kruskal–Wallis test for continuous variables and the chi-square or exact test for categorical variables, as appropriate. Paired changes in NIHSS were assessed using the Wilcoxon signed-rank test.

For the primary outcome analysis, patients with favourable and poor 1-year outcomes were compared using Welch's t test or the Mann–Whitney U test for continuous variables and the chi-square or Fisher exact test for categorical variables. Multivariable binary logistic regression was then used to identify independent baseline predictors of poor 1-year outcome. The presurgical model included age, sex, stroke subtype, admission NIHSS, admission GCS, premorbid mRS, midline shift, radiological herniation, and time from stroke onset to surgery. Postoperative complications were not entered into the primary baseline predictor model. Adjusted associations were reported as adjusted odds ratios (aORs) with 95% confidence intervals. Regression analysis was performed using complete cases. All tests were two-sided, and $p<0.05$ was considered statistically significant.

Ethical considerations: The study was undertaken after approval from the Institutional Ethics Committee. All extracted data were de-identified before analysis, and confidentiality was maintained throughout the study.

Results

Study population and baseline profile: A total of 352 records were screened, of which 65 were excluded after application of the study eligibility criteria. The final cohort comprised 287 patients who underwent decompressive craniectomy for stroke. Ischemic stroke accounted for 179 (62.4%) patients, intracerebral haemorrhage for 86 (30.0%), and cerebral venous sinus thrombosis for 22 (7.7%). The mean age was 54.8 ± 12.8 years, and 197 (68.6%) patients were male. Baseline clinical characteristics differed across stroke subtypes, particularly with respect to age, sex distribution, admission neurological severity, vascular risk profile, and time to surgery [Table 1].

Radiological, surgical, and in-hospital course: The median midline shift was 9.7 mm (IQR 7.3–11.8), with radiological herniation present in 102 (35.5%) patients. Mechanical ventilation was required in 219 (76.3%) patients and inotropic or vasopressor support in 82 (28.6%). Pneumonia occurred in 62 (21.6%), sepsis in 30 (10.5%), seizures in 43 (15.0%), and acute kidney injury in 24 (8.4%). In-hospital mortality was 44

(15.3%). Detailed radiological, surgical, and complication profiles according to stroke subtype are shown in [Table 2].

Functional and neurological outcomes: Discharge mRS was available for all 287 patients, while approximately 3-month, 1-year, and latest >1-year mRS assessments were available for 242 (84.3%), 264 (92.0%), and 200 (69.7%) patients, respectively. At approximately 1 year, 149 (56.4%) patients had a favourable outcome (mRS 0–4) and 115 (43.6%) had a poor outcome (mRS 5–6); the latter included 49 (18.6%) deaths. The complete ordinal mRS distributions are summarized in Table 3 and Figure 1. Among patients with paired NIHSS observations, neurological deficit improved significantly from admission to each subsequent assessment, including the 1-year comparison (n=184, Wilcoxon W=23.0, p<0.001).

Factors associated with poor 1-year functional outcome: In univariable analyses, poor 1-year outcome was associated with older age, higher admission NIHSS, lower admission GCS, pupillary abnormality, clinical evidence of herniation, greater midline shift, radiological herniation, and hypertension [Table 4]. Postoperative complications were examined separately from baseline predictors and were not entered into the primary presurgical predictor model.

In the multivariable model (n=232), older age remained independently associated with poor outcome (aOR 1.92, 95% CI 1.43–2.58, p<0.001), as did greater admission neurological severity measured by NIHSS (aOR 1.63, 95% CI 1.19–2.22, p=0.002) and lower admission GCS (aOR 1.29, 95% CI 1.09–1.52, p=0.002). The overall model was significant (likelihood-ratio χ^2=65.35, df=10, p<0.001; McFadden R^2=0.205). Adjusted estimates for all prespecified candidate predictors are presented in [Table 5 and Figure 2].

Table 1: Baseline demographic and clinical characteristics according to stroke subtype

