

Research Article

The impact of emergent suboccipital craniectomy upon outcome & prognosis of massive cerebellar infarction: A single institutional study

Mohamed Abdelbari Mattar^{a,*}, Hala Maher^b, Wael K. Zakaria^a

^a Department of Neurosurgery, College of Medicine, Mansoura University, Egypt

^b Diagnostic Radiology Dept., College of Medicine, Aswan University, Egypt

ARTICLE INFO

Keywords:

Suboccipital craniectomy
Cerebellar infarction
CSF diversion
Prognosis

ABSTRACT

Objective: The dilemma of management of post-ischemic cerebellar massive swelling is still debatable. This study focuses on the usefulness and prognosticators influencing its surgical outcome in a locality with limited resources.

Methods: a sum of 42 patients (36 men, 6 women; mean \pm SD age = 66 ± 13 years, range 43–80 years) who were admitted, and subjected to emergent suboccipital craniectomy (ESC) after radiological evidence of malignant cerebellar swelling were reviewed. Prerequisites were (1) Glasgow Coma Scale (GCS) ≤ 13 (2) evident brainstem pressure and/or associated obstructive hydrocephalus. CSF diversion was done concurrently or later, depending on what the circumstances dictate.

Results: upon approaching the postoperative third month, 36 patients (85.7%) had lived, 25 (59.5%) of whom were independent (modified Rankin scale ≤ 2). 17 cases (40.4%) were either completely dependent or passed away. Statistical analysis disclosed that the factors influencing the prognosis were: Admission GCS ($p < 0.05$), Time (from onset to surgery) ($p < 0.05$), Obstructive hydrocephalus ($p < 0.05$), and another acute intracranial Infarction ($p < 0.01$).

Conclusions: emergent suboccipital craniectomy (ESC) is indicated for treating post-ischemic cerebellar massive swelling in patients with GCS ≤ 13 . Good prognosis could be attained with (ESC) in those cases with higher GCS, who performed it shortly from the onset, and in absence of elsewhere intracranial infarction. In those who already have obstructive hydrocephalus at the time of surgery, a poor prognosis ensues.

1. Introduction

Decompressive craniectomy has been developed as a peerless neurosurgical tool and treatment option in combat for life-threatening edema in response to brain infarction. Contrary to the debatable argument regarding operative therapy of expansive supratentorial infarction, operative management is largely believed as the primary treatment in massive cerebellar infarction and highly indicated in contemporary guidelines of stroke therapy [1,6]. This malignant cerebellar infarction is a life-threatening incident, considered to be an emergency as any post fossa hemorrhage or space-occupying lesion and has been estimated to record a 20% incidence of occurrence and it is featured by the presence of ongoing depression of conscious level as a result of associated significant edema, compression/or distortion of the brainstem, brain herniation whether downward or upward and obstructive hydrocephalus. A graver presentation is that of bilateral cerebellar affection caused by

posterior inferior cerebellar artery (PICA) acute occlusion which is typically associated with infarction of the brainstem [4,21].

It has been recognized that whenever decompressive suboccipital craniectomy (DSC) is not achieved, an 84% mortality rate follows for those cases with acute cerebellar infarction associated with extensive swelling compromising the normal CSF outflow, and/or brainstem distortion and resulting in deterioration of consciousness [7]. Emergency decompressive craniotomy can be used also in other neurosurgical conditions like epidural hematomas due to venous bleeding with similar mortality [28,29]. Although there is numerous evidence that DSC is effective [7,18,26], yet, it is still unestablished when and on whom one should operate putting into consideration favorable and unfavorable prognostic criteria and providing the patient the utmost chance for recovery for a better outcome. So that we conveyed this study to find an answer to the above important inquiries.

* Corresponding author.

E-mail addresses: noramattar@yahoo.com, mabdelbarimattar@yahoo.com (M. Abdelbari Mattar).

<https://doi.org/10.1016/j.inat.2021.101223>

Received 19 February 2021; Received in revised form 10 April 2021; Accepted 11 April 2021

Available online 16 April 2021

2214-7519/© 2021 The Authors.

Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license

(<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

2. Methods

2.1. Study populations

In retrospection, we studied 42 adult patients who underwent emergent suboccipital craniectomy (ESC) for massive cerebellar infarction. They were admitted to the neurosurgery department at *Mansoura University Hospital* from January 2009 to March 2018. The standards for patient inclusion were (1)-adult age >18 years, (2)-Glasgow Coma Scale score (GCS) ≤ 13 , (3)-obstructive hydrocephalus and/or brainstem compression. (4)-anesthetically fit and only operable cases. The criteria for exclusion were: extremely ill, unfit patients for surgery, previous ischemic stroke, and those who are suffering from malignancy or advanced hepatic or renal diseases. Informed consent from the patient's family was obtained before proceeding toward surgery. Clinical and radiological follow-up was done shortly postoperatively and then periodically employing brain CT scan or MRI every single couple of months.

