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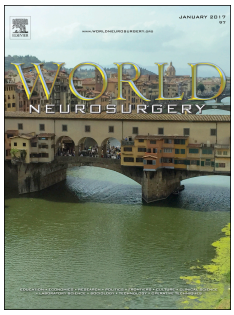

Posterior Fossa Surgery For Stroke: Differences In Outcomes Between Cerebellar Haemorrhage and Infarcts.

Lester Lee, MBBS, MMed (Surg), FRCS (SN), Daniel Loh, MBBS, AKC, Nicolas Kon Kam King, FRCS (SN), PhD

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Posterior Fossa Surgery For Stroke: Differences In Outcomes Between Cerebellar Haemorrhage and Infarcts.

Lester Lee[a,b,c] MBBS, MMed (Surg), FRCS (SN); Daniel Loh[a,b] MBBS, AKC; Nicolas Kon Kam King[a,b,c], FRCS (SN), PhD

[a]Department of Neurosurgery, National Neuroscience Institute, Singapore

[b]Department of Neurosurgery, Singapore General Hospital, Singapore

[c]Duke-NUS Medical School, Singapore

Corresponding Author:

Dr Lester Lee

National Neuroscience Institute

11 Jalan Tan Tock Seng

Singapore 308433

Tel number: +6563577191

lester_ch_lee@nni.com.sg

Posterior Fossa Surgery For Stroke: Differences In Outcomes Between Cerebellar Haemorrhage and Infarcts.

Abstract

Introduction

Posterior fossa surgery is the established treatment for large cerebellar strokes with brainstem compression. Despite this, there is a paucity of data for long-term outcomes.

Methods

A retrospective analysis of patients who underwent posterior fossa surgery for cerebellar haemorrhages and infarcts was performed to compare their difference in 6-month outcomes and to identify factors which affect outcomes. Patients were dichotomised into groups with good outcomes (mRS 0-3) or poor outcomes (mRS 4-6). Sex, age, pre-operative GCS, Charleston comorbidity index, time to surgery, intraventricular haemorrhage, surgical complications, length of ICU and hospital stay, shunt dependence and tracheostomy rates were analysed.

Results

126 patients were recruited: 76 in haemorrhage group and 50 in infarct group. There was a higher mortality in the haemorrhage group ($p = 0.0730$). At 6 months, more patients in the haemorrhage group had poor outcomes ($p = 0.0074$, OR 3.04) and higher mortality ($p = 0.0730$, OR 2.20). More patients in the haemorrhage group required a tracheostomy ($p = 0.0245$). Factors predictive of poor outcome include older age ($p = 0.0108$), GCS ≤ 8 ($p = 0.0011$) and tracheostomy ($p = 0.0269$). 69.2% of patients had improvements in mRS at 6

months. Shorter length of stay (p = 0.0003) and discharge to a rehabilitation hospital (p = 0.0001) were predictive of functional improvement.

Conclusion

Patients who underwent posterior fossa surgery for cerebellar haemorrhages had worse outcomes compared to patients with cerebellar infarcts and were more likely to require a tracheostomy. Rehabilitation helped to improved outcomes.

Introduction

Since the first reported cases of surgery for cerebellar stroke in 1956[1,2], posterior fossa surgery has become the recommended treatment for large cerebellar haemorrhages or cerebellar infarcts with swelling leading to brainstem compression[3,4,5]. In the treatment of cerebellar stroke, posterior fossa surgery is performed to remove the suboccipital bone to decompress the cerebellum or evacuate the haematoma, to reduce the mass effect on the brainstem. It has been found to be more effective with better outcomes when compared to the insertion of an external ventricular drain alone to treat hydrocephalus that may arise secondary to fourth ventricular obstruction from haematoma or cerebellar swelling[6,7,8].

Mortality in comatose patients with space-occupying cerebellar strokes managed with conservative therapy, can be as high as 85%[9]. Mortality for patients after posterior fossa surgery is still high, ranging from 19.9% to 40%[6, 10, 11, 12], but that is significantly lower than treatment with maximal medical therapy alone. As such, suboccipital decompressive craniectomy has also been shown to result in reduced mortality in studies comparing surgery to best medical therapy[13].

Despite evidence showing the life-saving benefits of posterior fossa surgery for cerebellar strokes, there is a paucity of data for the long term outcomes for these patients. There are also very few studies that compare the long term outcomes between patients who have cerebellar haemorrhages and infarcts who have undergone posterior fossa surgery. We conducted a study to compare the difference in long term outcome between patients with cerebellar haemorrhages and infarcts who have undergone posterior fossa surgery, as well as to determine factors which can contribute to improving these outcomes.

Methods

We conducted an institution review board approved retrospective study of all patients admitted to the National Neuroscience Institute, Singapore from October 2006 to November 2017 who underwent posterior fossa decompressive craniectomy for cerebellar haemorrhage and cerebellar infarcts with significant mass effect. They were then divided into an infarct group for patients who had cerebellar infarcts and a haemorrhage group for patients with cerebellar haemorrhage.

Surgical Technique

All patients included in our study underwent the insertion of an external ventricular drain followed by posterior fossa decompressive craniectomy for cerebellar haemorrhage or cerebellar infarct. Patients who underwent only the insertion of an external ventricular drain to treat hydrocephalus were not included.