Characteristic	Overall (N=287)	Ischemic stroke (n=179)	ICH (n=86)	CVST (n=22)	Test statistic
Age, years	54.8 ± 12.8	56.8 ± 11.5	55.8 ± 11.9	34.3 ± 8.2	F=38.43
Male sex	197 (68.6%)	136 (76.0%)	54 (62.8%)	7 (31.8%)	χ^2=19.70, df=2
BMI, kg/m²	25.5 ± 3.9	25.5 ± 4.0	25.4 ± 3.4	25.6 ± 4.4	F=0.02
Hypertension	163 (56.8%)	105 (58.7%)	52 (60.5%)	6 (27.3%)	χ^2=8.54, df=2
Diabetes mellitus	94 (32.8%)	60 (33.5%)	32 (37.2%)	2 (9.1%)	χ^2=6.42, df=2
Smoking/tobacco use	122 (43.7%)	83 (47.4%)	34 (41.0%)	5 (23.8%)	χ^2=4.62, df=2
Dyslipidaemia	147 (51.2%)	94 (52.5%)	44 (51.2%)	9 (40.9%)	χ^2=1.06, df=2
Previous stroke	70 (24.4%)	55 (30.7%)	13 (15.1%)	2 (9.1%)	χ^2=10.70, df=2
Ischemic heart disease	63 (22.0%)	38 (21.2%)	24 (27.9%)	1 (4.5%)	Fisher-Freeman-Halton
Atrial fibrillation	73 (25.4%)	60 (33.5%)	12 (14.0%)	1 (4.5%)	χ^2=17.21, df=2
Premorbid mRS	0.0 (0.0–1.0)	0.0 (0.0–1.0)	0.0 (0.0–1.0)	0.0 (0.0–1.0)	H=1.37
Admission NIHSS	22.0 (18.0–25.0)	23.0 (19.0–26.0)	21.0 (16.0–24.0)	15.0 (12.0–20.0)	H=24.43
Admission GCS	10.0 (9.0–12.0)	11.0 (9.0–12.0)	9.0 (8.0–11.0)	11.0 (10.0–12.0)	H=21.97
Pupillary abnormality	46 (16.0%)	29 (16.2%)	16 (18.6%)	1 (4.5%)	Fisher-Freeman-Halton
Clinical evidence of herniation	41 (14.3%)	18 (10.1%)	22 (25.6%)	1 (4.5%)	Fisher-Freeman-Halton
Neurological deterioration before surgery	174 (60.6%)	109 (60.9%)	54 (62.8%)	11 (50.0%)	χ^2=1.21, df=2
Onset-to-presentation, h	4.1 (2.5–7.3)	4.3 (2.8–7.3)	3.2 (1.9–4.5)	12.8 (4.9–17.0)	H=36.21
Onset-to-surgery, h	17.3 (11.2–23.7)	19.2 (14.1–24.6)	10.9 (7.2–14.8)	30.3 (20.2–40.5)	H=79.73

Data are n (%) unless otherwise stated. Continuous variables are mean ± SD or median (IQR). Between-subtype comparisons used one-way ANOVA, Kruskal–Wallis, Pearson chi-square, or Fisher–Freeman–Halton exact tests as appropriate. Percentages for variables with missing data are based on available observations.

Table 2: Neuroimaging, surgical, perioperative, complication, and in-hospital outcome profile according to stroke subtype

Characteristic	Overall (N=287)	Ischemic stroke (n=179)	ICH (n=86)	CVST (n=22)	Test statistic	p value
Infarct volume, mL	218.8 (160.7–262.1)	228.6 (177.6–266.6)	—	129.3 (97.3–141.2)	U=2546.0	<0.001
Haematoma volume, mL	62.6 (44.8–76.5)	—	62.6 (44.8–76.5)	—	—	—
Midline shift, mm	9.7 (7.3–11.8)	9.6 (7.1–11.6)	10.2 (8.1–12.1)	9.4 (6.9–11.1)	H=2.18	0.337
Ventricular compression	172 (59.9%)	107 (59.8%)	50 (58.1%)	15 (68.2%)	χ^2=0.74, df=2	0.691
Basal cistern effacement	109 (38.0%)	68 (38.0%)	33 (38.4%)	8 (36.4%)	χ^2=0.03, df=2	0.985
Radiological herniation	102 (35.5%)	63 (35.2%)	31 (36.0%)	8 (36.4%)	χ^2=0.03, df=2	0.987
Intraventricular haemorrhage	34 (11.8%)	5 (2.8%)	27 (31.4%)	2 (9.1%)	Fisher-Freeman-Halton	<0.001
Haemorrhagic transformation	54 (18.8%)	41 (22.9%)	0 (0.0%)	13 (59.1%)	Fisher-Freeman-Halton	<0.001
Hydrocephalus	32 (11.1%)	12 (6.7%)	18 (20.9%)	2 (9.1%)	Fisher-Freeman-Halton	0.004