2.2. Demographic data & prognostic variables

Data and facts were retrieved involving age of patients at surgery, gender, symptoms at presentation (such as an increase of intracranial pressure, neurological deficits, or others), time from onset until the operation, elsewhere intracranial infarction (cerebral or brainstem), obstructive hydrocephalus, the underlying pathology of the cerebellar infarction, and the infarction size. Preliminary imaging was via brain (CT) scan, yet (MRI) brain namely, the diffusion-weighted image study (DWI), was added whenever needed in some cases for further confirmation and/or full brain study so long as the condition of the patient permits. For all cases, the volume of infarcted cerebellar tissue was manually outlined on a CT scan by two qualified radiologists who were blinded to patient data besides other findings. The exact size was demarcated section-by-section employing ITK-SNAP 2.2.0 software [10].

The clinical outcome was assessed via a 3-month post-stroke modified Rankin Scale (*mRS*), where *mRS* scores ≤ 2 refer to good outcomes, while *mRS* scores 5 to 6 refer to counterproductive outcomes [5]

Table 1
Clinical characteristics.

Criterion	Number of cases	(%)
GCS		
12–13	16	38.1%
10–11	12	28.5%
8–9	9	21.4%
6–7	5	11.9%
CT findings		
- Hydrocephalus	31	73.8%
- Unilateral cerebellar infarction	33	78.5%
- Bilateral cerebellar infarction	9	21.4%
- Concurrent Cerebral/brainstem acute ischemia	12	28.5%
Etiology		
-Cardiogenic	27	64.2%
-Atherothrombotic	12	28.5%
-Unknown (artery to artery)	3	7.1%
Responsible vessel		
PICA	36	85.7%
SCA	15	35.7%
AICA	3	7.14%
Multiple	21	50%
Cause of death		
-Severe pulmonary embolism	3	7.14%
-Chest infection/aspiration pneumonia	3	7.14%
mRS (3-months post-stroke)		
≤ 2	25	59.5%
≥ 3 –5	11	26.2%
6	6	14.2%

(Table 1).

2.3. Surgical procedures

The prone position was the preferred one for all operated cases. A midline linear skin incision was made one cm above theinion to the 4th cervical spinal process level. For every case, emergent suboccipital craniectomy (ESC) was made from the limits of the transverse sinus bilaterally until the foramen magnum. The first cervical (C1) vertebra laminectomy step was added in cases found to be non-satisfactory on (ESC) alone. The dura mater was incised in an inverted Y-shaped fashion, and decompression of the cerebellum (e.g., by eliminating infarcted matter) was achieved subsequently. Dural substitutes (Autologous such as fascia lata graft or synthetic dura) were used whenever needed to fill the dural gaps and to provide more room for adequate decompression. CSF diversion via temporary external ventricular drain (EVD) was done in whom preoperative hydrocephalus was detected or expected to arise after ESC, depending on the intraoperative observations. Thereafter, all cases were managed through ICU teamwork.

2.4. Statistical methodology

The assembled information was encoded, administered, and examined using the SPSS (*Statistical Package for Social Sciences*) version 22 for Windows® (IBM, SPSS Inc, Chicago, IL, USA). Statistically significant differences between these factors regarding the prognosis were examined. Normally distributed quantitative data (VAS score) were expressed as mean (range) and comparison between preoperative and post-operative values was done using paired samples *t*-test. The χ^2 test was utilized for comparison of clinical characteristics among the patients with improvement and those without improvement on the diagnosis scale. A *P* value that indicates statistical significance was (<0.05).

The inter-observer erraticism of the infarcted cerebellar tissue manual calculations was judged by creating scatterplots and the measurement of the Pearson correlation coefficient and its 95% confidence interval. To evaluate the prejudice and limits of agreement, Bland-Altman analysis was performed, where the bias was demarcated as the mean paired difference and the limits of agreement were defined as the bias ± 1.96 times the SD of the difference. For the relative difference (paired difference divided by the average), the mean and SD were calculated.

Table 2
Summary of number of cases with different criteria in relation to mRS.