All decompressive craniectomies were performed in prone position. An external ventricular drain was inserted first before the decompressive craniectomy. A midline incision starting from the suboccipital bone to the mid cervical region was used. Dissection was performed to expose the suboccipital bone, as well as the posterior arch of the first cervical vertebrae. A suboccipital craniectomy as well as a C1 laminectomy was then performed. The dura was opened in a Y-shaped fashion. If there was a cerebellar bleed, the clot would then be removed via a cortisectomy. A dural graft was then stitched to the dura to form a watertight closure. The layers of muscle and fascia were then stitched together.

Outcome assessment

The outcome assessment used in our study was the modified Rankin scale (mRS). We defined patients with an mRS of 1-3 as having a good outcome and patients with mRS of 4-6 as having a poor outcome. Outcome assessments were performed at discharge, at the first follow-up visit which is usually between 4-8 weeks from discharge and after 6 months. Patients' mRS were scored from their discharge summaries and outpatient clinical notes.

Variables collected

We also collected variables to look for factors that may affect the outcome out patients. Variables collected include: sex, age, pre-operative Glasgow Coma Scale, patient's Charleston Comorbidity Index (CCI), time from admission to surgery, presence of intraventricular haemorrhage, surgical complications, ICU length of stay, overall length of hospital stay, shunt dependence and the need for long term tracheostomy. In the haemorrhage group, addition variables recorded include the diameter of the blood clot and the degree of evacuation (Total – 100% removed, subtotal >50% removed, partial <50% removed).

Patients who were repatriated back to their countries of origin after their initial hospital stays would have their inpatient variables collected for analysis, but not for analysing long term outcomes as there is no longer follow-up with us.

Data analysis

R version 3.3.0 was used for all statistical analyses. The Shapiro-Wilk test was used to test for normality. Non-normally distributed variables were represented by medians and interquartile ranges, while normally distributed variables were described using means and standard deviations. A Kaplan-Meier survival graph was plotted. Fischer's T-test was used to

compare discontinuous variables to look for statistical significance. Predictors of survival and improvement were determined using multivariate analysis followed by regression analysis. A p value of <0.05 was considered statistically significant.

Results

Patient demographics

126 patients were recruited into our study. 88 patients were male while 38 were female. There were 76 patients in the haemorrhage group and 50 patients in the infarct group. Both groups had similar comorbitities with the CCI ranging from 0-8 (2) in the haemorrhage group and 0-7 (2) in the infarct group. Age was also similar in both groups, ranging from 20 – 80 (59.3 ±13.7) years in the haemorrhage group and 28 – 79 (57.3 ± 12.0) years. There were more patients with poorer pre-operative GCS. 42 patients had a pre-operative GCS of ≤ 8 in the haemorrhage group while 12 patients had a pre-operative GCS ≤ 8 in the infarct group (p = 0.0008). Patients with haemorrhage underwent surgery with shorted average times during their admission compared with the infarct group (13.9 ± 45 hours versus 47.3 ± 55.1 hours). More patients in the haemorrhage group (n= 50) had intraventricular haemorrhage compared to the infarct group (n= 2) (p= 0.0001) (see **Table 1**). The overall follow-up period was 6-144 months with an average of 42.2 months.

Length of ICU stay for the haemorrhage group ranged from 3 – 45 days (9.4 ± 7.0) while it ranged from 3-69 days (8.2 ± 9.5) for the infarct group. More patients in the haemorrhage group (n = 27) required a tracheostomy compared to the infarct group (n = 8) (p = 0.0245) (see **Figure 1**). 15 patients from the haemorrhage group underwent the insertion of a ventriculoperitoneal shunt by discharge, compared to 8 in the infarct group (p = 0.6449). 28

patient from the haemorrhage group and 23 patients from the infarct group was sent to a rehabilitation hospital for further intensive rehabilitation. The overall hospital length of stay was 2-158 (37.0 ± 32.6) days for the haemorrhage group and 3-180 (40.1 ± 41.5) days for the infarct group. Total inpatient mortality was 29.3% (n = 37) with 27 patients in the haemorrhage group and 10 patients in the infarct group. More patient died in hospital from cerebellar haemorrhage than infarcts (p = 0.0730, OR 2.2). (see **Figure 2**). Predictive factors for inpatient mortality include male gender (p = 0.0076, OR 2.9), post-operative complications (p = 0.0040, OR 1.08), longer ICU length of stay (p = 0.0002) and longer hospital length of stay (p = 0.0001).

Overall outcomes at 6 months

9 patients were repatriated back to their countries of origin after their initial hospital stays and were lost to long term follow-up. 117 patients had fully documented outcomes for at least 6 month in our study. Overall 6 month mortality was 33.33% (n=39), comprising of 28 patients in the haemorrhage group (36.84%) and 11 patients in the infarct group (22%). There was a higher incidence of mortality in the haemorrhage group, but it was not statistically significant (p=0.1084, OR 2.04).

At 6 months, 49 (69.0%) patients in the haemorrhage group had poor outcome (mRS 4-6) compared to 20 (43.5%) patients in the infarct group (p = 0.0074, OR 3.04) (see **Figure 3**). 2 patients deteriorated further and died between discharge and 6 months. Factors predictive of poor outcome include older age (p = 0.0108), GCS ≤ 8 (p = 0.0011, OR 9.04) and the need for a tracheostomy (p = 0.0269, OR 14.3).