Frontotemporoparietal hemicraniectomy	274 (95.5%)	179 (100.0%)	73 (84.9%)	22 (100.0%)	Fisher-Freeman-Halton	<0.001
Haematoma evacuation	72 (25.1%)	0 (0.0%)	67 (77.9%)	5 (22.7%)	χ^2=187.68, df=2	<0.001
Resection of infarcted tissue	20 (7.0%)	16 (8.9%)	0 (0.0%)	4 (18.2%)	Fisher-Freeman-Halton	<0.001
Mechanical ventilation	219 (76.3%)	132 (73.7%)	70 (81.4%)	17 (77.3%)	χ^2=1.89, df=2	0.388
Ventilation duration, days	5.6 (3.8–8.0)	5.6 (4.0–7.8)	5.9 (3.9–9.0)	4.7 (3.5–5.9)	H=3.05	0.218
Inotropic/vasopressor support	82 (28.6%)	55 (30.7%)	25 (29.1%)	2 (9.1%)	χ^2=4.51, df=2	0.105
ICU stay, days	12.0 (9.0–16.0)	12.0 (9.0–15.0)	13.0 (10.0–17.0)	10.0 (9.0–14.0)	H=2.88	0.237
Hospital stay, days	20.0 (16.0–25.0)	20.0 (16.0–25.0)	21.0 (16.0–26.8)	20.0 (16.0–23.8)	H=0.99	0.609
Sepsis	30 (10.5%)	20 (11.2%)	8 (9.3%)	2 (9.1%)	Fisher-Freeman-Halton	0.952
Pneumonia	62 (21.6%)	39 (21.8%)	21 (24.4%)	2 (9.1%)	Fisher-Freeman-Halton	0.312
Seizures	43 (15.0%)	24 (13.4%)	19 (22.1%)	0 (0.0%)	Fisher-Freeman-Halton	0.015
Acute kidney injury	24 (8.4%)	18 (10.1%)	6 (7.0%)	0 (0.0%)	Fisher-Freeman-Halton	0.281
Repeat neurosurgical intervention	14 (4.9%)	4 (2.2%)	7 (8.1%)	3 (13.6%)	Fisher-Freeman-Halton	0.010
In-hospital mortality	44 (15.3%)	28 (15.6%)	16 (18.6%)	0 (0.0%)	Fisher-Freeman-Halton	0.064

Data are n (%) unless otherwise stated; continuous variables are median (IQR). Infarct volume was compared between ischemic stroke and CVST among patients with available measurements; haematoma volume was applicable to intracerebral haemorrhage only. Fisher–Freeman–Halton exact testing was used for sparse categorical tables.

Table 3: Functional outcome and serial neurological status

Outcome	Overall	Ischemic stroke	ICH	CVST	Test statistic	p value
Discharge mRS available	287 (100.0%)	179 (100.0%)	86 (100.0%)	22 (100.0%)	—	—
Discharge mRS	5.0 (4.0–5.0)	5.0 (4.0–5.0)	5.0 (4.0–5.0)	4.0 (4.0–5.0)	H=4.82	0.090
Discharge mRS 5–6	144 (50.2%)	91 (50.8%)	46 (53.5%)	7 (31.8%)	χ^2=3.37, df=2	0.185
3-month mRS available	242 (84.3%)	150 (83.8%)	73 (84.9%)	19 (86.4%)	Fisher-Freeman-Halton	0.966
3-month mRS	4.0 (3.0–5.0)	4.0 (3.0–5.0)	4.0 (3.0–5.0)	4.0 (3.0–4.0)	H=4.52	0.104
3-month mRS 5–6	97 (40.1%)	64 (42.7%)	30 (41.1%)	3 (15.8%)	χ^2=5.12, df=2	0.077
1-year mRS available	264 (92.0%)	167 (93.3%)	79 (91.9%)	18 (81.8%)	Fisher-Freeman-Halton	0.174
1-year mRS	4.0 (3.0–5.0)	4.0 (3.0–5.0)	4.0 (3.0–5.5)	2.5 (2.0–4.0)	H=7.84	0.020
Poor outcome at 1 year (mRS 5–6)	115 (43.6%)	74 (44.3%)	38 (48.1%)	3 (16.7%)	χ^2=6.00, df=2	0.050
Latest >1-year mRS available	200 (69.7%)	127 (70.9%)	62 (72.1%)	11 (50.0%)	χ^2=4.41, df=2	0.110
Latest >1-year mRS	4.0 (3.0–6.0)	4.0 (3.0–5.0)	4.0 (2.2–6.0)	3.0 (2.0–4.0)	H=3.27	0.195
Latest >1-year mRS 5–6	81 (40.5%)	53 (41.7%)	27 (43.5%)	1 (9.1%)	Fisher-Freeman-Halton	0.088

mRS values are median (IQR) or n (%), based on available assessments at each time point. mRS 5–6 denotes severe disability or death.

