ORIGINAL ARTICLE

Ventriculosubgaleal shunt in the management of obstructive hydrocephalus caused by cerebellar infarction

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Abstract *Introduction:* Cerebellar infarction is relatively uncommon. Small infarctions only cause cerebellar manifestations e.g. ataxia and nystagmus and are treated medically. Large cerebellar infarctions, however, can be life threatening. It cause brain stem compression and can obstruct the cerebrospinal fluid pathway causing obstructive hydrocephalus. It has to be treated promptly and may require besides the medical treatment surgical intervention as well. This is mainly in the form of posterior fossa decompression. In this study, we studied the beneficial effect of inserting a temporary ventriculoperitoneal shunt to relieve the supratentorial hydrocephalus in addition to posterior fossa decompression on the morbidity and mortality of patients in comparison to posterior fossa decompression alone.

Aim of the work: The aim of this study was to evaluate the role of ventriculosubgaleal shunt in cerebellar infarction causing supratentorial ventricular dilatation.

Methods: This was a retrospective study that included ten patients having extensive cerebellar infarction causing spratentorial hydrocephalus. They were divided into two groups, group (1) was submitted to posterior fossa decompression alone and group (2) was submitted to posterior fossa decompression in addition to temporary ventriculosubgaleal shunt insertion.

Results: Group (2) which had posterior fossa decompression in addition to temporary.
ventriculosubgaleal shunt had much better results than group (1) which had only posterior fossa decompression. Group (2) had a lower morbidity and mortality and a shorter hospital stay than group (1).

Conclusion: Temporary insertion of ventriculosubgaleal shunt is recommended in patients having extensive cerebellar infarction causing supratentorial hydrocephalus in addition to posterior fossa decompression. It results in a lower morbidity and mortality and a shorter hospital stay.

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1. Introduction

The cerebellum is relatively an uncommon site of brain infarction.[1,2] It is mainly caused by vascular occlusion but also may be traumatic. Small infarctions usually cause only cerebellar manifestations without obstructive symptoms.[3–5] Large cerebellar infarctions, however, can have devastating effects. In addition to cerebellar manifestations, the edema caused by

cerebellar infarction can cause brain stem compression, resulting in various brain stem manifestations, including deteriorating level of consciousness, respiratory complications as well as cranial nerve manifestations.[6–8] Fourth ventricular compression is also a complication of extensive cerebellar infarction. It can result in obstructive hydrocephalus causing increased intracranial pressure with its sequelae.[9–11]

The standard intervention of extensive cerebellar infarction causing brain stem compression is posterior fossa decompression in addition to the usual medical treatment.[12,13] However, in many cases, posterior fossa decompression alone is not enough to relieve supratentorial ventricular dilatation. For this reason, a temporary ventriculosubgaleal shunt is inserted at the same session to relieve pressure. The usual procedure used in different institutions to relieve pressure caused by supratentorial hydrocephalus is the use of external ventricular drainage to relieve and to measure the intracranial pressure. However, this usually has a high cost and can be complicated by intracranial infection.[14–16] For this reason, we investigated the efficacy of temporary ventriculosubgaleal shunt as a cheap and effective way to decrease intracranial pressure caused by hydrocephalus in patients with large cerebellar infarctions.

2. Aim of the work

The aim of this study was to evaluate the role of ventriculosubgaleal shunt in cerebellar infarction causing supratentorial ventricular dilatation.

3. Patients and methods

This is a retrospective study where data were collected from the archives of patients. Ten patients presenting with extensive cerebellar infarction causing obstructive hydrocephalus were included in the study. Any age and both sexes were included.

Preoperatively, patients were submitted to the following procedures:

- Complete history taking.
- Complete general and neurological examination.
- Investigations in the form of:
    Laboratory investigations.
    Radiological investigations in the form of computerized tomography scanning of the brain and magnetic resonance imaging for selected patients.
Patients were divided into two groups:

Group (1): had only posterior fossa decompression done (5 patients).
Group (2): had both posterior fossa decompression as well as ventriculosubgaleal shunt done (5 patients).
The posterior fossa decompression was in the form of bony decompression as well as expansive duroplasty.