There were 78 surviving patients at 6 months. 54 (69.2%) patients showed improvement from the initial stroke at 6 months. 21 (26.9%) patients showed improvement of mRS ≥2 at 6 months. 22 patients in the infarct group and 19 patients in the haemorrhage group showed functional improvement by mRS ≥1 from discharge to the first follow-up (p=0.1225). 13 patients from the infarct group and 14 patients from the haemorrhage group showed delayed functional improvement by mRS ≥1 from the first follow-up to the period after 6 months (p=0.4738) (see **Figure 4**). Factors that influenced improvement in our patients include shorter total length of stay (p = 0.0003) and discharged from hospital to undergo further rehabilitation at a dedicated rehabilitation hospital (p = 0.0001).

Cerebellar infarcts

There were 50 patients in the infarct group. 38 patients were male while 12 were female. Their age ranged from 28-79 (57.3 ± 12.0). Time to surgery ranged from 1 to 333 (47.3 ±55.1) hours. Vascular territories of infarction included: right PICA – 7 patients, left PICA – 5, bilateral PICA – 1 patient, right PICA and AICA – 3 patients, Right PICA and SCA – 4 patients, right PICA, AICA and SCA – 2 patients, left PICA and AICA – 3 patients, left PICA and SCA – 8 patients, left PICA, AICA and SCA – 5 patients, bilateral PICA and SCA – 3 patients, bilateral PICA, AICA and SCA – 2 patients, right AICA and SCA – 2 patients, right SCA – 2 patients, bilateral SCA – 2 patients, bilateral SCA and AICA – 1 patient. Predictors of poor outcome at discharge include male gender (p = 0.0748, Or 3.92), ICU (p = 0.0558) and total hospital length of stay (p = 0.0681), but they were not statistically significant. Predictors of good outcome at 6 months include shorter ICU (p = 0.0406) and total length of stay (p = 0.0224) as well as better mRS at discharge (p = 0.0294).

Cerebellar bleeds

There were 76 patients in the haemorrhage group. 50 patients were male while 26 were female. Their age ranged from 20 - 83 (59.3 ±13.7) years. Time to surgery ranged from 0 - 336 (13.9 ± 45) hours. Areas of haemorrhage included: right cerebellum – 37 patients, left cerebellum – 28 patients, cerebellar midline – 7 patients and bilateral cerebellum – 4 patients. Predictors of poor outcome at discharge include GCS ≤ 8 ($p = 0.0158$, OR 7.2) and larger bleed diameter ($p = 0.0143$). Predictors of good outcome at 6 months include shorter total length of stay ($p = 0.0459$) and discharge to a dedicated rehabilitation centre ($p = 0.0005$).

Discussion

Surgery for Cerebellar Strokes

Large cerebellar strokes causing mass effect results in poor outcomes for patients managed conservatively[9, 14]. Cytotoxic and vasogenic oedema from large cerebellar infarcts result in brain swelling, causing compression of the fourth ventricle and brainstem giving rise to hydrocephalus and loss of consciousness[15]. In cerebellar haemorrhage, clinical deterioration can result from increased mass effect from surrounding clot oedema or expansion of the haematoma from repeated bleeding. Either of these mechanisms can cause brainstem compression which leads to either upwards herniation through the tentorium incisura or downward tonsillar herniation through the foramen magnum. Cerebellar clots can also directly compress the fourth ventricle or rupture into the ventricular system, resulting in obstructive hydrocephalus which is another mechanism of clinical decline[16, 17].

Since 1956, the potential of posterior fossa surgery has been recognised to address this issue[1,2]. Multiple studies have since shown the safety and efficacy of posterior fossa decompressive craniectomy in the relief of cerebellar swelling and brainstem compression[8, 18, 19, 20, 21, 22, 23, 24]. Suboccipital craniectomy with the insertion of a ventricular drain is

potentially more favourable than the insertion of a ventricular drain alone when dealing with cerebellar swelling[7]. By removing the suboccipital bone and the resection of the posterior arch of the atlas in combination with dural enlargement, the cerebellum is therefore able to swell posteriorly, thus relieving pressure on the brainstem. Suboccipital decompression also allows access for removal of a cerebellar haematoma or infarcted tissue, thereby decreasing pressure in the cerebellum. Treatment with an external ventricular drain alone however, may also result in a rare phenomenon of upward herniation called “inverse herniation”, which is a complication recognised since the 1960s[25, 26]. It is characterised by a displacement of parts of the posterior fossa through the tentorium in a caudal to cranial manner[27].

The indications for surgery are more estabilished. The American Heart Association/American Stroke Association (AHA/ASA) recommend suboccipital craniectomy with dural expansion for patients who deteriorate clinically despite medical management. A ventriculostomy should be accompany the suboccipital decompression to treat hydrocephalus[5]. For cerebellar haematomas, AHA/ASA recommend surgical removal of haematomas more than 3cm in diameter, in patients who deteriorate neurologically or in patients who have brainstem compression and/or hydrocephalus from ventricular obstruction[4]. In patients with significant space occupying oedema, resection of the atlantic arch and a duraplasty may be necessary[27]. Other accepted treatment algorithms for cerebellar haematomas include Kobayashi et al’s protocol which uses the patient’s GCS score at admission and the haematoma size on a CT brain scan as discriminating factors – patients with a high GCS score (14 or >14) on admission with a less than 4cm haematoma can be managed conservatively, while patients with a lower GCS score (13 or <13) should undergo surgical decompression[28]. Kirollos et al proposed a protocol that does not take into account the size of the haematoma. The indication for surgery was instead based on the compression of the 4th ventricle and the presenting GCS.

