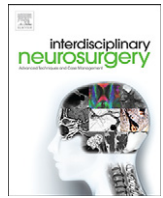




Contents lists available at ScienceDirect

Interdisciplinary Neurosurgery: Advanced Techniques and Case Management

journal homepage: www.inat-journal.com

Case Report & Case Series (CRP)

Surgical management of spontaneous hypertensive brainstem hemorrhage



Bal Krishna Shrestha, Lu Ma, Zhi-gang Lan, Hao Li, Chao You *

Department of Neurosurgery, West China Hospital, Sichuan University, 37 GuoXue Xiang Street, Chengdu 610041, P. R. China

ARTICLE INFO

Article history:

Received 8 March 2015

Revised 24 June 2015

Accepted 28 June 2015

Keywords:

Brainstem hemorrhage

Hypertension

Surgery

ABSTRACT

Spontaneous hypertensive brainstem hemorrhage is the spontaneous brainstem hemorrhage associated with long term hypertension but not having definite focal or objective lesion. It is a catastrophic event which has a poor prognosis and usually managed conservatively. It is not uncommon, especially in eastern Asian populations, accounting approximately for 10% of the intracerebral hemorrhage. Before the advent of computed tomography, the diagnosis of brainstem hemorrhage was usually based on the clinical picture or by autopsy and believed to be untreatable via surgery. The introduction of computed tomography permitted to categorize the subtypes of brainstem hemorrhage with more predicted outcome. Continuous ongoing developments in the stereotactic surgery and microsurgery have added more specific surgical management in these patients. However, whether to manage conservatively or promptly with surgical evacuation of hematoma is still a controversy. Studies have shown that an accurate prognostic assessment based on clinical and radiological features on admission is critical for establishing a reasonable therapeutic approach. Some authors have advocate conservative management, whereas others have suggested the efficacy of surgical treatment in brainstem hemorrhage. With the widening knowledge in microsurgical techniques as well as neuroimaging technology, there seems to have more optimistic hope of surgical management of spontaneous hypertensive brainstem hemorrhage for better prognosis. Here we present five cases of severe spontaneous hypertensive brainstem hemorrhage patients who had undergone surgery; and explore the possibilities of surgical management in patients with the spontaneous hypertensive brainstem hemorrhage.

© 2015 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/>).

1. Introduction

Spontaneous hypertensive brainstem hemorrhage is specifically associated with long term hypertension but not having definite focal or objective lesion. It is a catastrophic event which has a poor prognosis and usually managed conservatively [1–6]. It is not uncommon, especially in eastern Asian populations, accounting approximately for 10% of the intracerebral hemorrhage (ICH) [6,7]. It may occur in pons, pontomedullary junction, pontomesencephalic junction, midbrain, and medulla. Most of the hematomas in brainstem originate from the branches of the basilar artery supplying the pons. Midbrain and medulla oblongata are practically less common sites of origin of brainstem hemorrhage [1,8]. Before the advent of computerized tomography (CT), the diagnosis of brainstem hemorrhage was usually made based on the clinical picture or by autopsy, and believed to be untreatable via surgery [1,2]. The introduction of computed tomography (CT) permitted to categorize the subtypes of brainstem hemorrhage with more predicted outcome [6,9,10]. Continuous ongoing developments in the stereotactic surgery and microsurgery have added more specific surgical management in these patients [3,10,11]. However, whether to manage conservatively or promptly with

surgical evacuation of hematoma is still a controversy. Studies have shown that an accurate prognostic assessment based on clinical and radiological features on admission is critical for establishing a reasonable therapeutic approach [1–9,12]. Some authors have advocate conservative management [2,4], whereas others have suggested the efficacy of surgical treatment in brainstem hemorrhage [3,10,11]. With the widening knowledge in microsurgical techniques as well as neuroimaging technology, there seems to have more optimistic hope of surgical management of spontaneous hypertensive brainstem hemorrhage for better prognosis. However, the efficacy of surgical management is still debatable. Here we present five cases of severe spontaneous hypertensive brainstem hemorrhage patients who had undergone surgery; and explore the possibilities of surgical management in such patients as well as share our achievements in managing these patients.

