Accepted Manuscript

Delayed deterioration in isolated traumatic subarachnoid hemorrhage Report of two cases

Toshiaki Hayashi, Hiroshi Karibe, Ayumi Narisawa, Motomobu Kameyama

PII: S1878-8750(15)01349-2

DOI: 10.1016/j.wneu.2015.09.108

Reference: WNEU 3301

To appear in: World Neurosurgery

Received Date: 31 July 2015

Revised Date: 28 September 2015

Accepted Date: 29 September 2015

Please cite this article as: Hayashi T, Karibe H, Narisawa A, Kameyama M, Delayed deterioration in isolated traumatic subarachnoid hemorrhage Report of two cases, *World Neurosurgery* (2015), doi: 10.1016/j.wneu.2015.09.108.

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.



Delayed deterioration in isolated traumatic subarachnoid hemorrhage Report of two cases

Toshiaki Hayashi, Hiroshi Karibe, Ayumi Narisawa, Motomobu Kameyama

Department of Neurosurgery, Sendai City Hospital 1-1-1 Asutonagamachi Taihaku-ku, Sendai, Miyagi 982-8502, Japan Tel: +81-022-308-7111; Fax: +81-022-308-7153 →

Future correspondence and reprint request should be addressed to: Toshiaki Hayashi E-mail: hayashi@nsg.med.tohoku.ac.jp

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

Key words: abruption of perforator, delayed deterioration, Sylvian hematoma, traumatic subarachnoid hemorrhage

Abbreviations:

tSAH: traumatic subarachnoid hemorrhage; mTBI: mild traumatic brain injury; CT: computed tomography; GCS: Glasgow coma scale; MRA: magnetic resonance angiography; MCA: middle cerebral artery

Abstract

Background: Isolated traumatic subarachnoid hemorrhage (isolated tSAH) in association with mild traumatic brain injury is considered to be a less severe finding that is not likely to require surgical intervention. No previous reports appear to have described cases warranting craniotomy for isolated tSAH by itself. Clinical presentation: We report two cases of minor head trauma with isolated tSAH that showed delayed clinical deterioration requiring immediate surgical intervention. Initial computed tomography (CT) showed isolated tSAH in the basal cistern and Sylvian fissure in both cases. Angiography showed no aneurysmal source. Within 24 h of each accident, both disturbance of consciousness and hemiparesis deteriorated. Follow-up CT showed formation of intracerebral hematoma adjacent to the Sylvian fissure. Intraoperative findings showed abruption injury of a perforating branch arising from the middle cerebral artery as the cause of bleeding. Impact at the time of injury could have caused traction on the middle cerebral artery in the Sylvian fissure, resulting in abruption of the perforator. Conclusion: We conclude that isolated tSAH seen in the basal cistern and Sylvian fissure carries a risk of late deterioration. A possible cause of hematoma expansion is abruption of a perforating branch arising from the MCA at the time of head injury. Once hematoma expansion is identified, surgical evacuation of the hematoma is indicated. The surgical evacuation should be safely performed with the knowledge of the bleeding point in such patients.

Background and Importance:

Recent studies^{1,3-5,7,8} have suggested that isolated traumatic subarachnoid hemorrhage (tSAH) in mild traumatic brain injury (mTBI) is a relatively less severe finding that is unlikely to require surgical intervention. Although the potential need for repeat computed tomography (CT) to check for the development of delayed intracranial hematoma formation has been suggested in elderly patients^{4,5,10} a risk of delayed clinical deterioration in patients with isolated tSAH has not been reported previously. Moreover, no reports have clearly shown the origin of an isolated tSAH. We report two cases of isolated tSAH in which delayed clinical deterioration required emergency surgical intervention. Clinical characteristics and possible pathophysiological mechanisms are discussed.