Table 3A: Paired change in NIHSS from admission to follow-up

Paired comparison	Paired n	Admission NIHSS, median (IQR)	Follow-up NIHSS, median (IQR)	Median change	Wilcoxon statistic	p value
Admission to discharge	221	21.0 (17.0–25.0)	17.0 (12.0–21.0)	-4.0	W=35.0	<0.001
Admission to 3 months	160	21.0 (17.0–25.0)	13.0 (6.8–16.2)	-9.0	W=0.0	<0.001
Admission to 1 year	184	21.0 (17.0–25.0)	11.0 (4.0–17.0)	-10.0	W=23.0	<0.001
Admission to latest >1 year	119	22.0 (17.0–26.0)	10.0 (5.5–16.0)	-12.0	W=0.0	<0.001

Negative change indicates improvement in NIHSS. Paired comparisons used the Wilcoxon signed-rank test.

Table 4: Factors associated with 1-year functional outcome

Characteristic	Overall evaluable (n=264)	Favourable mRS 0–4 (n=149)	Poor mRS 5–6 (n=115)	Test statistic	p value
Baseline/presurgical factors					
Age, years	55.1 ± 12.8	51.4 ± 12.6	59.8 ± 11.4	t=-5.63, df=255	<0.001
Male sex	181 (68.6%)	107 (71.8%)	74 (64.3%)	χ^2=1.68, df=1	0.195
BMI, kg/m²	25.6 ± 3.9	25.5 ± 4.1	25.7 ± 3.7	t=-0.47, df=244	0.637
Hypertension	149 (56.4%)	75 (50.3%)	74 (64.3%)	χ^2=5.18, df=1	0.023
Diabetes mellitus	88 (33.3%)	44 (29.5%)	44 (38.3%)	χ^2=2.23, df=1	0.136
Smoking/tobacco use	111 (43.4%)	58 (40.0%)	53 (47.7%)	χ^2=1.54, df=1	0.215
Previous stroke	67 (25.4%)	36 (24.2%)	31 (27.0%)	χ^2=0.27, df=1	0.605
Atrial fibrillation	66 (25.0%)	31 (20.8%)	35 (30.4%)	χ^2=3.21, df=1	0.073
Stroke subtype: Ischemic stroke	167 (63.3%)	93 (62.4%)	74 (64.3%)	χ^2=6.00, df=2	0.050
Intracerebral haemorrhage	79 (29.9%)	41 (27.5%)	38 (33.0%)		
CVST	18 (6.8%)	15 (10.1%)	3 (2.6%)		
Premorbid mRS	0.0 (0.0–1.0)	0.0 (0.0–1.0)	0.0 (0.0–1.0)	U=7561.0	0.608
Admission NIHSS	22.0 (17.5–25.0)	20.0 (15.0–24.2)	23.0 (20.0–26.0)	U=5743.0	<0.001
Admission GCS	10.0 (9.0–12.0)	11.0 (10.0–12.0)	9.0 (8.0–11.0)	U=11388.0	<0.001
Pupillary abnormality	43 (16.3%)	17 (11.4%)	26 (22.6%)	χ^2=5.97, df=1	0.015
Clinical evidence of herniation	39 (14.8%)	13 (8.7%)	26 (22.6%)	χ^2=9.94, df=1	0.002
Neurological deterioration before surgery	159 (60.2%)	82 (55.0%)	77 (67.0%)	χ^2=3.85, df=1	0.050
Midline shift, mm	9.6 (7.3–11.8)	8.9 (7.0–10.9)	10.7 (8.3–12.4)	U=6293.0	<0.001
Ventricular compression	158 (59.8%)	84 (56.4%)	74 (64.3%)	χ^2=1.72, df=1	0.190
Basal cistern effacement	103 (39.0%)	51 (34.2%)	52 (45.2%)	χ^2=3.29, df=1	0.070
Radiological herniation	97 (36.7%)	47 (31.5%)	50 (43.5%)	χ^2=3.98, df=1	0.046
Onset-to-surgery, h	17.2 (11.2–23.5)	17.3 (11.5–24.5)	17.2 (11.1–22.8)	U=9164.5	0.332
Postoperative/perioperative factors (exploratory)					
Mechanical ventilation	201 (76.1%)	112 (75.2%)	89 (77.4%)	χ^2=0.18, df=1	0.674
Ventilation duration, days	5.5 (3.9–8.0)	5.0 (3.7–7.7)	5.8 (4.3–8.5)	U=4234.5	0.067
Inotropic/vasopressor support	76 (28.8%)	37 (24.8%)	39 (33.9%)	χ^2=2.61, df=1	0.106
Sepsis	26 (9.8%)	12 (8.1%)	14 (12.2%)	χ^2=1.24, df=1	0.265
Pneumonia	58 (22.0%)	28 (18.8%)	30 (26.1%)	χ^2=2.01, df=1	0.156
Ventilator-associated complication	19 (9.5%)	10 (8.9%)	9 (10.1%)	χ^2=0.08, df=1	0.776
Seizures	42 (15.9%)	28 (18.8%)	14 (12.2%)	χ^2=2.12, df=1	0.145
Acute kidney injury	23 (8.7%)	10 (6.7%)	13 (11.3%)	χ^2=1.72, df=1	0.189
Repeat neurosurgical intervention	12 (4.5%)	9 (6.0%)	3 (2.6%)	χ^2=1.76, df=1	0.184
ICU stay, days	12.0 (9.0–16.0)	12.0 (9.0–15.0)	13.0 (9.0–17.0)	U=7893.0	0.272
Hospital stay, days	20.0 (16.0–25.0)	20.0 (16.0–25.0)	21.0 (17.0–25.0)	U=7982.0	0.341