2. Methods

We reviewed five patients (admitted in our hospital from January 2010 to April 2014) who had spontaneous hypertensive brainstem hemorrhage and had undergone surgical removal of hematoma with the approval of IRB of the University. Our study is not a clinical trial and there were no comparators. Brainstem hemorrhage with the pathological diagnosis of cavernous angioma, arteriovenous malformation,

* Corresponding author. Tel./fax: +86 28 85422490, +86 8280092897 (Mobile).
E-mail address: chaoyou_scu@163.com (C. You).

tumor apoplexy and aneurysm were excluded in the study. The surgical approaches were subtemporal tentorial approach, suboccipital midline approach and suboccipital retrosigmoid approach, which was individualized according to the sites of hematoma.

When the patient was discharged, Glasgow outcome score (GOS) were assessed just before discharge from hospital. GOS was also assessed at six month's follow up.

3. Results

In our five cases, there were two males and three females with ages ranging from 37 years to 70 years. All patients underwent prompt surgical removal of hematoma (Table 1). At 6-month follow up, two patients could perform all the daily tasks without any help; one patients was wheelchair bound and needed help to perform routine daily activities; and 2 patient remained bedridden needing total nursing care.

3.1. Case I

A 44-year-old man with known hypertensive history for ten years had sudden loss of consciousness while playing Majiang (a Chinese game) three hours ago. He had been using antihypertensive drugs on and off in the past eight years. On arrival to emergency ward the patient was comatose (GCS of 3) with unstable vitals (R 2–3/min, T 39.5 °C, BP 201/145 mm Hg) and sluggishly reactive pupil. Head CT scan revealed a large hematoma compromising the left midbrain and thalamus with extension to the lateral ventricles and third ventricle, causing acute obstructive hydrocephalus (Fig. 1A). The hematoma on the CT measured 2.2 × 1.5 cm and was visible on four slices of eight millimeter thickness. Emergency surgery was performed after getting the family's consent and the hematoma was evacuated via left subtemporal tentorial approach two hours after admission. His GCS improved and responded to stimuli the next day. His postoperative head CT scan showed complete removal of the hematoma with improved ventricular size (Fig. 1C). He was discharged to rehabilitation center one week after the surgery. The patient could walk and perform all daily activities with minimal help at 40 days after surgery (GOS 4).

3.2. Case II

A 37-year-old man with known hypertension for three years had sudden disturbances in consciousness accompanied with facial nerve palsy and vomiting 4 hours ago. On arrival to hospital, he was comatose (GCS of 5) and his vitals were severely disrupted (T: 39 °C, P: 56 beats/min, R: 4 times/min, BP: 219/139 mm Hg), with pupil pinpoint and unreactive. An emergency head CT scan showed a hematoma mainly located in the pontine level with some extension into the fourth ventricle (Supplemental Fig. 2A). The hematoma on the CT scan measured 2.1 × 1.3 cm and was visible on four slices of nine millimeter thickness. The patient underwent emergency surgery after having the consent from the patient's family and the hematoma was evacuated via suboccipital midline approach. His GCS improved and could respond to simple command 12 hours after the surgery. Postoperative head CT scan showed complete removal of the hematoma and slight edema around the operative site (Supplemental Fig. 2B). The patient was discharged home two weeks after surgery. He was able to perform all the daily tasks without any help at six-month follow-up (GOS 4).

Table 1

Cases of brainstem hemorrhage who underwent surgery.

Case no.	Age (years)	Gender	GCS (Admission)	Surgical approach	Tracheostomy	GOS (6 months after surgery)
1	44	M	3	Subtemporal tentorial	No	4
2	37	M	5	Suboccipital midline	Yes	4
3	70	F	4	Suboccipital retrosigmoid	yes	3
4	59	F	6	Subtemporal tentorial	Yes	2
5	42	F	7	Suboccipital midline	Yes	2

3.3. Case III

A 70-year-old woman, with a history of systemic hypertension for ten years, had suddenly collapsed and became unconscious while shopping in the local food store two hours prior. On arrival to the emergency department, she was in coma with a GCS of 4. Her vital signs were severely disrupted with a respiratory rate of 2–3/min and fluctuating BP between 199/102 mm Hg and 232/111 mm Hg. An emergency head CT scan revealed a large hematoma located in pons extending to medulla with fourth ventricle occlusion (Supplemental Fig. 3A). The hematoma on the CT measured 3.1 × 2.1 cm and was visible on three slices of eight millimeter thickness. Emergency surgery was performed after having the consent from the patient's family and the hematoma was removed through a right suboccipital retrosigmoid approach. Postoperative head CT scan showed the complete removal of the hematoma and reappearance of the fourth ventricle (Supplemental Fig. 3B). The patient was discharged to rehabilitation center two weeks after surgery. He was wheelchair bound and needed help for daily activities at six-month follow-up (GOS 3).