	Modified Rankin score (mRS)		
	0–2	3–5	6
CT FINDINGS			
-Hydrocephalus	14	16	1
-Unilateral cerebellar infarction	28	3	2
-Bilateral cerebellar infarction	2	6	1
-Concurrent cerebral/brainstem acute ischemia	0	10	2
INFARCTION VOLUME			
30–50ml	11	2	1
50–70 ml	10	5	3
70–90 ml	2	3	1
90–110 ml	2	1	1
GCS			
All cases	25 (59.5%)	11(26%)	6 (14.2%)
Score = 12–13	12	4	–
Score = 10–11	9	3	–
Score = 8–9	4	2	3
Score = 6–7	–	2	3

3. Results

Tables 1–3 show the facts of the 42 patients included in the study (36 men, 6 women; mean \pm SD age = 66 ± 13 years, range 43–80 years). Cerebellar ischemic infarction was caused by intracranial embolism from multiple input sources as follows: a) cardiogenic in 27 patients, (b) athero-thrombotic in 12, and (c) artery-to-artery by unknown cause in 3. The embolized vessels incriminated in these cases were as follows: (a) PICA (posterior inferior cerebellar artery) in 36 patients, (b) SCA (superior cerebellar artery) in 15, (c) AICA (anterior inferior cerebellar artery) in 3, and (d) multi-vascular (i.e., PICA, SCA, and AICA) in 21. In all cases, brainstem distortion and/or hydrocephalic changes were detected on computed tomography (CT) or MRI before ESC. The mean time (hours: minutes) estimated from onset to surgery \pm SD time was $17:30 \pm 8:20$ (range 9:00–48:15). The mean volume of the infarcted tissue just before DSC \pm SD was 60.2 ± 17.4 cc (range 30.6–105.3 cc). There were no major complications related to the surgical operation apart from superficial wound infection in ten cases, which has been completely cured by appropriate antibiotic therapy. After 3 months, 25 (59.5%) were independent (modified Rankin scale ≤ 2), seventeen cases (40.4%) were either completely dependent or passed away, where 11 patients (26.2%) required full support (modified Rankin scale ≥ 3 –5). The other 6 patients (14.2%) had died (modified Rankin scale = 6). The cause of death was: Severe pulmonary embolism in 3 bed-ridden patients, septicemia due to an intractable chest infection, and aspiration pneumonia in 3 cases (Tables 1 and 2).

Judgment between favorable ($mRS \leq 2$) and unfavorable ($mRS \geq 3$) prognoses relative to different variables, demonstrated no substantial difference in age, and sex of the study population, infarction volume, or the source of embolism of the cerebellar infarction. On the other hand, factors influencing the prognosis were: admission GCS ($p < 0.05$), time (from onset to surgery) ($p < 0.05$), obstructive hydrocephalus ($p < 0.05$), and co-existence of another acute intracranial infarction ($p < 0.01$), where significant differences were appreciated (Tables 2 and 3).

4. Discussion

It has been reported that cerebellar infarction accompanied with edema represents about 17%–54% [9,13]. Many causes have been incriminated for the subsequent disturbed level of consciousness, such as cytotoxic edema resulting from a vasogenic one due to thrombosed vessel recanalization, hydrocephalus due to CSF outflow obstruction, brainstem distortion or compression, and either upwards or downwards cerebellar herniation (via tentorial notch in the former or foramen magnum in the later) [13,27]. Surgical decompression and its role in such cases is still debatable regarding whether or when to operate. However, several scientific recommendations have been proposed to

employ decompressive craniectomy as an essential part of management protocols as stated by the American Heart Association/American Stroke Association (AHA/ASA) and Stroke Treatment Guidelines in Japan 2015 [18,25]. Additionally in a recent study by Oliver *et al.* they advocated that a better outcome for cerebellar infarction could be obtained via suboccipital decompressive craniotomy in comparison to hemispheric infarctions that were similarly treated by decompressive surgery [19]. Though lacking sufficient evidence of support, many other studies [7,18,26] reported the significance of decompressive craniectomy for the management of infarction of cerebellum, where Tsitsopoulos *et al.* [26] found a 53.1% good prognosis in his decompressive surgery for 32 patients and, Ogasawara *et al.* [18] reported 70% (7 patients out of 10) favorable recovery after decompressive surgery. Additionally, Feely [7] reported the mortality rate to be 28% and 84% for cases operated with decompressive craniectomy and for non-operated cases, respectively. Remarkably, the mortality in the current study was only reported for 6 cases (14.28%) and for reasons that are irrelevant to neurological status.

The impact of age in patients suffering from massive cerebellar infarction was variable among different literature studies. Oliver *et al.*, reported less mortality with mean age < 60 years by sensitivity analysis in their study [19]. Nevertheless, Tsitsopoulos *et al.*, found an unchanged prognosis even for those who are beyond seventy years old who performed the decompressive surgery [12,26]. Also, Pfefferkorn *et al.* reported no influence on outcome for those who are above 60 years of age [21]. Similarly, in our study neither age nor gender presented any statistical value or influence upon the surgical outcome of patients subjected to ESC.