The fourth ventricle was graded into I to III, with I being normal, II being compressed and III being absent. The patient's presenting GCS was then assessed and dicotomised into either less than 13, or more than 13. Surgical evacuation was recommended for conscious patients with grade III fourth ventricular compression. Their study team believed that large haematomas (>4cm) may not need to be evacuated if there is no fourth ventricular compression[29].

Differences in Outcomes Between Cerebellar Haemorrhage and Infarcts

There is currently a paucity of data in the literature comparing the differences in long term outcomes for patients who have undergone posterior fossa surgery for cerebellar haemorrhage and infarcts. Previous studies either compared the long-term outcomes as a group comprised of patients with cerebellar haemorrhages and infarcts who have undergone surgery[11], or analysed the efficacy of conservative versus surgical management in these two types of stroke[14]. One of the reasons there is a lack of data maybe the small number of patients requiring surgery. As such, studies on this population tend to have small cohorts. The 126 patients in our study were recruited from two of the largest neurosurgical centres in Singapore, over a period of 11 years. It is one of the largest studies looking at long term outcome in patients who have undergone posterior fossa surgery for cerebellar stroke.

In our study, patients in the haemorrhage group had poorer outcomes (mRS 4-6) at 6 months compared to the infarct group (p=0.0074) (See **Figure 3**). Patients with cerebellar haemorrhages were operated on earlier compared to cerebellar infarcts (mean 13.9 hours vs 47.3 hours), had poorer preoperative GCS and had significantly more intraventricular haemorrhage, suggesting greater neurological damage (see **Table 1**). There was also a higher incidence of mortality in the haemorrhage group, but it was not statistically significant when

compared to the infarct group (see **Figure 2**). More patients in the haemorrhage group also required a tracheostomy compared to the infarct group (p=0.0245) (see **Figure 1**). The risk of prolonged ventilator dependence is poorly described in this population, but other studies have suggested that respiratory recovery maybe slower in patients with cerebellar haemorrhage[30, 31]. The poorer pre-operative GCS seen in patients with cerebellar haemorrhage is likely due to the nature of the haemorrhage which can compress the brainstem or the 4th ventricle resulting in hydrocephalus. Cerebellar haemorrhage occurs rapidly and large haematomas can compress the brainstem fairly quickly, causing patients to become comatose. Neurological deterioration can develop rapidly and occur over minutes to hours[3,4,5]. Intraventricular haemorrhage from cerebellar clots which rupture into the ventricular system can also cause hydrocephalus resulting in poor GCS. This is in contrast to cerebellar infarcts whereby infarcted tissue results in oedema which can compress the 4th ventricle and brainstem over a longer period of time[24].

With regards to long term improvements, there was no difference in the number of patients who improved with an MRS $\geq$ 1 in both groups of patients from discharge to 6 months. This suggests that function improvements are independent of the pathology of the cerebellar haemorrhage and infarcts causing brain injury.

Factors Affecting Long-term Outcome

Our results showed that over time, there was a gradual trend of functional improvement in both groups of patients with cerebellar haemorrhage and infarcts (see **Figure 4**). A shorter length of stay in hospital and discharge to a rehabilitation centre for further rehabilitation were both factors that were associated with an improvement in mRS after discharge from hospital. The shorter length of stay can possibly be attributed to having a better neurological

status after surgery, as patients with surgical complications or depressed neurological state would need more medical care and be discharged later. Studies have shown that rehabilitation can be useful in improving the overall long-term functional outcomes in patients with brain injury[32, 33]. This correlates with our findings but it should be interpreted with caution as patients who have very severe injury may not be able to participate in a rehabilitation programme and may not be have the natural ability to improve.

Factors predictive of poor outcome include need for tracheostomy, lower pre-operative GCS (≤ 8) and older age. A tracheostomy would be needed for patients with poorer neurological state, but older age and lower pre-operative GCS have been associated with poorer outcomes in other studies as well[30, 34, 35].

Study Limitations

Due to the retrospective nature of our study, there were some limitations involved. The number of patients in both groups were not evenly matched, this is because more patients with haemorrhagic strokes were operated on compared to ischaemic strokes. We were unable to follow-up the patients who went back to the country of their origin and therefore, we were unable to analyse their long-term outcomes. We were also unable to follow up our patients at regular intervals since their follow-up at discharge would vary. In other to produce a trajectory of recovery over time, we had to group their outcome assessments over three intervals; at discharge, at their first follow-up which is within 6 months and after 6 months at their last review.

Conclusion

Our study showed that patients who underwent posterior fossa surgery for cerebellar bleeds had worse long-term functional outcomes compared to patients with cerebellar infarcts. This could be due to worse neurological damage as characterised by shorter time to surgery, poorer pre-operative GCS and higher incidence of intraventricular haemorrhage. They were also more likely to be ventilator dependent, requiring a tracheostomy. Patients who were older also had poorer outcomes.