3.4. Case IV

A 59-year-old woman had sudden headache, vomiting and loss of consciousness for six hours. She was taken to the local hospital where head CT scan showed brainstem hemorrhage (Supplemental Fig. 4A). The hematoma on the CT scan measured 2.5 × 2.0 cm and was visible on three slices of nine millimeter thickness. For further management she was transferred to our hospital. On arrival, her vitals were as follows: T: 37 °C, P: 78 beats/min, R: 5 times/min, BP: 132/72 mm Hg; GCS of 6; and anisocoria (right pupil 3.5 mm and left 1.5 mm) with absent light reflex; neck stiffness; tetraplegia; flaccid tone and loss of physiological reflexes. The patient underwent emergency surgery via right subtemporal tentorial approach after having the family's consent for the surgery. Postoperative scan showed a complete removal of the hematoma (Supplemental Fig. 4B). The Patient remained bedridden and required total nursing care. The patient was transferred to the rehabilitation center after three months with the GOS of 2.

3.5. Case V

A 42-year-old woman had sudden headache, dizziness, nausea and vomiting for a day associated with quadriplegia, blurred vision and aphasia. She progressed to a comatose state six hours prior to admission. CT head was performed in the emergency department which showed brainstem hemorrhage (Supplemental Fig. 5A). The hematoma on the CT measured 2.6 × 2.0 cm and was visible on three slices of eight mm thickness. On arrival, her vitals were as follows: T: 37.8 °C, P: 108 beats/min, R: 5 times/min, BP: 188/106 mm Hg; GCS of 6; bilateral pupil was equal and reactive to light; bilateral muscle power was III/V; decreased superficial reflexes; and no pathological reflexes. The patient underwent emergency surgery via suboccipital midline approach after having the family's consent for the surgery. Postoperative scan showed complete removal of the hematoma and communicating fourth ventricle (Supplemental Fig. 5C). The patient remained bedridden and required total nursing care. She was referred to the rehabilitation center after 47 days of admission with the GOS of 2.