Although cerebellar infarction may result from vascular occlusion of one or more of its blood supply arteries, Pfefferkorn *et al.* reported the exclusive affection of the PICA territory in all studied cases, suggesting that isolated AICA and SCA infarction may escape cerebellar swelling, thereby supporting the risk of life-threatening edema for those harboring PICA infarctions [21]. In the current series, considering the incriminated vessels leading to infarction; the PICA came first, while the AICA was the least [36(85.7%), and 3(7.14%) patients respectively]. However, none of the above was statistically significant relative to the patients' outcome. It is noted that a cardiogenic source of embolism was evident in 27 patients (64.2%).

The initial level of consciousness plays an essential role in predicting outcome, and it has been reported that in nearly $\frac{1}{2}$ of comatose patients and those free of brainstem infarction an improvement in prognosis follows decompressive craniectomy [12,26]. Further, it has been considered by the GASCIS (German-Austrian Space-Occupying Cerebellar Infarction Study) as the individual prognosticator for poor outcome, where the estimated mRS score for outcome was proportionally corresponding to the level of consciousness. Although GASCIS is a prospective multicenter design, it was not randomized and consequently was subject to selection bias. Also, their study design prevented the comparison of therapy choices, as the groups are unbalanced regarding the infarction severity [11]. The current series found the preoperative GCS has impacted the outcome of studied patients, where a favorable mRS (i.e. ≤ 2) were closely related to patients with better GCS less than 13, while lower GCS values corresponded to unfavorable mRS (p value < 0.05) (Figs. 1 and 2).

It has been shown that not only surgical intervention but also its early employment should be encouraged whenever possible, as suggested by Ogasawara *et al.*, who recommended performing decompressive surgery whenever deterioration of consciousness happens, even for those who became somnolent only, and they claimed that those patients would develop coma unless EDC is established immediately [18]. Tsitsopoulos *et al.* also reported a favorable outcome for cases presented at the surgery time with a high CT score & who did surgery earlier within 24 hrs from the onset. However, they couldn't confidently relate rapid surgery to an improved outcome, owing to the limited number of cases in their cohort study [26]. Similarly, current study observations revealed that patients who operated earlier got a better

Table 3

Comparison between favorable ($mRS \leq 2$) and unfavorable ($mRS \geq 3$) prognoses relative to different variables.

Variables	$mRS \leq 2$	$mRS \geq 3$	p
Number of patients (n)	25	17	
Age of patients (years) \pm SD	64.7 ± 8.1	67.3 ± 5.1	IS**
Male gender (n)/(%)	19 (76%)	17(100%)	IS**
Admission GCS (n)/(%)	25 (60%)	17 (40%)	$< 0.05^*$
Time (from onset to surgery) (H:min) \pm SD	$12:30 \pm 5:00$	$23:15 \pm 4:30$	$< 0.05^*$
Another acute intracranial Infarction (n)	0	12	$< 0.01^*$
Infarction volume (ml^3) \pm SD	58.0 ± 8.0	75.0 ± 11.0	IS**
Hydrocephalus (n)	14	17	$< 0.05^*$
Source of embolism (n):			
-Cardiogenic	14	13	IS**
-Others	11	4	

mRS, modified Rankin Scale; IS, Insignificant.

* χ^2 test.

** Student's *t*-test.

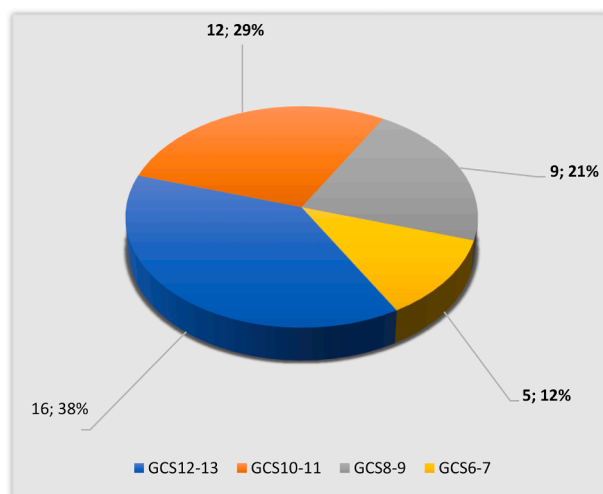


Fig. 1. Admission GCS distribution among patients.