Data are n (%) unless otherwise stated. Continuous variables are mean ± SD or median (IQR). Baseline/presurgical factors and postoperative/perioperative factors are presented separately. Postoperative variables were not included in the primary baseline predictor model.

Table 5: Multivariable logistic regression of baseline predictors of poor 1-year functional outcome

Predictor	Scale/reference	Adjusted OR	95% CI	Wald z	p value
Age	per 10-year increase	1.92	1.43–2.58	4.36	<0.001
Female sex	vs male	1.35	0.70–2.62	0.90	0.370
Intracerebral haemorrhage	vs ischemic stroke	1.11	0.52–2.38	0.28	0.782
CVST	vs ischemic stroke	2.54	0.47–13.85	1.08	0.281
Admission NIHSS	per 5-point increase	1.63	1.19–2.22	3.06	0.002
Lower admission GCS	per 1-point decrease	1.29	1.09–1.52	3.03	0.002
Premorbid mRS	per 1-point increase	0.85	0.57–1.29	-0.75	0.451
Midline shift	per 5-mm increase	1.47	0.87–2.48	1.44	0.150
Radiological herniation	yes vs no	1.18	0.60–2.32	0.49	0.624
Onset-to-surgery time	per 12-hour increase	0.90	0.58–1.39	-0.47	0.635

Dependent variable: poor 1-year outcome (mRS 5–6). Complete-case model N=232 with 102 poor-outcome events. Likelihood-ratio χ^2=65.35, df=10, p<0.001; McFadden R^2=0.205; AIC=274.9. Reference stroke subtype: ischemic stroke.

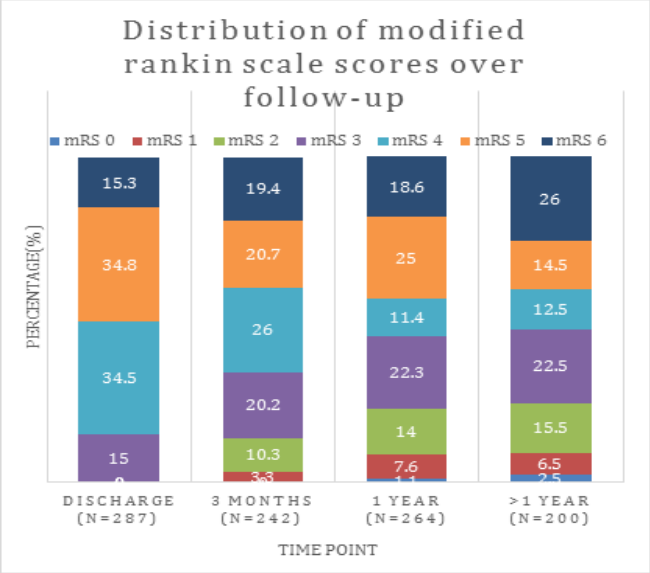

Figure 1: Distribution of modified Rankin Scale scores at discharge and follow-up.

Stacked bar chart showing the percentage distribution of mRS scores (0-6) at four time points: discharge (n=287), 3 months (n=242), 1 year (n=264), and >1 year (n=200). mRS 0 represents no symptoms, mRS 1-2 represent functional independence, mRS 3-4 represent moderate to moderately severe disability, mRS 5 represents severe disability, and mRS 6 represents death. The distribution shows a shift from severe disability (mRS 4-5) at discharge toward improved functional outcomes (mRS 0-3) and increased mortality over time.