Clinical presentation:

Case 1

A 73-year-old man with a history of Parkinsonism and hypertension presented to our emergency department after falling from a bed and hitting his forehead on a wooden desk. He had been alert and oriented, with a Glasgow coma scale (GCS) score of 15. Initial CT on referral demonstrated SAH in the left Sylvian fissure (Fig. 1A). Magnetic resonance angiography (MRA) found no aneurysmal source (Fig. 2A). He was admitted to our neurosurgical department for course observation. Results of laboratory coagulation tests on admission were within normal range. The patient had no platelet disorders, and did not take oral anticoagulants or anti-platelet drugs. His blood pressure was well controlled by administration of calcium channel blockers. Twenty hours after the accident, he developed right-sided motor weakness and disturbance of consciousness (GCS, 10: E3V2M5). Follow-up CT revealed left Sylvian hematoma with surrounding intracerebral hematoma (Fig. 1B). Emergency craniotomy was performed for evacuation of the hematoma and hemostasis. Intraoperative examination identified a tear in the one of perforating branch arising from the M2 branch of middle cerebral artery (MCA), feeding the insular cortex as the cause of bleeding (Fig. 2B). The arterial tear was reconstructed using 10-0 nylon sutures. Postoperative course was uneventful. The patient was discharged to rehabilitation with mild right hemiparesis.

4

Case 2

A 52-year-old woman with no significant past medical history was transferred from a referring hospital after a minor collision with a motor vehicle while walking along a road. Initial GCS score at the referring hospital was 15, without any significant neurological deficits. Initial CT at the referring hospital demonstrated SAH in the left Sylvian fissure (Fig. 3A). MRA did not reveal any aneurysmal source. Three hours after the accident, she developed right-sided motor weakness and motor aphasia. Follow-up CT revealed left intracerebral hematoma concomitant with Sylvian hematoma (Fig. 3B). She was then transferred to our neurosurgical department for further treatment. On admission, GCS score was 7 (E1V1M5) with right hemiparesis. CT angiography showed neither an aneurismal source nor a point of bleeding. Results of laboratory coagulation tests on admission were within normal range. The patient had no platelet disorders. Her blood pressure was well controlled using calcium channel blockers. Emergency craniotomy was performed to alleviate the mass effect caused by the hematoma (Fig. 4A, B). Intraoperative findings showed abruption of the small perforating branch arising from the MCA (frontal branch of M2) that penetrated to the left frontal operculum as the cause of bleeding (Fig. 4C). The stump of the pulled-out artery was electrocoagulated (Fig. 4D). Postoperative course was uneventful. Hemiparesis improved to allow normal activities of daily living, and she was discharged to rehabilitation with mild motor aphasia.

Discussion:

Despite the absence of evidence-based guidelines for managing mTBI in patients with tSAH, recommendations have been published. Most reports have stated that tSAH itself is not a significant prognostic factor predictive of further medical or surgical treatment of mTBI^{1,3-5,7,8}. Recent reports have found no neurological decline or need for neurosurgical procedures in patients with mTBI (GCS score \geq 13) and isolated tSAH^{1,3,7,8}. Lee et al.⁴ reported that only three of 661 patients (0.45%) with isolated tSAH underwent any sort of aggressive neurosurgical or endovascular intervention. They described two patients who received burr hole surgery to control intracranial pressure, and one patient who underwent an endovascular procedure to treat symptomatic vasospasm. No patients needed craniotomy. Conversely, Wu et al.¹⁰