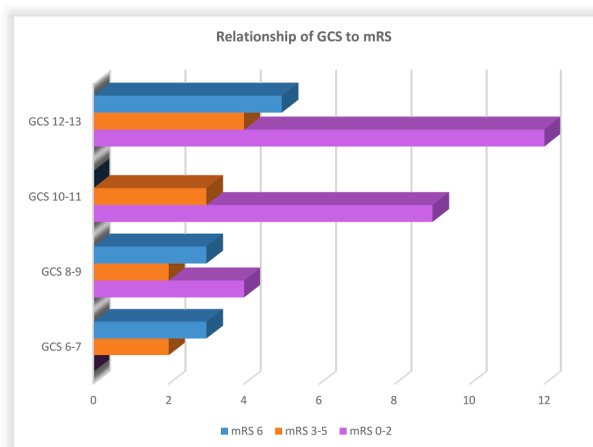


Fig. 2. Relationship of GCS to mRS.

mRS, indicating the importance of early surgical interference for improving patients' prognosis. (p value < 0.05). On the other hand, *Pfefferkorn* and co-workers concluded that the timing of surgery (from symptom onset to surgery) did not look to impact the outcome in 57

mono-centric patients in their retrospective case study, where 82% performed EVD and 56% infarcted tissue debridement in addition to decompressive craniectomy (DC). So, this may argue against prophylactic operation before clinical worsening, however, they postulated that in the absence of prospective controlled trials, it looks wise to put into account DC promptly once early signs of secondary clinical worsening happen [21].

Regarding the volume of infarction, it doesn't represent a significant prognostic factor for our patients' outcome, and this may be explained by the fact that the presence of mass effect in all our studied cases (which is one of the inclusion criteria), which is likely created by the resultant post-ischemic edema (cytotoxic/or vasogenic). (Fig. 3) However, via a retrospective-matched case-control study *Kim et al.*, found that favorable outcomes correlate well to radiologically evident infarction volume ratio between 0.25 and 0.33, only if it is limited to 72 h since an established diagnosis [15]. However their study was based on CT scan manual calculation of the infarction volume ratio, where they related the infarcted cerebellar volume not only to the remaining healthy cerebellum but also included the normal brain stem volume as representative to total cerebellar volume, this feature in addition to the absence of conscious worsening were considered as limitations as stated by authors.

The co-existence of elsewhere acute intracranial infarction or hydrocephalus significantly influenced the prognosis in the current series (p value < 0.01 and < 0.05 respectively), where a poor *mRS* (i.e., ≥ 3) was related to twelve cases suffered from additional intracranial infarction (cerebral/or brainstem). (Fig. 4)

Similarly, *Pfefferkorn et al.* in their retrospective case series observed unfavorable outcome ($mRS > 2$) in 60% of cases and 76% in the subcategory with superadded brainstem infarction, and the death rate was 40% and 58%, respectively [21]. Also in a recent study by *Suyama Y. et al.*, [23] comparing the good and poor prognosis groups, a significant difference was found in "Infarction in areas other than the cerebellum". Whereas several studies [18,24,26] support this belief, controversy is still present regarding the usefulness of ventricular drainage alone versus that in conjunction with suboccipital decompressive surgery for the management of the associated hydrocephalus. *Pallesen et al.*, in their recent review study, recommend implementation of suboccipital decompressive surgery and EVD insertion, while being ready for EVD withdrawal as soon as postoperative follow-up imaging exhibit returned usual CSF flow dynamics [20]. Similarly, *Rieke et al.* reported 42 cerebellar infarction cases, of whom 20 cases managed conservatively, 7 went for suboccipital decompressive surgery, and 15 only performed ventricular drainage. They recommended ventricular drainage for hydrocephalus-induced stupor, while for those who are comatose

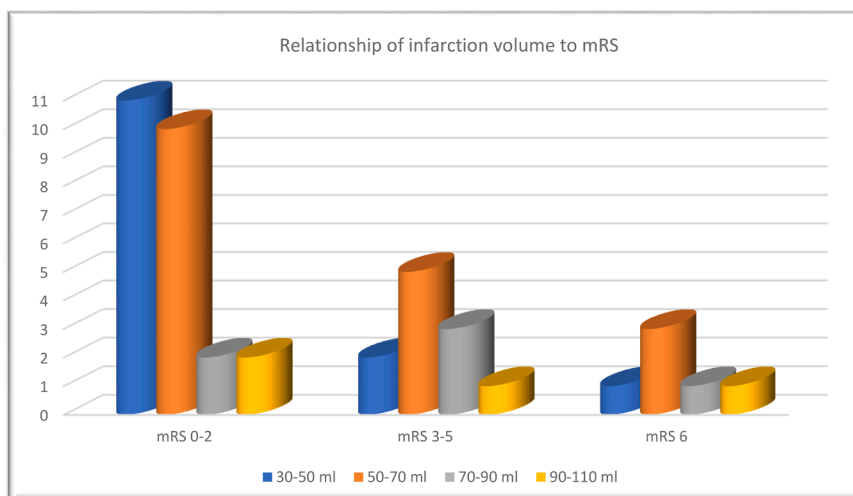


Fig. 3. Relationship of infarction volume to mRS.