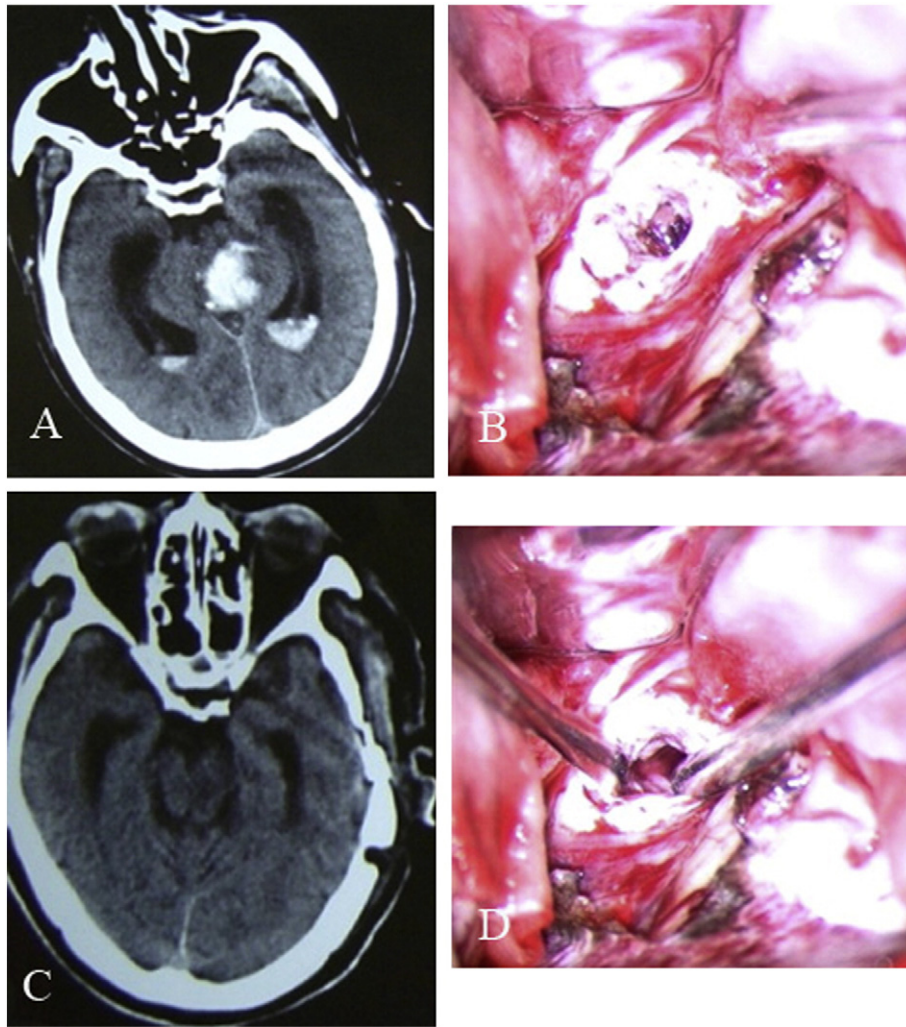


Fig. 1. Left basal–tegmental pontine hemorrhage with fourth ventricular extension. (A) CT head showing the hyperdense hematoma at the postero–basal–tegmental area of pons invading the fourth ventricle. (B) Intraoperative view of hematoma. (C) Post-operative CT head showing the resolution of the hematoma with encephalomalacic changes. (D) Intraoperative view after complete removal of hematoma.

4. Discussion

Severe spontaneous hypertensive brainstem hemorrhage is a catastrophic event and the prognosis is generally poor with conservative management while surgical management is still controversial. This brainstem hemorrhage usually dissects rather than destroys adjacent structures and may expand so rapidly that the life of the patient is acutely endangered [1]. A patient with the brainstem hemorrhage may rapidly develop coma, and show disturbance of vegetative function (disturbance of respiration, cardiac dysrhythmias, hyperthermia, hypertension), miosis and other neuroophthalmologic symptoms and flaccid tetraparesis. Further, blood may enter into the ventricle and obstruct the CSF pathway. In such severe patients, conservative management alone is not enough, hence, surgical removal of the hematoma in combination with establishment of extra ventricular drainage is another option. In our five cases, patients had severe brainstem compression with unstable vital signs (hypertension/hypotension, bradycardia, kussmal/apneustic respiration) and were rapidly deteriorating. Therefore, we discussed the pros and cons of each treatment method to make a final decision. Surgery was performed only after having the consent from patient's family for the surgery. Intraparenchymatous bleeding limited to the brainstem do not generally represent a primary indication for surgery [2]. Surgical removal of the hematoma is indicated only in isolated cases with massive and progressive symptoms [13]. Our indication for

emergency surgical evacuation of hematoma were as follows: i) brain stem hematoma volume > 5 ml (concentrated and space occupying); ii) GCS < 8 with progressive deterioration; iii) unstable vital signs especially those needing mechanical ventilation for disturbance of respiration (Kussmal or apneustic respiration with decreases SPO_2); iv) site of hematoma <1 cm from the brainstem cortex and/or rupture to the fourth ventricle; v) time of hemorrhage <24 h. However, prompt removal of hematoma is still contraindicated in those patients who show absence of all brainstem reflexes.

The location and size of the brainstem hematoma dictate the surgeon's operative approach for removal (Table 2). The complex neuro-anatomic structure of the brainstem and surrounding vasculature must be taken into consideration when planning the most optimal route to the hematoma. CT scan is fast and provides anatomical site and size of hematoma along with information of secondary insults like brainstem edema and obstructive hydrocephalus; and we can get portable as well, thus, it is the first choice for preoperative evaluation in these progressively deteriorating comatose patients with brainstem hemorrhage. MRI scan is relatively time consuming which should be done in comatose patients after they become stable as well as in non-comatose patients to rule out other causes such as cavernoma, arteriovenous malformation, and tumor. MRI findings such as "popcorn" or "mulberry" lesion, or the "flow voids" or "contrast enhanced lesion" suggest definite pathology and are very helpful in determining a better candidate for surgery. In

Table 2
Surgical approaches for brainstem hemorrhages in different locations.