Despite the high rates of mortality, most patients who undergo posterior fossa surgery for cerebellar strokes improve functionally over the long term. Discharge from hospital to a rehabilitation centre was shown to result in better improvements in functional outcomes in both groups of patients. Predictive factors identified in this study can help in future patient selection for surgery and improve management to optimise patient outcomes.

References

1. Fariburn B, Oliver LC. Cerebellar softening: A surgical emergency. BMJ. 1956. 1: 1335-1336.

2. Lindgren SO. Infarctions simulating brain tumours in the posterior fossa. J Neurosurg. 1956. 13: 575-581.

3. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, Krieger D, Mayberg M, Morgenstern L, Ogilvy CS, Vespa P, Zuccarello M. Guidelines for the management of spontaneous intracerebral haemorrhage in adults. Circulation. 2007. 116: e391-e413.

4. Hemphill JC, Greenberg SM, Anderson CS, Becker K, bendok BR, Cushman M, Fung GL, Goldstein JN, Macdonald RL, Mitchell PH, Scott PA, Selim MH, Woo D. Guidelines for the management of spontaneous intracerebral haemorrhage. Stroke. 2015. 46: 000-000.

5. Wijdicks EFM, Sheth KN, Carter BS, Greer DM, Kasner SE, Kimberly WT, Schwab S, Smith EE, Tamargo RJ, Wintermark M. Recommendations for the management of cerebral and cerebellar infarct with swelling. Stroke. 2014. 45: 1222 – 1238.

6. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, Dichgans M, Grau S. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. Stoke. 2009. 40: 3045-3050.

7. Kudo H, Kawaguchi T, minami H, Kuwamura K, Miyata M, Kohmura E. Controversy of surgical treatment for severe cerebellar infarction. J Stroke Cerebrovasc Dis. 2007.16 (6): 259-262.

8. Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German Austrian Cerebellar Infarction Study. J neurol. 1997. 246: 257 – 264.

9. Heros RC. Cerebellar haemorrhage and infacrtion. Stroke. 1982. 13: 106 – 109.

10. Juttler E, Schweickert S, Ringleb PA, Huttner HB, Kohrmann M, Aschoff A. Long-term outcome after surgical treatment for space occupying cerebellar infarction. Experience in 56 patients. Stroke. 2009. 40: 3060–3066.

11. Puffer RC, Graffeo C, Rabinstein A, Gompel JJ. Mortality rates after emergent posterior fossa decompression for ischaemic or haemorrhagic strokes in older patients. World Neurosurg. 2016. 92: 166-170.

12. Ayling OGS, Alotaibi NM, Wang JZ, Fatehi M, Ibrahim GM, Benavente O, Field TS, Gooderham PA, Macdonald RL. Suboccipital deompressive craniectomy for cerebellar infarction: A systemic review and meta-analysis. World Neurosurg. 2018. 110: 450 – 459.

13. Luney MS, English SW, Longworth A, Simpson J, Gudibande S, Matta B, Burnstein RM, Veenith. Acute posterior cranial fossa haemorrhage – Is surgical decompression better than expectant medical management? Neurocrit Care. 2016. 25: 365 – 370.

14. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D. Neurosurgical management of cerebellar haematoma and infarct. J Neurol Neurosurg Psychiatry. 1995. 59: 287-292.

15. Chen HJ, Lee TC, Wei CP. Treatment of cerebellar infarction by decompressive suboccipital craniectomy. Stroke. 1992. 23: 957-961.

16. Amar AP. Controversies in the neurosurgical management of cerebellar haemorrhage and infarction. Neurosurg Focus. 2012. 32 (4): 1-9.

17. Datar S, Rabinstein AA. Cerebellar Haemorrhage. Neurol Clin. 2014. 32: 993 – 1007.

18. Kim MJ, Park SK, Song J, Oh S, lim YC, Sim SY, Shin YS, Chung J. Preventive suboccipital decompressive craniectomy for cerebellar infarction. A retrospective match case control study. Stroke. 2016. 47: 2565-2573.

19. Michel P, Arnold M, Hungerbuhler HJ, Muller F, Staedler C, Baumgartner RW, Georgiadis D, Lyrer P, Mattle HP, Sztajzel R, Weder B, Tettenborn B, Nedeltchev K, Englter S, Weber SA, Basciani R, Fandino J, Fluri F, Stocker R, Keller E, Wasner M, Hanggi M, Gasche Y, Paganoni, Regli L. Decompressive craniectomy for space occupying hemispheric and cerebellar ischaemic strokes: Swiss recommendations. Int J Stroke. 2009. 218-223.

20. Pallesen LP, Barlinn K, Puetz V. Role of decompressive craniectomy in ischaemic stroke. Front Neurol. 2019. 9: 1119.

21. Hackenberg KAM, Unterberg AW, Jung CS, Bosel J, Schonenberger S, Zweckberger K. Does suboccipital decompression and evacuation of intraparenchymal hematoma improve neurological outcome in patients with spontaneous cerebellar haemorrhage? Clin Neurol Neurosurg. 2017. 155: 22-29.