Postoperatively, patients were submitted to the following:

Complete general and neurological examination. Radiological investigations in the form of computerized scanning of the brain.
Patients who underwent ventriculosubgaleal shunt had this shunt removed after two weeks of the operation.

All patients had clinical follow-up for six months. Computerized scanning of the brain was done in the follow-up period when required.

The confidentiality of the patients was kept and ethical concerns were met.

4. Statistical analysis

Data were presented as numbers and percentages.

5. Results

The percentages of patients were highest in the young age group between 15 to less than 30 years (60%) and decreased progressively with increasing age as shown in Table 1.

Males were more common (70%) than females (30%) as shown in Table 2.

Vascular occlusion was present in 60% of cases, while traumatic infarction was present in 40% of cases as shown in Table 3.

On admission Glasgow coma scale of 3–9 represented 50% of cases which was the most common presentation as shown in Table 4.

On admission Glasgow coma scale of 3–9 represented 60% of cases in group (1) patients which was the most common presentation as shown in Table 5.

On admission Glasgow coma scale of 3–9 as well as from 10 to 12 represented 40% each of group (2) patients as shown in Tables 6 and 7.

Preoperative MRI of the brain was used for earlier detection of cerebellar infarction in some of the cases (Fig. 1). Preoperative CT scan of the brain showed hydrocephalus with radiological manifestations of increased intracranial pressure (Figs. 2 and 3). These radiological manifestations included increased Evans' index to 0.3 or greater and temporal horn dilatation in all cases of both groups. Periventricular edema around the frontal horn occurred in 60% of patients in group (1) and in 40% of patients in group (2).

Postoperative CT scan of the brain was done for all cases to detect the adequacy of posterior fossa decompression (Fig. 4) and the placement of the tip of the ventriculosubgaleal shunt in the frontal horn of the lateral ventricle as well as the adequacy of supratentorial ventricular drainage (Fig. 5).

One month postoperatively, 40% of group (1) patients had GCS of 13–15, while 20% of patients had GCS of 3–9 as shown in Table 8. Two patients in this group died within one month of surgery.

One month postoperatively, 80% of group (2) patients had GCS of 13–15, while 20% of patients had GCS of 10–12. None of the patients had GCS of 3–9 as shown in Table 9.

Table 1 Distribution of patients according to age.

Age group	Number of patients	(%)
15 to less than 30 years	6	60
30 to less than 45 years	3	30
45 years and more	1	10

Table 2 Distribution of patients according to sex.

Sex	Number of patients	(%)
Males	7	70
Females	3	30

Table 3 Etiology of cerebellar infarction.

Etiology of cerebellar infarction	Number of patients	(%)
Vascular occlusion	6	60
Trauma	4	40

Table 4 Glasgow comma scale on admission.

Glasgow comma scale	Number	(%)
3–9	5	50
10–12	3	30
13–15	2	20

Table 5 Preoperative GCS in group (1) patients who had only posterior fossa decompression.

Glasgow comma scale	Number	(%)
3–9	3	60
10–12	1	20
13–15	1	20
Total	5	

Table 6 Preoperative GCS in group (2) patients who had posterior fossa decompression as well as ventriculosubgaleal shunt.

Glasgow comma scale	Number	(%)
3–9	2	40
10–12	2	40
13–15	1	20
Total	5	

Table 7 Preoperative radiological manifestations of increased intracranial pressure caused by supratentorial hydrocephalus on CT scan of the brain.

Radiological manifestation	Group (1)	(%)	Group (2)	(%)
Evans' index (0.3 or greater)	5	100	5	100
Periventricular edema	3	60	2	40
Temporal horn dilatation	5	100	5	100

Postoperative complications included meningitis in one patient and wound infection in two patients as shown in Table 10.

The number of postoperative days till recovery of the conscious state decreased significantly in patients who had
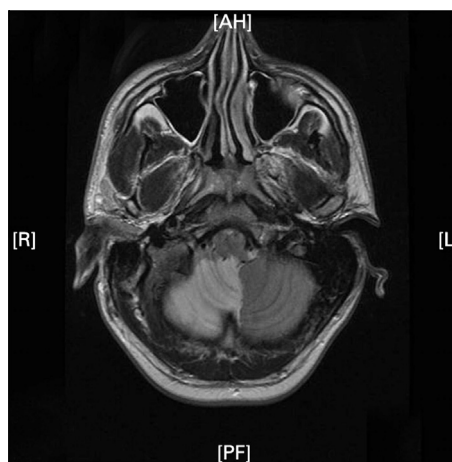
**Figure 1** Axial T2-weighted MRI of the brain showing right cerebellar infarction.

ventriculosubgaleal shunt in addition to posterior fossa decompression as shown in Table 11.