Brainstem division	Location of hemorrhage	Surgical approach
Midbrain	Tectum	Occipital transtentorial approach
	Lateral surface	Subtemporal tentorial approach
Pons	Dorsal surface involving fourth ventricle	Suboccipital midline approach
	Anterolateral surface	Subtemporal tentorial approach
	Lateral of inferior anterolateral surface	Suboccipital retrosigmoid approach
Medulla oblongata	Dorsal involving fourth ventricle	Suboccipital midline approach
	Lateral or anterolateral	Suboccipital midline approach or far lateral approach

our five cases, surgical approaches were individualized, and the most optimal routes that would minimize the risk of injury to neurovascular structures were adopted. Hematoma reaching the lateral surface of the midbrain was managed by subtemporal tentorial approach. Suboccipital retrosigmoid approach was used for inferior anterolateral hematoma of the pons. And suboccipital midline approach was used for dorsal pontine and medullary hemorrhage involving the fourth ventricle.

Surgical removal of the brainstem hematoma was done under high-definition microscope. Usually local protrusion or color change at the site of hematoma could be noticed under the microscope. The longitudinal incision on the brainstem was made through those sites to gain access to the hematoma. After partial removal of the hematoma, the tension in the brainstem decreased. Thereafter, by changing the angle of the microscope the residual hematoma could be removed under direct visualization (Fig. 1B, D). It is not advisable to perform en bloc resection of large or hardened hematoma through the small incision made in the brainstem. Instead of enlarging the incision, it is recommended to use micro-forceps to break the hematoma into small pieces and then remove them in a piecemeal way. The surgical removal must be confined to hematoma, and completed when delineation between hematoma and normal brainstem tissue is identified. The precise movements and good intraoperative judgement are essential throughout the surgery. Sometimes, parts of the cerebellar vermis might need to be retracted aside to get extra exposure of the hematoma cavity instead of excessive retraction of the brainstem. The retractor in the brainstem, if needed, should act as a barrier between hematoma and normal parenchyma with minimal pressure. When hemorrhage occurred while removing hematoma, we used cotton paddy for gentle compression for venous small bleeding; for active bleeding, we carefully lifted the bleeder and coagulated with low bipolar cautery to avoid collateral heat damage to the brainstem. Sufficient irrigation while removing the hematoma not only helped to flush out the residual hematoma but also helped for better anatomical exposure. Sometimes hemostatic materials are used, but we recommend to leave less hemostatic materials in the surgical cavity. Excessive stuffed-in materials might prevent the cavity from closing and hinder the re-connection of the local circuits in the brainstem. Intraoperative neurophysiologic monitoring (motor evoked potential, sensory evoked potential) and neuronavigation are quite helpful for preserving neurofunctions. Post-operatively, the patient needs to be ventilated overnight and often needs early tracheostomy.

In our cases, the hematomas were completely removed and no arteriovenous abnormalities, or tumor apoplexy was seen during surgery. American Heart Association guidelines as well as European Stroke Organisation guidelines have not mentioned definite management plan for the spontaneous hypertensive brainstem hemorrhage [14,15]. Although the consciousness and vital signs were disrupted in our cases, all patients survived during hospitalization period. In our cases, two patients had a GOS of 4, one had a GOS of 3 and two had a GOS of 2. The prognosis of the patient had been relatively better in three cases who arrived within six hours of ictus. This might be due to prompt

surgical decompression before the initiation of secondary brainstem injury, while both cases with a GOS of 2 were transferred from local hospital and arrived after six hours. The space-occupying effect of the hemorrhage often with surrounding vasogenic edema causing secondary brainstem injury might have led to the slow recovery of the neurological function. Due to the academic controversy, currently no strong recommendations can be used in the therapeutic approach for the patients. However, it is reasonable to try a surgical removal of brainstem hematoma in patients with significant brainstem compression with progressively deteriorating clinical features, followed by postoperative medical management in neurosurgical intensive care unit.