22. Sykora M, Diedler J, Juttler E, Steiner T, Zweckberger K, Hacke W, Unterberg A. Intensive care management of acute stroke: surgical treatment. Int J Stroke. 2010. 170-177.

23. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N. Clinical outcome following surgical treatment for bilateral cerebellar infarction. Acta Neurol Scand. 2011. 123: 345-351.

24. Neugebauer H, Witsch J, Zweckberger K, Juttler E. Space occupying cerebellar infarction: complications, treatment and outcome. Neurosurg Focus. 2013. 34 (5): E8.

25. Cuneo RA, Caronna JJ, Pitts L, Townsend J, Winestock DP. Upward transtentorial herniation: seven cases and a literature review. Arch Neurol. 1979. 36: 618 – 623.

26. Dinsdale HB. Spontaneous haemorrhage in the posterior fossa. A study of primary cerebellar and pontine haemorrhages with observations on their pathogenesis. Arch Neurol. 1964. 10: 200 – 217.

27. Witsch J, Neugebauer H, Zweckberger K, Juttler. Primary cerebellar haemorrhage: complications, treatment and outcome. Clin Neurol Neurosurg. 2013. 115: 863 – 869.

28. Kobayashi S, Sato A, Kageyama Y, Nakamura H, Watanabe Y, Yamaura A. Treatment of hypertensive cerebellar haemorrhage – surgical or conservative management? Neurosurgery. 1994. 34: 246-250.

29. Kirollos RW, Tyagi AK, Ross SA, van Hille PT, Marks PV. Management of spontaneous cerebellar haematomas: a prospective treatment protocol. Neurosurgery. 2001. 49: 1378 – 1386.

30. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N. Prognostic factors and long term outcome following surgical treatment of 76 patients with spontaneous cerebellar haematoma. Acta Neurochir (Wien). 2012. 154: 1189-1195.

31. Arnone GD, esfahani DR, Wonais M, Kumar P, Scheer JK, Alaraj A, Amin-Hanjani S, Charbel FT, Mehta AI. Surgery for cerebellar haemorrhage: A National Surgical Quality Improvement Program database analysis of patient outcomes and factors associated with 30 day mortality and prolonged ventilation. World Neurosurg. 2017. 106: 543-550.

32. Klingshim H, Grill E, Bender A, Strobl R, Mittrach R, Braitmeyer K, Muller M. Quality of evidence of rehabilitation interventions in long term care for people with severe disorders of sconsciousness after brain injury: A systemic review. Journal of Rehabilitative Medicine. 2017. 47: 577 – 585.

33. Lee L, Lo YT, See AAQ, Hsieh PJ, James ML, King NKK. Long-term recovery profile of patients with severe disability or in vegetative states following severe primary intracerebral haemorrhage. J Crit Care. 2018. 48: 269-275.

34. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N. Surgical treatment of patients with unilateral cerebellar infarcts: clinical outcome and prognostic factors. Acta Neurochir. 2011. 153: 2075-2083.

35. Tewari MK, Tripathi M, Sharma RP, Mishra GP, Lad SD. Surgical management of moderate sized spontaneous cerebellar haematoma: role of intracranial pressure monitoring. Turk Neurosurg. 2015. 25 (5): 712 – 720.

Figures and Tables:

	Total	Haemorrhage	Infarct	p-value
Sex				0.2409
Male (n)	88	50	38	
Female (n)	38	26	12	
Age (years)	20-83 (58.5 ± 13)	20-83 (59.3 ±13.7)	28-79 (57.3 ± 12.0)	
Pre-op GCS				**0.0008**
≤ 8 (n)	54	42	12	
> 8 (n)	72	34	38	
Charlson Comorbidity Index	2 (0-8)	2 (0-8)	2 (0-7)	
Time to surgery (hours)	0.33-336 (27.2 ± 51)	0.33-336 (13.9 ± 45)	1-333 (47.3 ±55.1)	
Presence of intraventricular haemorrhage				**0.0001**
Yes (n)	52	50	2	
No (n)	74	26	48	
Shunt dependence				0.6449
Yes (n)	22	15	8	
No (n)	104	61	42	
Tracheostomy				**0.0245**
Yes (n)	35	27	8	
No (n)	91	49	42	
Disposition to rehab				0.6652
Yes (n)	49	28	23	
No (n)	31	17	17	
ICU length of stay (days)	1-69 (8.9 ± 8.0)	2-45 (9.4 ± 7.0)	1-69 (8.2 ± 9.5)	
Hospital length of stay (days)	2-180 (38.2 ± 36.3)	2-158 (37.0 ± 32.6)	3-180 (40.1 ± 41.5)	
Inpatient mortality (n)	37	27	10	0.0734
Mortality at 6 months (n)	39	28	11	0.1084
Follow-up length (months)	6-144 (42.2 ± 33.8)	7-120 (40.4 ± 34.1)	6-144 (44.5 ± 33.9)	

Table 1: Patient demographics.