6. Discussion

The posterior fossa is a tight space that harbors the cerebellum and the brain stem. Different etiologies can cause cerebellar swelling including tumors, hemorrhage and infarctions.[17–19] Cerebellar infarctions often result from vascular occlusion but can also result from trauma. Small cerebellar infarctions often only cause cerebellar dysfunction manifestations without compressive manifestations.[20–23] However, large cerebellar infarctions can cause compressive manifestations in the form of brain stem compression and compression of the fourth ventricle. The later can cause obstructive supratentorial hydrocephalus.[24–26]

Medical treatment can be used for treatment of small cerebellar infarction. However, for large cerebellar infarction with compressive manifestations, surgical decompression is usually needed to relieve the compressive symptoms.[27–29] Relieve of supratentorial hydrocephalus was not done in all cases and that caused increased morbidity and mortality. Therefore, insertion of temporary ventriculosubgaleal shunt during the initial procedure was advocated to relieve the supratentorial hydrocephalus and thus expedes the recovery.[30–32]

This study included 10 patients who had extensive cerebellar infarction from different etiologies that resulted in brain stem compression, fourth ventricular obstruction and supratentorial hydrocephalus. Five patients were treated with only posterior fossa decompression that included bony decompression as well as expansive duroplasty. The other five patients had posterior fossa bony decompression, expansive duroplasty as well as ventriculosubgaleal shunt using a catheter at the time of the operation that was removed two weeks postoperatively. Not all the cases were drained because this was dependent on the surgeons' preference and many neurosurgeons think that whatever the degree of supratentorial hydrocephalus, it would be resolved by the mere posterior fossa decompression without the need for supratentorial CSF drainage. Actually there was not a selection bias in this study because these 10 patients were all
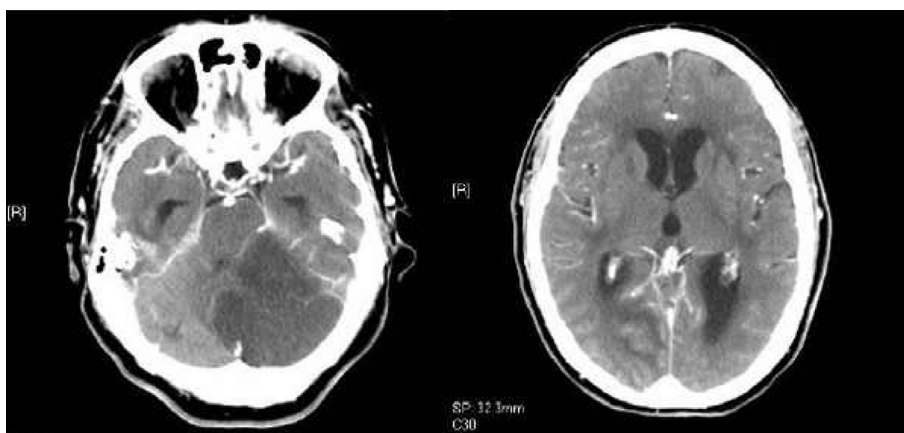
Figure 2 Axial CT scan of the brain showing extensive left cerebellar infarction (on the left side) and supratentorial ventricular dilatation (on the right side).

Figure 3 Axial CT scan of the brain showing left cerebellar infarction.

Figure 4 Postoperative CT scan of the brain showing posterior fossa decompression for left cerebellar infarction.

Figure 5 Postoperative CT of the brain showing the tip of ventriculosubgaleal shunt in the right frontal horn.

Table 8 Postoperative GCS in group (1) patients who had only posterior fossa decompression (one month postoperatively).

Glasgow coma scale	Number	(%)
3–9	1	20
10–12	0	0
13–15	2	40
Total	3	

Table 9 Postoperative GCS in group (2) patients who had posterior fossa decompression as well as ventriculosubgaleal shunt (one month postoperatively).