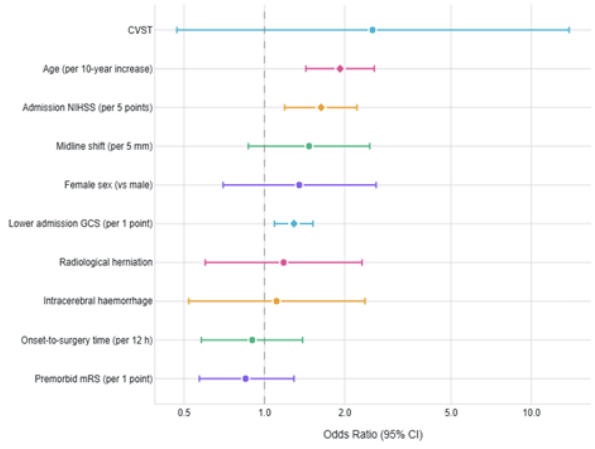

Figure 2: Forest Plot of Predictors of Poor Outcome

Points indicate adjusted odds ratios and horizontal lines indicate 95% confidence intervals. The vertical dashed line represents an odds ratio of 1.0.

Odds ratios with 95% confidence intervals for predictors of poor outcome. The vertical dashed line represents an odds ratio of 1.0 (no effect). Diamond markers indicate statistically significant predictors (confidence interval does not cross 1.0); circle markers indicate non-significant predictors. Three predictors were statistically significant: Age (per 10-year increase) (OR 1.92, 95% CI 1.43–2.58); Admission NIHSS (per 5 points) (OR 1.63, 95% CI 1.19–2.22); Lower admission GCS (per 1 point) (OR 1.29, 95% CI 1.09–1.52). CVST showed the highest point estimate (OR 2.54) but with a wide confidence interval (0.47–13.85) that crossed 1.0. Premorbid mRS and onset-to-surgery time showed odds ratios below 1.0, suggesting potential protective effects, but neither reached statistical significance.

Discussion

In the present study, real-world data on functional outcome following decompressive craniectomy in three different stroke subtypes is provided. Ischemic stroke was the most common stroke type in almost 2/3 of the cohort, followed by intracerebral haemorrhage (ICH) and cerebral venous sinus thrombosis (CVST). There was a high level of baseline neurological severity, but in-hospital mortality was 15.3% and 56.4% of those with an assessable 1-year outcome had an mRS of 0–4. Neurological deficit also showed a gradual improvement at follow-up. Most importantly, age, admission NIHSS and admission GCS scores were independently associated with poor 1-year functional outcome, whereas stroke subtype, midline shift, radiological herniation and time to surgery were not.

Our results are similar to those of malignant hemispheric infarction in non-randomized trials.[10] The mortality rate and proportion of patients with mRS ≤4 in our larger stroke cohort are comparable, with the exception of the longer primary follow-up and the presence of haemorrhagic and venous strokes in our cohort, which limits direct comparison.[10]

Age was the strongest independent predictor in our model, with the odds of poor outcome increasing almost two-fold for every 10-year increment. This is consistent with the overall literature on decompressive surgery.[11] These results suggest that age is only one of the factors that influences prognosis, and that an infarct-volume threshold of around 270 cm³ was particularly informative: 62 of 67 patients with volumes >270 cm³ had an unfavourable outcome (mRS 4–6), whereas only 15 of 29 patients with volumes ≤270 cm³ had an unfavourable outcome.[11]

In our cohort, baseline neurological severity was also significant. Each 5-point increase in NIHSS was associated with a 63% increase in adjusted odds of poor outcome, while each 1-point decrease in admission GCS was associated with a 29% increase in odds of poor outcome. This is in keeping with the clinical concept that neurological deficit and altered consciousness is due to both primary tissue damage and progressive mass effect. This is similar to our finding that onset-to-surgery time was not independently associated with 1-year outcome after adjustment for baseline severity, and is consistent with the results of the meta-analysis performed by Goedemans et al., which also showed no clear excess risk with surgery beyond 48 hours.[12]

It is important to note that there was no independent association between surgical timing and outcome in our study. The time from stroke onset to surgery is closely related to disease evolution, referral patterns, recognition of malignant oedema

and when neurological deterioration is evident. In another multicentre analysis, Kamran et al. found no significant difference in functional outcome between patients decompressed within and beyond 48 hours, and mortality was also comparable between the two groups.[13] In that study, age ≥55 years, additional vascular-territory infarction, septum pellucidum deviation ≥1 cm, and uncal herniation were stronger predictors of poor outcome than timing itself.[13] Taken together with our findings, these observations suggest that the biological severity of brain injury at the time of intervention may be more informative than elapsed time considered in isolation.