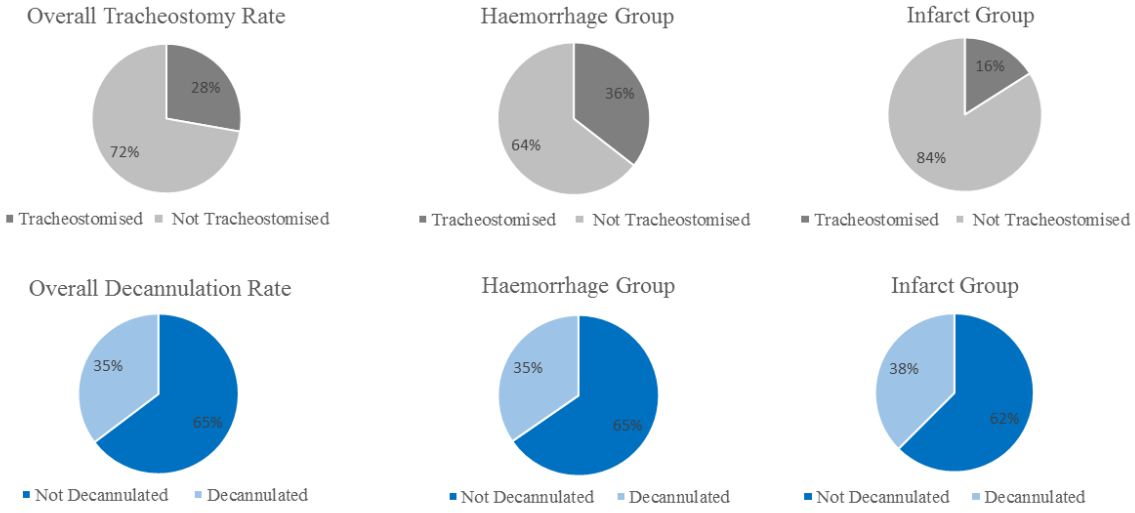

Figure 1: Tracheostomy rates and decannulation rates in our study population. There was significantly more patients in the haemorrhage group requiring a tracheostomy compared to the infarct group (p=0.0245). The overall rates of decannulation were roughly similar in both the haemorrhage and infarct groups.

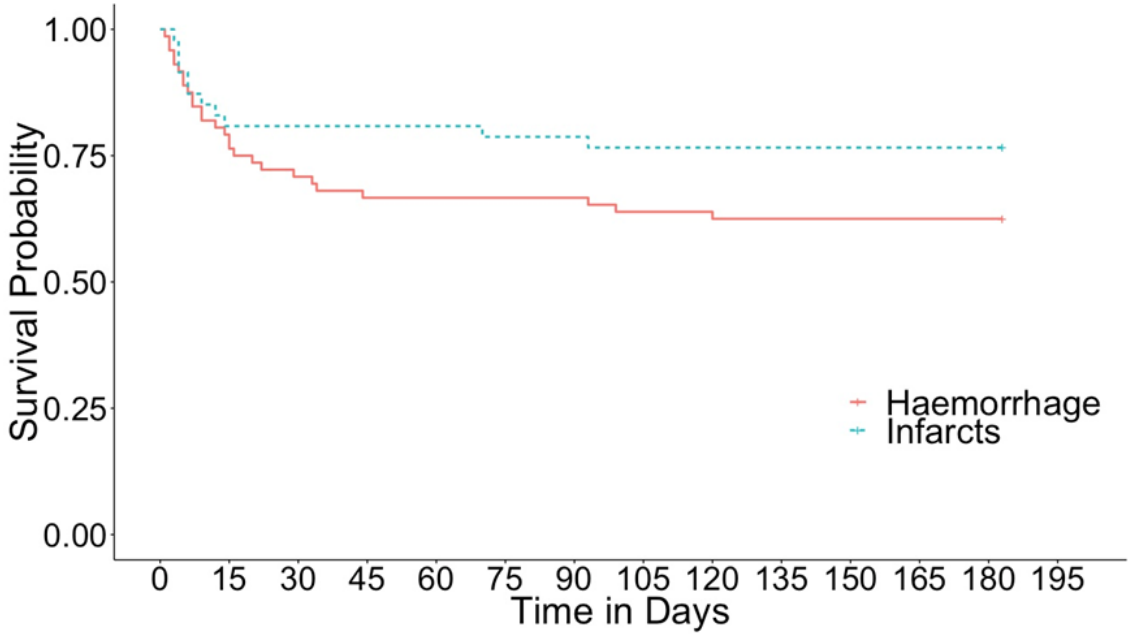

Figure 2. Kaplan Meier curve showing overall mortality between the haemorrhage and infarct groups at 6 months. Most of the patient deaths occurred within 30 days. There were a proportionally larger number of deaths occurring in the haemorrhage group, but this was not statistically significant ($p = 0.1084$).