Glasgow coma scale	Number	%
3–9	0	0
10–12	1	20
13–15	4	80
Total	5	

the patients that could be found in the hospital records who had large cerebellar infarction with supratentorial hydrocephalus.

Table 10 Postoperative complications.

Type of complication	Number of patients ($n = 3$)
Meningitis	1
Wound infection	2
Total	3

Table 11 Number of postoperative days till full recovery of the conscious state.

Type of intervention	Average number of days	Range
Only posterior fossa decompression	12	3–21
Posterior fossa decompression as well as ventriculosubgaleal shunt	3	1–9

The age of the patients ranged from 18 to 46 years and the most common age group was from 15 years to less than 30 years (60%) (Table 1). The younger age preponderance could be explained by the larger brain volume and thus tighter posterior fossa in younger age group and thus more compressive manifestations. In the younger age group, the cerebellar tissue is occupying the whole tight posterior fossa space and any cerebellar swelling would easily obstruct the CSF pathways causing hydrocephalus, in contrast to the more capacious subarachnoid space around the cerebellum in older age group that can accommodate cerebellar infarction without causing obstructive hydrocephalus which was a major criterion to admit patients in our study. In addition, the large subarachnoid space due to brain atrophy in older age group usually makes supratentorial ventricular dilatation clinically insignificant. Forty percentage of cases were traumatic which usually occurs in younger age group. These cases were obtained from patients' records without selection bias and these were the patients' age found, probably because of the more preponderance of younger age group in our country. This age range in our study was different from other studies.[33–35]

Seventy percentage of cases were males and 30% were females, which could be explained by the post-traumatic cases who were only males, thus increased the percentage of males in the study (Table 2). Vascular occlusion was the etiologic factor in 60% of cases, while trauma (edematous contusions with mass effect) was the etiology in 40% of cases; all of them were males (Table 3). As regards to the Glasgow Coma Scale at the time of admission, 50% of cases had GCS of 3–9, 30% from 10 to 12 and 20% from 13 to 15 (Table 4). There was no significant difference in the preoperative GCS between group (1) and group (2) patients (Tables 5 and 6).

Preoperative radiological manifestations of increased intracranial pressure caused by supratentorial hydrocephalus on CT scan of the brain were almost identical in both groups. These radiological manifestations included increased Evans' index to 0.3 or greater and temporal horn dilatation in all cases of both groups. Periventricular edema around the frontal horn occurred in 60% of patients in group (1) and in 40% of patients in group (2) (Table 7).

Postoperatively, there was a significant difference in the GCS between the two groups. Only 40% of patients in group (1) had GCS of 13–15 one month postoperatively as compared to 80% of patients in group (2) (Tables 8 and 9). Forty

percentage of patients who underwent only posterior fossa decompression had their supratentorial hydrocephalus completely resolved immediately after surgery, while all cases that had ventriculosubgaleal shunt done had their supratentorial hydrocephalus resolved immediately postoperatively. One month after surgery, the cases that had only posterior fossa decompression had 60% survival rate (three out of five patients), while all cases that also had ventriculosubgaleal shunt done were alive. Two patients died within one month of surgery. They had manifestations of transtentorial herniation, did not respond to dehydrating measures and later had brain stem infarction on postoperative CT scan of the brain. Postoperative morbidity occurred in 3 patients; one of them had meningitis. This patient had ventriculosubgaleal shunt and resolved after a course of antibiotic. Two other patients had superficial wound infection, one patient from each group responded well to medical treatment (Table 10). These results agreed with other authors' results.[36–38] In addition, cases that had ventriculosubgaleal shunt in addition to posterior fossa decompression had a shorter period needed for regaining full consciousness (average 3 days) as compared to an average of 12 days in the group that had only posterior fossa decompression done (Table 11). This was similar to results of other authors.[39–43]

7. Conclusion and recommendations

Because of the small numbers in our patient series and the clinical bias in the distinction of the patient groups, no definite conclusions can be drawn from this retrospective analysis. However, our experience confirms other reports from larger series.[14–16] We recommend that patients with large cerebellar infarction causing mass effect and supratentorial hydrocephalus with radiological signs of increased intracranial pressure be treated with a surgical procedure, combining posterior fossa decompression and expansive duroplasty and a temporary CSF drainage using a ventriculosubgaleal shunt to expedie recovery and to have a lower mortality rate.