We included patients with spontaneous ICH, which increases the clinical relevance of the cohort, as there is less evidence for decompressive surgery in haemorrhagic stroke than in malignant MCA infarction. In our cohort, ICH accounted for 30% of all procedures and 48.1% of patients with ICH had poor outcome at 1 year, which was not significantly different from the outcomes observed in the other stroke subtypes. These observations further highlight the importance of separating out the concepts of survival benefit from functional independence when counselling patients undergoing decompression for haemorrhagic stroke.[14]

The clinical profile of the CVST subgroup was different. The patients were significantly younger than those with arterial ischemic stroke or ICH and the number with poor 1-year outcome was the lowest. In the previous studies, severe CVST had good recovery potential even after imminent herniation, although the number of patients in this subgroup was small; of the 10 patients with severe CVST in this study, 8 survived, 5 of whom had no disability at 12 months (mRS 0–1), and 2 had mRS scores of 2 and 3, respectively, at 12 months; interestingly, 9 of the 10 had space-occupying haemorrhagic infarction, and the median preoperative midline shift was 9 mm, indicating that favourable recovery was possible despite the striking radiological severity.[15]

The results should be interpreted in the context of the relatively good functional outcome observed in our patients with CVST, but the limited number of patients with CVST and the broad confidence interval for the adjusted effect of stroke subtype does not allow for definitive conclusions about the prognosis of CVST compared with other stroke types.[15-17]

Univariable analysis showed strong association with poor outcome for radiological markers (midline shift and herniation), but these markers were not significant after adjustment. This does not mean that mass effect is not clinically significant. Instead, midline shift, herniation, GCS and NIHSS are biologically related aspects of the same progressive malignant process and may be less influential when included together in a multivariable model. Hecht et al. have shown that total infarct volume is an independent predictor of outcome.[11] and Kamran et al. have reported that septal deviation and uncal herniation are associated with outcome, in addition to neurological severity.[13] More detailed volumetric and topographical imaging variables are therefore suggested to be used in future models, instead of only midline shift.

Another clinical finding of interest was the change in functional status over time. At discharge, about half of our patients had mRS 5–6, while this rate decreased in evaluable survivors during follow-up and NIHSS scores were significantly better at 1 year and beyond compared to admission. This course highlights the fact that neurological outcome at discharge may underestimate the subsequent recovery after severe stroke requiring decompression. Meanwhile, there was a build-up of mortality over the follow-up period, reflecting the competing nature of late functional improvement in survivors and ongoing mortality in the overall population. Therefore, long-term assessment is more desirable than discharge status alone to evaluate the results of decompressive surgery.

The study has a number of strengths, such as the consecutive recruitment over 5 years, the assessment of functional outcome up to 1 year and beyond, the inclusion of ischemic, haemorrhagic and venous stroke, and the separate evaluation of presurgical predictors and postoperative complications. Use of both mRS and serial NIHSS also allowed assessment of disability as well as neurological recovery.

The main drawbacks are that it was a retrospective, single-centre study, that some follow-up time points were incomplete, and that the primary regression analysis used complete cases. There may also have been referral and selection bias in the clinical selection of patients for surgery, while the small number of patients in the CVST subgroup may limit subtype-specific conclusions. In addition, imaging variables were not consistently comparable across stroke types, and the definition of favourable outcome (mRS 0–4) differed from that used in some previous studies.

Overall,long-term outcome in this heterogeneous group seemed to be related more to age and baseline neurological severity than to stroke subtype or surgical timing. These factors can therefore be of special value for prognosis and counseling of patients and families.

Conclusion

Decompressive craniectomy was associated with meaningful long-term functional recovery in selected patients with severe stroke. Older age, higher admission NIHSS, and lower GCS independently predicted poor 1-year outcome, highlighting the importance of baseline neurological severity in prognostication.

References

1. Vahedi K, Hofmeijer J, Jüttler 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(3):215-222. doi:10.1016/S1474-4422(07)70036-4.
2. Vahedi K, Vicaut E, Mateo J, Kurtz A, Orabi M, Guichard JP, et al. Sequential-design, multicenter, randomized, controlled trial of early decompressive craniectomy in malignant middle cerebral artery infarction (DECIMAL Trial). Stroke. 2007;38(9):2506-2517. doi:10.1161/STROKEAHA.107.485235.
3. Jüttler E, Schwab S, Schmiedek P, Unterberg A, Hennerici M, Woitzik J, et al. Decompressive surgery for the treatment of malignant infarction of the middle cerebral artery (DESTINY): a randomized, controlled trial. Stroke. 2007;38(9):2518-2525. doi:10.1161/STROKEAHA.107.485649.