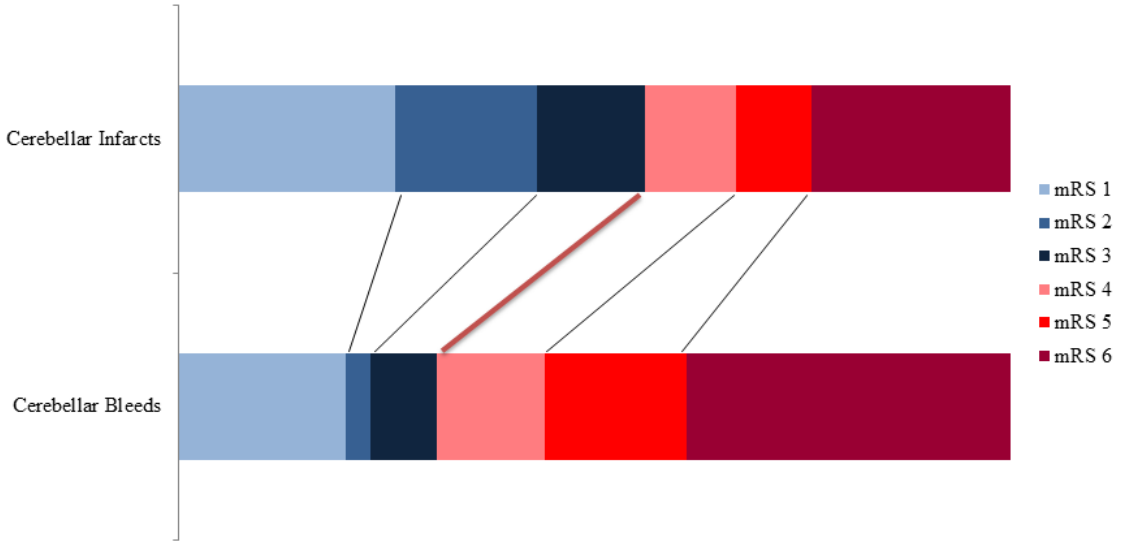

Figure 3: Difference in mRS between cerebellar haemorrhages and infarcts at latest follow-up after 6 months. Patients in the haemorrhage group had poorer functional outcome (mRS 4-6) compared to patients in the infarct group ($p = 0.0074$, OR 3.04).

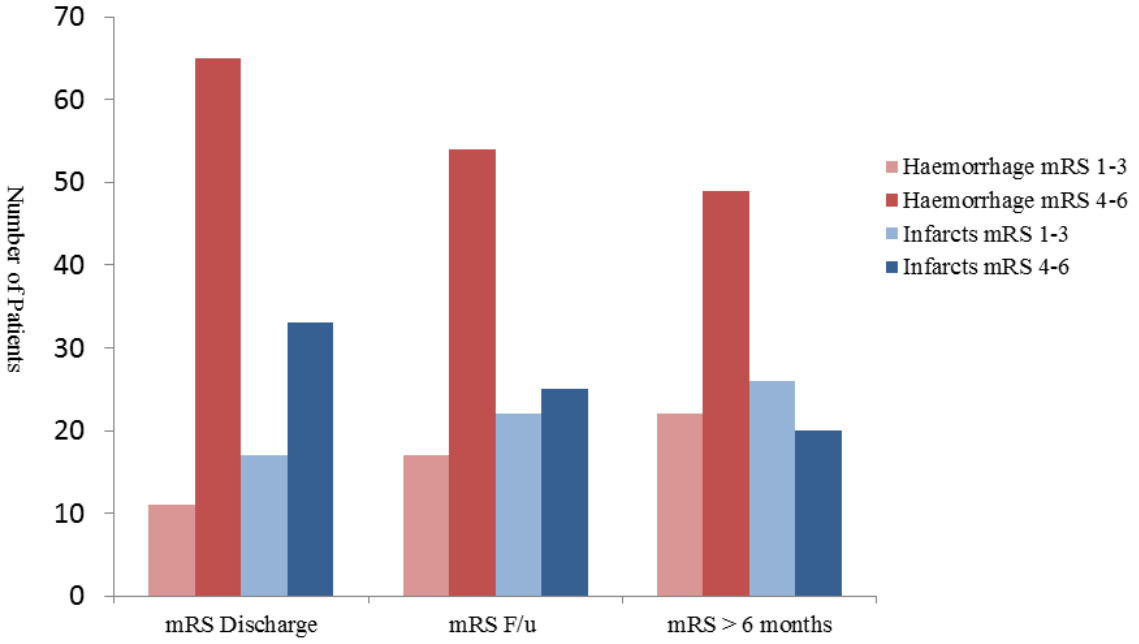

Figure 4: Trajectory of recovery as demonstrated by change in mRS over time – at discharge, at follow-up (between discharge and 6 months) and after 6 months. 69.2% of patients' overall mRS improved over time. More patients changed from poor outcome (mRS 4-6) to good outcome (mRS 1-3) over time.

AUTHORS CONTRIBUTION SECTION

1. Lester Lee: Conceptualisation, methodology, formal analysis, investigation, writing - original draft, writing - review and editing.
2. Daniel Loh: Formal analysis, investigation.
3. Nicolas Kon Kum King: Conceptualisaion, writing – review and editing, supervision.

LIST OF ABBREVIATIONS

mRS – Modified Rankin's Score

GCS – Glasgow Coma Scale

ICU – Intensive Care Unit

CCI - Charleston Comorbidity Index

AHA/ASA – American Heart Association/ American Stroke Association

DISCLOSURE-CONFLICT OF INTEREST

Dear Editor,

I would like to declare that neither my coauthors nor I have any financial sources to disclose for this study as it is a retrospective study conducted with the data from our institution and there are no conflicts of interest to disclose. In the conduct of this study, I would like to state that my coauthors and I have adhered to all ethical considerations. Our study has not been previously published in whole or in part or submitted elsewhere for review.

Yours sincerely,

Dr Lester Lee