5. Conclusion

Patients having brainstem hemorrhage resulting in severe brainstem compression and progressive deterioration of clinical features should undergo prompt surgical removal of hematoma, followed by postoperative medical management in neurosurgical intensive care unit. With the widening knowledge in microsurgical techniques as well as neuroimaging technology, there seems to have more optimistic hope for the surgical management of spontaneous hypertensive brainstem hemorrhage for better prognosis.

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.inat.2015.06.005>.

Conflict of Interest

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest, or non-financial interest in the subject matter or materials discussed in this manuscript.

References

- [1] E. Freytag, Fatal hypertensive intracerebral haematomas: a survey of the pathological anatomy of 393 cases, *J. Neurol. Neurosurg. Psychiatry* 31 (1968) 616–620.
- [2] M. Komiyama, Y.E. Boo, H. Yagura, T. Yasui, M. Baba, A. Hakuba, et al., A clinical analysis of 32 brainstem haemorrhages; with special reference to surviving but severely disabled cases, *Acta Neurochir. (Wien)* 101 (1989) 46–51.
- [3] J.R. Mangiardi, F.J. Epstein, Brainstem haematomas: review of the literature and presentation of five new cases, *J. Neurol. Neurosurg. Psychiatry* 51 (1988) 966–976.
- [4] E.M. Manno, J.L. Atkinson, J.R. Fulgham, E.F. Wijdicks, Emerging medical and surgical management strategies in the evaluation and treatment of intracerebral hemorrhage, *Mayo Clin. Proc.* 80 (2005) 420–433.
- [5] A.A. Rabinstein, S.H. Tisch, R.L. McClelland, E.F. Wijdicks, Cause is the main predictor of outcome in patients with pontine hemorrhage, *Cerebrovasc. Dis.* 17 (2004) 66–71.
- [6] T. Wessels, W. Moller-Hartmann, J. Noth, C. Klotzsch, CT findings and clinical features as markers for patient outcome in primary pontine hemorrhage, *AJNR Am. J. Neuroradiol.* 25 (2004) 257–260.
- [7] J.H. Jang, Y.G. Song, Y.Z. Kim, Predictors of 30-day mortality and 90-day functional recovery after primary pontine hemorrhage, *J. Korean Med. Sci.* 26 (2011) 100–107.
- [8] J.S. Raison, G. Bourbotte, T.P. Baum, M. Pages, Primary brain stem hemorrhage: retrospective study of 25 cases, *Rev. Neurol. (Paris)* 164 (2008) 225–232 [Abstract].
- [9] C.S. Chung, C.H. Park, Primary pontine hemorrhage: a new CT classification, *Neurology* 42 (1992) 830–834.
- [10] T. Hara, K. Nagata, S. Kawamoto, J. Sashida, T. Abe, A. Wada, et al., Functional outcome of primary pontine hemorrhage: conservative treatment or stereotaxic surgery, *No Shinkei Geka* 29 (2001) 823–829 [Abstract].
- [11] H. Takahama, K. Morii, M. Sato, K. Sekiguchi, S. Sato, Stereotactic aspiration in hypertensive pontine hemorrhage: comparative study with conservative therapy, *No Shinkei Geka* 17 (1989) 733–739 [Abstract].
- [12] S. Takeuchi, G. Suzuki, Y. Takasato, H. Masaoka, T. Hayakawa, N. Otani, et al., Prognostic factors in patients with primary brainstem hemorrhage, *Clin. Neurol. Neurosurg.* 115 (2013) 732–735.
- [13] S.J. Haines, H.D. Mollman, Primary pontine hemorrhagic events. Hemorrhage or hematoma? Surgical or conservative management? *Neurosurg. Clin. N. Am.* 4 (1993) 481–495.
- [14] L.B. Morgenstern, J.C. Hemphill III, C. Anderson, K. Becker, J.P. Broderick, E.S. Connolly Jr., et al., Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association, *Stroke* 41 (2010) 2108–2129.
- [15] T.I. Steiner, R. Al-Shahi Salman, R. Beer, H. Christensen, C. Cordonnier, L. Csiba, et al., European Stroke Organisation (ESO) guidelines for the management of spontaneous intracerebral hemorrhage, *Int. J. Stroke* 9 (2014) 840–855.