4. Hofmeijer J, Kappelle LJ, Algra A, Amelink GJ, van Gijn J, van der Worp HB. 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(4):326-333. doi:10.1016/S1474-4422(09)70047-X.
5. Jüttler E, Unterberg A, Woitzik J, Bösel J, Amiri H, Sakowitz OW, et al. Hemicraniectomy in older patients with extensive middle-cerebral-artery stroke. N Engl J Med. 2014;370(12):1091-1100. doi:10.1056/NEJMoa1311367.
6. Lo YT, See AAQ, King NKK. Decompressive craniectomy in spontaneous intracerebral hemorrhage: a case-control study. World Neurosurg. 2017;103:815-820.e2. doi:10.1016/j.wneu.2017.04.025.
7. Coutinho JM, Majoie CBLM, Coert BA, Stam J. Decompressive hemicraniectomy in cerebral sinus thrombosis: consecutive case series and review of the literature. Stroke. 2009;40(6):2233-2235. doi:10.1161/STROKEAHA.108.543421.
8. Bruno A, Paletta N, Verma U, Grabowska ME, Haughey HM, Batchala PP, et al. Predicting functional outcome after decompressive craniectomy for malignant hemispheric infarction: clinical and novel imaging factors. World Neurosurg. 2022;158:e1017-e1021. doi:10.1016/j.wneu.2021.12.027.
9. Rai VK, Bhatia R, Prasad K, Srivastava MVP, Singh S, Rai N, et al. Long-term outcome of decompressive hemicraniectomy in patients with malignant middle cerebral artery infarction: a prospective observational study. Neurol India. 2014;62(1):26-31. doi:10.4103/0028-3886.128273.
10. Kamran S, Akhtar N, Salam A, Alboudi A, Rashid H, Kamran K, et al. Decompressive hemicraniectomy for malignant middle cerebral artery stroke: South Asian experience. J Stroke Cerebrovasc Dis. 2017;26(10):2306-2312. doi:10.1016/j.jstrokecerebrovasdis.2017.05.018.
11. Hecht N, Neugebauer H, Fiss I, Pinczolits A, Vajkoczy P, Jüttler E, et al. Infarct volume predicts outcome after decompressive hemicraniectomy for malignant hemispheric stroke. J Cereb Blood Flow Metab. 2018;38(6):1096-1103. doi:10.1177/0271678X17718693.
12. Goedemans T, Verbaan D, Coert BA, Kerklaan BJ, van den Berg R, Coutinho JM, et al. Outcome after decompressive craniectomy for middle cerebral artery infarction: timing of the intervention. Neurosurgery. 2020;86(3). doi:10.1093/neuros/nyz522.
13. Kamran S, Akhtar N, Salam A, Alboudi A, Kamran K, Ahmed A, et al. Revisiting hemicraniectomy: late decompressive hemicraniectomy for malignant middle cerebral artery stroke and the role of infarct growth rate. Stroke Res Treat. 2017;2017:2507834. doi:10.1155/2017/2507834.
14. Beck J, Fung C, Strbian D, Bütikofer L, Z'Graggen WJ, Lang MF, et al. Decompressive craniectomy plus best medical treatment versus best medical treatment alone for spontaneous severe deep supratentorial intracerebral haemorrhage: a randomised controlled clinical trial. Lancet. 2024;403(10442):2395-2404. doi:10.1016/S0140-6736(24)00702-5.
15. Zuurbier SM, Coutinho JM, Majoie CBLM, Coert BA, van den Munckhof P, Stam J. Decompressive hemicraniectomy in severe cerebral venous thrombosis: a prospective case series. J Neurol. 2012;259(6):1099-1105. doi:10.1007/s00415-011-6307-3.
16. Ferro JM, Crassard I, Coutinho JM, Canhão P, Barinagarrementeria F, Cucchiara B, et al. Decompressive surgery in cerebrovenous thrombosis: a multicenter registry and a systematic review of individual patient data. Stroke. 2011;42(10):2825-2831. doi:10.1161/STROKEAHA.111.615393.
17. Misra S, Sudhir P, Nath M, Sharma VK, Vibha D. Decompressive surgery in cerebral venous thrombosis: a systematic review and meta-analysis. Eur J Clin Invest. 2023;53(4). doi:10.1111/eci.13944.