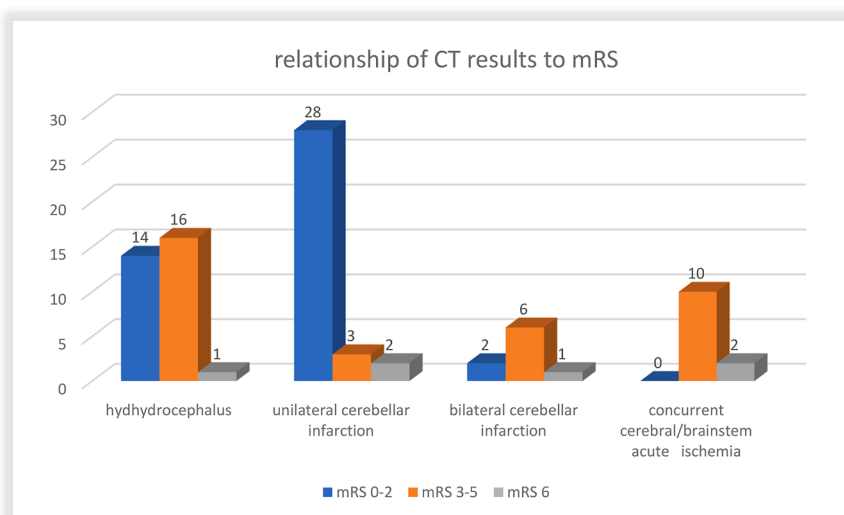


Fig. 4. Relationship of CT-findings to mRS.

because of brainstem compression, suboccipital decompressive surgery is indicated [22]. On the other hand, *Jauss et al.* found no difference between ventricular drainage and decompressive surgery for their studied 84 cases presented by CT evidence of mass effect induced by acute cerebellar infarction, where suboccipital decompressive surgery was done for 34 cases and ventricular drainage for another 14 cases [11]. To resolve this conflict a meta-analysis study by *Oliver et al.* reported less mortality with growing rates of synchronously external ventricular drain insertion, and infarcted tissue debridement [19]. Additionally, because of assumed transtentorial upward herniation following enhanced CSF drainage [8], ventricular drainage alone has been discouraged as it may predispose to serious consequences (hemiplegia and disturbed level of consciousness) [2,3,14,16,18]. However, the necessity for ventricular CSF drainage alone may be overcome by early suboccipital decompressive surgery. The dilemma of this issue needs to be resolved through extended researches and more analytical studies in the forthcoming future.

In the current study, hydrocephalus has been found in 31 (73.8%) cases and represented a poor prognostic factor ($p < 0.05$). The majority (17 cases, 54.8%) has fallen in the category of mRS ≥ 3 , where 16 cases (51.6%) belong to mRS = 3–5, and 1 case (3.22%) belong to mRS = 6. However, the synchronous ventriculostomy for CSF diversion and ESC in

the rest of the cases ($n = 14, 45.1\%$) offered a better outcome (mRS ≤ 2), that is comparable to those who also benefit from ESC alone ($n = 11$) since they escaped the development of hydrocephalus but still suffering the mass effect on the surrounding neuronal structures caused by the massive cerebellar swelling. It is to be stressed here that none of our cases did ventricular drainage solely throughout their management. Lastly, it is to be noted that twenty-five (59.5%) out of 42 patients achieved mRS above 2, which is comparable to results in previous studies by *Feely [7]* and *Tsitsopoulos et al. [26]* whereas, others like *Ogasawara et al. [18]* reported 70% (7 patients out of 10) favorable recovery after decompressive surgery, and *Suyama Y. et al. [23]* who also reported positive results in 10 (71.4%) of 14 patients, however, the little number of investigated cases in their studies made these results questionable and incomparable to the current one (Fig. 5).

This study may be limited by its retrospective methodology and the relatively small number of cases; however, it still can support our study conclusion. Many kinds of research studying outcomes encountered limitations such as inadequate sample size, retrospective data assembly, deficiency of control groups, single-center experience, and other selection biases [17]. These limitations seem to be widespread in many previous studies, as stated by *Oliver et al., [19]* in a recent analytical study where they concluded that: “a significant drawback to build up a

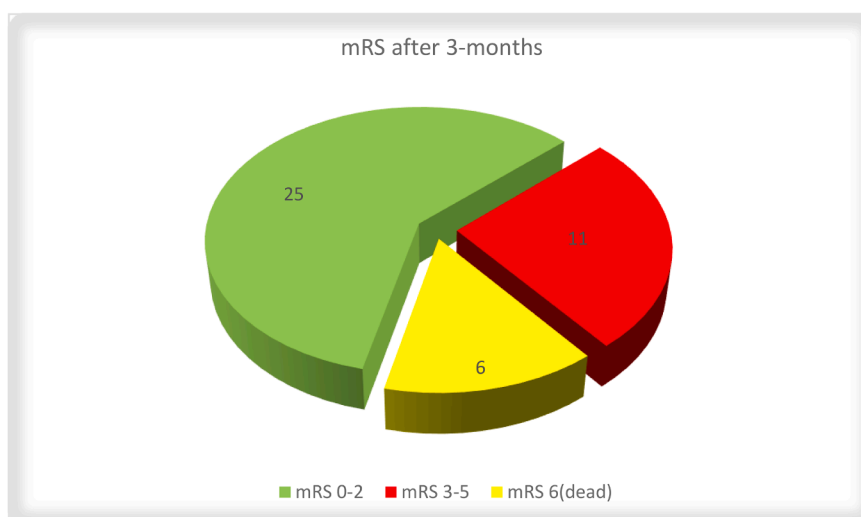


Fig. 5. Outcome values via three months post-stroke mRS.