References

1. Chaves CJ, Caplan LR, Chung CS, Tapia J, Amarenco P, Teal P, et al. Cerebellar infarcts in the New England medical center posterior circulation stroke registry. *Neurology* 1994;**44**:1385–90.
2. Adams Jr HP, Kappelle LJ, Biller J, Gordon DL, Love BB, Gomez F, et al. Ischemic stroke in young adults: experience in 329 patients enrolled in the Iowa registry of stroke in young adults. *Arch Neurol* 1995;**52**:491–5.
3. Barinagarrementeria F, Figueroa T, Huebe J, Cantú C. Cerebral infarction in people under 40 years: etiologic analysis of 300 cases prospectively evaluated. *J Stroke Cerebrovasc Dis* 1996;**6**:75–9.
4. Amarenco P, Caplan LR. Vertebrobasilar occlusive disease: review of selected aspects, III: mechanisms of cerebellar infarctions. *J Stroke Cerebrovasc Dis* 1993;**3**:66–73.
5. Barinagarrementeria F, Cantú Brito C, De La Peña A, Izaguirre R. Prothrombotic states in young people with idiopathic stroke: a prospective study. *J Stroke Cerebrovasc Dis* 1994;**25**:287–90.
6. Caplan LR. Migraine and posterior circulation stroke. In: Caplan LR, editor. *Posterior circulation diseases: clinical findings, diagnosis and management*. Cambridge, Mass: Blackwell Science; 1996. p. 544–68.
7. Jensen MB, St Louis EK. Management of acute cerebellar stroke. *Arch Neurol* 2005;**62**:537–44.

8. Koh MG, Phan TG, Atkinson JL, Wijdicks EF. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *J Stroke Cerebrovasc Dis* 2000;**31**:2062–7.

9. Adams Jr HP, Del ZG, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke. *Circulation* 2007;**115**: e478–534.

10. European Stroke Organisation (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis* 2008;**25**:457–507.

11. Hornig CR, Rust DS, Busse O, Jauss M, Laun A. Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke* 1994;**25**:372–4.

12. 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. *Eur J Neurol* 1999;**246**:257–64.

13. 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**:259–62.

14. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D. Neurosurgical management of cerebellar haematoma and infarct. *J Neurol Neurosurg Psychiatry* 1995;**59**:287–92.

15. Raco A, Caroli E, Isidori A, Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery* 2003;**53**:1061–5.

16. Rieke K, Schwab S, Krieger D, von KR, Aschoff A, Schuchardt V, et al. Decompressive surgery in space-occupying hemispheric infarction: results of an open, prospective trial. *Crit Care Med* 1995;**23**:1576–87.

17. Newcommon NJ, Green TL, Haley E, Cooke T, Hill MD. Improving the assessment of outcomes in stroke: use of a structured interview to assign grades on the modified Rankin Scale. *J Stroke Cerebrovasc Dis* 2003;**34**:377–8.

18. Anderson C, Laubscher S, Burns R. Validation of the short form 36 (SF-36) health survey questionnaire among stroke patients. *J Stroke Cerebrovasc Dis* 1996;**27**:1812–6.

19. Dorman PJ, Dennis M, Sandercock P. How do scores on the EuroQol relate to scores on the SF-36 after stroke? *J Stroke Cerebrovasc Dis* 1999;**30**:2146–51.

20. Kurth BM, Ellert U. The SF-36 questionnaire and its usefulness in population studies: results of the German Health Interview and Examination Survey 1998. *Soz Praventivmed* 2002;**47**: 266–77.

21. Skoglund TS, Eriksson-Ritzen C, Sorbo A, Jensen C, Rydenhag B. Health status and life satisfaction after decompressive craniectomy for malignant middle cerebral artery infarction. *Acta Neurol Scand* 2008;**117**:305–10.

22. Hofmeijer J, Amelink GJ, Algra A, van GJ, Macleod MR, Kappelle LJ, et al. Hemicraniectomy after middle cerebral artery infarction with life-threatening Edema trial (HAMLET). Protocol for a randomised controlled trial of decompressive surgery in space-occupying hemispheric infarction. *Trials* 2006;**7**:29.