consideration of neurosurgical consultation, similar to evidence of progressive hemorrhage on repeat CT. They reported two patients with such SAH who underwent craniotomy, although the types of surgery were not described. They suggested the existence of vascular malformation and cerebral aneurysm as associated with a need for craniotomy. Our report is the first to reveal that tSAH in the basal cistern or Sylvian fissure associated with subsequent hematoma formation through abruption of the perforating branch of the M2 segment of the MCA. We speculate that a traumatic impact could cause retraction of the M2 segment of the MCA floating in the Sylvian fissure concomitant with the shift in brain parenchyma, resulting in abruption of the perforating artery arising from the M2 segment of the MCA. The postoperative cerebral infarctions seen in our cases might suggest that the cause of the bleeding was injury to the perforating artery arising from MCA (Fig. 5), although the possibility that surgical manipulation caused injury to the parenchyma was not totally ruled out. Abruption or tear of the proximal portion of a small perforating artery caused SAH in the Sylvian fissure and basal cistern, while continuous bleeding caused gradual hematoma formation within the Sylvian fissure, finally extending to the adjacent cerebral parenchyma (Case 2). One previous case report⁶ showed traumatic false aneurysm formation at the M2 branch of the MCA. Initial findings on CT and the clinical course of later hematoma formation in the patient were very similar to those seen in our Case 1. That case would thus suggest a process of hematoma formation caused by a tear in the M2 branch of the MCA. Arterial bleeding from small vessels continued until local hemostasis was achieved by surrounding hematoma, which appeared to involve a false aneurysm, resulting in equilibrium between pressure from the hematoma cavity and local blood pressure at the bleeding point. Considering that it took 20 h for clinical deterioration to become apparent in Case 1, subsequent bleeding after primary hemostasis that could have been achieved by pseudoaneurysm formation had occurred by rupturing the aneurysm. Transarachnoid extension of late hemorrhage was another possible etiology for Case 1. Moreover, hemostasis by local vasospasm or thrombosis at the site of vascular injury might play a role for later hematoma expansion, as previously discussed in the cases of some SAH patients with unknown aneurysmal sources⁹. Patients with an aneurysmal pattern of hemorrhage on CT, but with negative results on angiography, are well known to still be at risk of developing secondary ischemia and

rebleeding. Our report may be the first to describe the bleeding point of such hematoma ascertained by surgical intervention, except for hidden aneurismal sources.

In our cases, types of injury and sites of bruising were varied. We could not find any reliable sign of late deterioration on initial CT or magnetic resonance imaging. Thick SAH in the Sylvian valleculae was the only common sign suggesting later deterioration caused by abruption of a small perforating artery, although that finding was not specific and was seen among many tSAH patients. Preoperative diffusion-weighted imaging failed to show any sign of the infarctions caused by such a small perforator injury, as revealed postoperatively (Fig. 5). MRA yielded negative results. Contrast-enhanced CT at the time of initial referral could show signs of active bleeding from injured small arteries, as well as spot signs in acute intracerebral hemorrhage², but this method seems less practical. Our institution is a core hospital with an emergency department and treats over 150 cases of admitted TBI each year, but has encountered no such cases during the last ten years.

Once clinical deterioration due to hematoma formation is identified, craniotomy should be performed to improve any mass effect from the hematoma and prevent further hematoma expansion. Before craniotomy, angiography should be performed to exclude the possibility of vascular malformation or aneurysm rupture, because the surgical strategies vary between those pathologies and abruption of a small artery. Bleeding caused by abruption of a small artery is easy to control, whereas others are not. We performed reconstruction of the artery in Case 1, but could not do so in Case 2. Considering that neither case showed neurological symptoms associated with cerebral infarction due to the injured perforating arteries, reconstruction of the injured artery would not have been a priority in any case. To improve the prognosis in such cases, clinicians should be aware that mTBI with tSAH may show late deterioration with hematoma expansion.

Conclusion:

We conclude that isolated tSAH seen in the basal cistern and Sylvian fissure carries a risk of late deterioration. One possible cause of hematoma expansion is abruption of a perforating branch arising from the MCA at the time of head injury. Identification of hematoma expansion is an indication for surgical evacuation of the hematoma. Surgical

evacuation should be safely performed in the knowledge of the bleeding point in such patients.