better concept about the impression of decompressive surgery on the outcome, had emerged from the deficiency of consistent reporting methods for a suboccipital decompressive craniectomy". Similarly, Neugebauer et al., declared that additional studies are required to explore relevant factors systematically and offer improved selection measures to support decisions concerning surgical management, which should constantly be implemented in close collaboration amongst neurosurgeons, neuro-intensivists, and neurologists [17]. Hence, from our point of view, fruitful knowledge and facts may emerge in the future through performing a meta-analysis of individual patient facts by assembling data from a set of relevant researches from numerous institutions, thus creating a large relevant cohort study to establish the guidelines and fundamental procedures that should be followed worldwide.

In brief, decompressive surgery of posterior fossa with or without EVD is a mandatory and valuable approach for the management of post-ischemic acute cerebellar swelling, however respecting the aforementioned prognostic factors and considering the pre-existing neuroradiological imaging, and the overall clinical status of each particular patient.

5. Conclusions

After inspecting the critical management of 42 patients suffering from acute ischemic cerebellar infarctions with mass effect, we can advocate that emergent suboccipital craniectomy (ESC) is indicated for treating post-ischemic cerebellar massive swelling in patients with GCS ≤ 13 , where good prognosis could be achieved with (ESC) in absence of intracranial infarction elsewhere, while in those who already have obstructive hydrocephalus at the time of surgery, a poor prognosis ensues.

6. Declarations

No Presentation at a conference.

No funding from any organization for this work.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