23. Juttler 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. *J Stroke Cerebrovasc Dis* 2007;**38**: 2518–25.

24. Vahedi K, Vicaut E, Mateo J, Kurtz A, et al. Sequential-design, multicenter, randomized, controlled trial of early decompressive craniectomy in malignant middle cerebral artery infarction (DECIMAL Trial). *J Stroke Cerebrovasc Dis* 2007;**38**:2506–17.

25. Baldauf J, Oertel J, Gaab MR, Schroeder HW. Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. *Neurosurgery* 2006;**59**:539–44.

26. Uhl E, Kreth FW, Elias B, Goldammer A, et al. Outcome and prognostic factors of hemicraniectomy for space occupying cerebral infarction. *J Neurol Neurosurg Psychiatry* 2004;**75**:270–4.

27. Hornig CR, Rust DS, Busse O, Jauss M, Laun A. Space-occupying cerebellar infarction: clinical course and prognosis. *J Stroke Cerebrovasc Dis* 1994;**25**:372–4.

28. Baldauf J, Oertel J, Gaab MR, Schroeder HWS. Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. *Neurosurgery* 2006;**59**:539–44.

29. Kase CS, Norrving B, Levine SR, Viken LB, Eliot HC, Wolf PA, et al. Cerebellar infarction. Clinical and anatomic observation in 66 cases. *J Stroke Cerebrovasc Dis* 1993;**24**:76–83.

30. Mohsenipour I, Gabl M, Schutzhard E, Twerdy K. Suboccipital decompressive surgery in cerebellar infarction. *Zentralbl Neurochir* 1999;**60**:68–73.

31. Jauss M, Krieger D, Hornig CR, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German–Austrian cerebellar infarction study. *Eur J Neurol* 1999;**246**:257–64.

32. Raco A, Caroli E, Isidori A, Vangelista T, Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery* 2003;**53**:1061–6.

33. Jauss M, Müffelmann B, Krieger D, Zeumer H, Busse O. A computed tomography score for assessment of mass effect in space-occupying cerebellar infarction. *J Neuroimaging* 2001;**11**: 268–71.

34. Schmitz-Hübsch T, du Montcel ST, Baliko L, et al. Scale for the assessment and rating of ataxia: development of a new clinical scale. *Neurology* 2006;**66**:1717–20.

35. Hacke W, Kaste M, Skyhoj Olsen T, Orgogozo JM, Bogousslavsky J. European stroke initiative (EUSI) recommendations for stroke management. The European stroke initiative writing committee. *Eur J Neurol* 2000;**7**:607–23.

36. European Stroke Initiative Executive Committee; EUSI Writing Committee, Olsen TS, Langhorne P, Diener HC, Hennerici M, Ferro J, Sivenius J, et al. European stroke initiative recommendations for stroke management-update 2003. *Cerebrovasc Dis* 2003;**16**:311–37.

37. Jones HR, Millikan CH, Sandok BA. Temporal profile (clinical course) of acute vertebrobasilar system cerebral infarction. *J Stroke Cerebrovasc Dis* 1980;**11**:173–7.

38. Langmayr JJ, Buchberger W, Reindl H. Cerebellar hemorrhage and cerebellar infarct: retrospective study of 125 cases. *Wien Med Wochenschrift* 1993;**143**:131–3.

39. Kanis KB, Ropper AH, Adelman LS. Homolateral hemiparesis as an early sign of cerebellar mass effect. *Neurology* 1994;**44**: 2194–7.

40. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D. Neurosurgical management of cerebellar haematoma and infarct. *J Neurol Neurosurg Psychiatry* 1995;**59**:287–92.

41. Koh MG, Phan TG, Atkinson JLD, Wijdicks EFM. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *J Stroke Cerebrovasc Dis* 2000;**31**:2062–7.

42. Kase CS, Wolf PA. Cerebellar infarction: upward transtentorial herniation after ventriculostomy. *J Stroke Cerebrovasc Dis* 1993;**24**:1096–8.

43. Tulyapronchote R, Malkoff MD, Selhorst JB, Gomez CR. Treatment of cerebellar infarction by decompression suboccipital craniectomy. *J Stroke Cerebrovasc Dis* 1993;**24**:478–80.