References

1. Borczuk P, Penn J, Peak D, Chang Y: Patients with traumatic subarachnoid hemorrhage are at low risk for deterioration or neurosurgical intervention. J Trauma Acute Care Surg 74: 1504-1509, 2013

 Brouwers HB, Goldstein JN, Romero JM, Rosand J: Clinical application of the computed tomography angiography spot sign in acute intracerebral hemorrhage. Stroke 43: 3427-3432, 2012

3. Ditty BJ, Omar NB, Foreman PM, Patel DM, Pritchard PR, Okor MO: The nonsurgical nature of patients with subarachnoid or intraparenchymal hemorrhage associated with mild traumatic brain injury. J Neurosurg 19: 1-5, 2014

4. Lee JJ, Segar DJ, Asaad WF: Comprehensive Assessment of Isolated Traumatic Subarachnoid Hemorrhage. J Neurotrauma 31: 595-609, 2014

5. Mattioli C, Beretta L, Gerevini S, Veglia F, Citerio G, Cormio M, Stocchetti N: Traumatic subarachnoid hemorrhage on the computerized tomography scan obtained at admission: a multicenter assessment of the accuracy of diagnosis and the potential impact on patient outcome. J Neurosurg 98: 37-42, 2003

6. Ohta M, Matsuno H: Proximal M2 false aneurysm after head trauma. Case report. Neurol Med chir (Tokyo) 41: 131-134, 2001

7. Phelan HA, Richter AA, Scott WW, Pruitt JH, Madden CJ, Rickert KL, Wolf SE: Does isolated traumatic subarachnoid hemorrhage merit a lower intensity level of observation than other traumatic brain injury? J Neurotrauma 31: 1733-1736, 2014

8. Quigley MR, Chew BG, Swartz CE, Willberger JE: The clinical significance of isolated traumatic subarachnoid hemorrhage. J Trauma Acute Surg 74: 581-584, 2013

9. Van Gijn J, RinkelGJ: Subarachnoid hemorrhage: diagnosis, causes and management. Brain 124: 249-278, 2001 10. Wu C, Orringer DA, Lau D, Fletcher JJ: Cumulative incidence and predictors of neurosurgical interventions following nonsevere traumatic brain injury with mildly abnormal head imaging findings. J Trauma Acute Care Surg 73: 1247-1253, 2012

Figure legends:

Figure 1: Case 1

A) Initial CT on admission shows SAH in the left basal cistern and Sylvian fissure. B) CT at the time of deterioration shows Sylvian hematoma. Note that SAH was not seen in the right basal cistern, suggesting gradual hematoma expansion.

Figure 2: Case 1

A) MRA on admission shows no aneurismal source. B) Intraoperative findings show a tear in the perforating artery arising from M2 branch of the MCA as the cause of bleeding (arrow). C) Postoperative CT shows removal of the hematoma.
*: Sylvian vein; F: frontal lobe; arrowhead: injured perforating artery.

Figure 3: Case 2

A) Initial CT soon after injury shows SAH in the left basal cistern and Sylvian fissure.B) CT obtained at the time of deterioration shows Sylvian hematoma and intracerebral hematoma. C) Postoperative CT shows removal of the hematoma.

Figure 4:

A) Intraoperative findings show the hematoma packing the Sylvian fissure. B) After removing Sylvian hematoma, MCA was revealed in the Sylvian fissure. C) The bleeding point was identified as an abruption of the perforating artery (arrow). D) The stump of the pulled-out artery was electrocoagulated.

*: Sylvian vein; F: frontal lobe; T: temporal lobe; arrowhead; injured perforating artery.

Figure 5:

A, B) Pre- (left) and postoperative (right) diffusion-weighted MRI for Case 1 (A) and Case 2 (B). Preoperative MRI does not show any cerebral infarction caused by injured perforating arteries arising from M2 branch of MCA, but infarctions are apparent postoperatively.













Highlights

We reported two cases of isolated traumatic SAH that showed late deterioration with Sylvian hematoma formation.

The abruption injury of a perforating branch of MCA caused hematoma formation. The mechanism of the late deterioration of traumatic SAH was discussed. Although these cases are rare, we should note that these cases do exist.

Once hematoma expansion is identified, surgical evacuation of the hematoma is indicated. The surgical evacuation should be safely performed with the knowledge of the bleeding point of such patients.

CER CER