- [1] H.P. Adams Jr, G. del Zoppo, M.J. Alberts, D.L. Bhatt, L. Brass, A. Furlan, et al., Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists, *Circulation* 115 (2007) e478–e534 (Erratum in *Circulation* 116:e515, 2007).
- [2] T. Andoh, N. Sakai, H. Yamada, T. Hattori, Y. Miwa, T. Hirata, Y. Tanabe, A. Ohkuma, T. Funakoshi, M. Takada, Cerebellar infarction: analysis of 33 cases, *No Shinkei Geka* 18 (1990) 821–828 (in Japanese).
- [3] L.M. Auer, T. Auer, I. Sayama, Indication for surgical treatment of cerebellar hemorrhage and infarction, *Acta Neurochir. (Wien)* 79 (1986) 74–79.
- [4] O.G.S. Ayling, N.M. Alotaibi, J.Z. Wang, M. Fatehi, G.M. Ibrahim, O. Benavente, et al., Suboccipital decompressive craniectomy for cerebellar infarction: a systematic review and meta-analysis, *World Neurosurg.* 110 (2018) 450–459.e5. Available from <https://linkinghub.elsevier.com/retrieve/pii/S1878875017318727>.
- [5] R. Bonita, R. Beaglehole, Modification of Rankin Scale: recovery of motor function after stroke, *Stroke* 19 (12) (1988) 1497–1500.
- [6] European Stroke Organisation (ESO) Executive Committee, Guidelines for management of ischemic stroke and transient ischemic attack 2008, *Cerebrovasc Dis* 25 (2008) 457–507.
- [7] M.P. Feely, Cerebellar infarction, *Neurosurgery* 4 (1979) 7–11.
- [8] Y. Hara, K. Hosoda, K. Ohta, Upward transtentorial herniation associated with severe posterior fossa subarachnoid hemorrhage due to vertebral artery dissecting aneurysm: a report of two cases, *Japanese J Neurosurg (Tokyo)* 16 (2007) 423–427 (in Japanese).
- [9] C.R. Hornig, D.S. Rust, O. Busse, M. Jauss, A. Laun, Space occupying cerebellar infarction: clinical course and prognosis, *Stroke* 25 (1994) 372–374.
- [10] <http://www.itksnap.org/pmwiki/pmwiki.php?n=Main.Downloads>.
- [11] M. Jauss, D. Krieger, C. Hornig, J. Schramm, O. Busse, Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study, *J Neurol* 246 (1999) 257–264.
- [12] E. Jüttler, S. Schwab, P. Schmiedek, A. Unterberg, M. Hennerici, J. Woitzik, S. Witte, E. Jenetzky, W. Hacke, Decompressive surgery for the treatment of malignant infarction of the middle cerebral artery (DESTINY): a randomized, controlled trial, *Stroke* 38 (2007) 2518–2525.
- [13] E. Jüttler, S. Schwickert, P.A. Ringleb, H.B. Huttner, M. Köhrmann, A. Aschoff, Long-term outcome after surgical treatment for space occupying cerebellar infarction: experience in 56 patients, *Stroke* 40 (2009) 3060–3066.
- [14] M. Khan, K.S. Polyzoidis, A.B. Adegbite, J.D. McQueen, Massive cerebellar infarction: "conservative" management, *Stroke* 14 (1983) 745–751.
- [15] M.J. Kim, S.K. Park, J. Song, S.Y. Oh, Y.C. Lim, S.Y. Sim, Y.S. Shin, J. Chung, Preventive suboccipital decompressive craniectomy for cerebellar infarction: a retrospective-matched case-control study, *Stroke* 47 (2016) 2565–2573.
- [16] A. Laun, O. Busse, V. Calatayud, N. Klug, Cerebellar infarcts in the area of the supply of the PICA and their surgical treatment, *Acta Neurochir (Wien)* 71 (1984) 295–306.
- [17] H. Neugebauer, et al., Space-occupying cerebellar infarction: complications, treatment, and outcome, *Neurosurg Focus*, 2013;34 5: E8 (<http://thejns.org/doi/abs/10.3171/2013.2.FOCUS12363>).
- [18] K. Ogasawara, K. Koshu, Y. Nagamine, S. Fujiwara, K. Mizoi, T. Yoshimoto, Surgical decompression for massive cerebral infarction, *No Shinkei Geka* 23 (1995) 43–48 (in Japanese).
- [19] Oliver G.S. Ayling, et al., Suboccipital decompressive craniectomy for cerebellar infarction: a systematic review and meta-analysis, *World Neurosurg.* 110 (2018) 450–459.e5.
- [20] L.-P. Pallesen, K. Barlinn, V. Puetz, Role of decompressive craniectomy in ischemic stroke, *Front. Neurol.* 9 (2019) 1119, <https://doi.org/10.3389/fneur.2018.01119>.
- [21] T. Pfefferkorn, U. Eppinger, J. Linn, T. Birnbaum, J. Herzog, A. Straube, et al., Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction, *Stroke* 40 (2009) 3045–3050. Available from <https://www.ahajournals.org/doi/10.1161/STROKEAHA.109.550871>.
- [22] K. Rieke, D. Krieger, H.P. Adams, A. Aschoff, U. Meyding-Lamadé, W. Hacke, Therapeutic strategies in space-occupying cerebellar infarction based on clinical neuroradiological and neurophysiological data, *Cerebrovasc. Dis.* 3 (1993) 45–55.
- [23] Y. Suyama, et al., Evaluation of clinical significance of decompressive suboccipital craniectomy on the prognosis of cerebellar infarction, *Fujita Med. J.* 5 (1) (2019), <https://doi.org/10.20407/fmj.2018-010>.
- [24] Z. Tchopev, M. Hiller, J. Zhuo, J. Betz, R. Gullapalli, K.N. Sheth, Prediction of poor outcome in cerebellar infarction by diffusion MRI, *Neurocrit Care* 19 (2013) 276–282.
- [25] The Japan Stroke Society. Japanese Guideline for Management of Stroke 2015. Tokyo: Kyowa-Kikaku; 2015: 66 (in Japanese).
- [26] P.P. Tsitsopoulos, L. Tobieson, P. Enblad, N. Marklund, Clinical outcome following surgical treatment for bilateral cerebellar infarction, *Acta Neurol. Scand.* 123 (2011) 345–351.
- [27] E.F. Wijdicks, K.N. Sheth, B.S. Carter, D.M. Greer, S.E. Kasner, W.T. Kimberly, S. Schwab, E.E. Smith, R.J. Tamargo, M. Wintermark, Recommendations for the management of cerebral and cerebellar infarction with swelling: a statement for healthcare professional from the American Heart Association/American Stroke Association, *Stroke* 45 (2014) 1222–1238.
- [28] N. Montemurro, G. Santoro, W. Marani, G. Petrella, Posttraumatic synchronous double acute epidural hematomas: two craniotomies, single skin incision, *Surg. Neurol. Int.* 11 (11) (2020) 435, <https://doi.org/10.25259/SNI.697.2020>. PMID: 33365197; PMCID: PMC7749931.
- [29] Cy Tao, H. Feng, Jj Wang, et al., Delayed posterior fossa epidural hematoma originating from occipital artery after infratentorial craniotomy, *Neurol Sci* 36 (2015) 1267–1269, <https://doi.org/10.1007/s10072-014-2003-2>.