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CASE REPORT

Clinical features and risk factors for delayed rupture of traumatic cerebral aneurysm: A case series

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Abstract

Background: Traumatic cerebral aneurysms (TA) are a subset of traumatic cerebrovascular injury (TCVI). Misdiagnosis of TA can be fatal. To investigate factors that predict TA formation and the optimal timing for searching, we present four suspected cases of delayed TA rupture during hospitalization.

Case Presentation: Medical records of head injury cases to have delayed TA rupture during hospitalization between April 2021 and March 2022 were retrospectively reviewed. Of the four patients included, only one met the TCVI screening criteria. All the patients had acute subdural hematoma (ASDH) on arrival; two had delayed expansion of the traumatic subarachnoid hemorrhage (tSAH) on repeat imaging. All the patients received anticoagulants. Ruptured TA occurred between days 5 and 11. Three patients died during hospitalization.

Conclusion: It is advisable to suspect TA when imaging studies show ASDH on admission and intracranial hematoma expansion during hospitalization. We suggest TA screening around day 5.

KEYWORDS
acute subdural hematoma, delayed traumatic intracranial hematoma, traumatic cerebrovascular injury

BACKGROUND

Traumatic cerebrovascular injury (TCVI) is associated with approximately 1% of blunt cranial trauma cases. Traumatic cerebral aneurysms (TA) are a subset of the TCVI. Misdiagnosis of TA can be fatal, with a mortality rate exceeding 50% upon rupture[1] this underscores the importance of early detection. The recommended TCVI screening criteria, as delineated in the Japanese Guidelines[2] (Table 1), primarily focus on the detection of carotid and vertebral arteries, and the American guidelines for blunt cerebrovascular injuries (BCVI),[3] have a similar concept; however, both may be insufficient for screening TA occurring intracranially. Given the lack of established factors predicting TA formation beyond the existing criteria and the unclear optimal timing for TA detection, we retrospective analyzed four suspected cases of delayed TA rupture during hospitalization to identify the risk factors for delayed TA rupture during hospitalization following head injury.

CASE PRESENTATION

Four patients with suspected delayed TA rupture during hospitalization at Nippon Medical School Chiba Hokusoh Hospital between April 2021 and March 2022 were included. Demographic data and clinical findings during the hospital stay were extracted. The diagnosis of TA rupture was confirmed by neurosurgical specialists based, on head computed tomography (CT) or cerebral angiography. Temporal progression on head CT imaging was defined as hematoma expansion over 24 h after injury. Table 2 presents clinical

TABLE 1 Traumatic cerebrovascular injury screening criteria.[2]

Sever cervical hyperextension/ rotation or hyperflexion (especially with diffuse axonal brain injury)
Focal neurologic defect (transient ischemic attack, amaurosis fugax, Horner's syndrome)
Stroke on computed tomography or magnetic resonance imaging
Seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling or altered mental status
Cervical bruit in a patient younger than 50 years
Basilar skull fracture with carotid canal involvement or foramen lacerum
Mid-face fracture (LeFort type II or III, complex mandibular fracture)
Cervical vertebral body fracture

characteristics and imaging findings. The patients were 33–79 years. The injury severity score (ISS) ranged from 13 to 22. The Glasgow coma scale (GCS) on arrival was 14–15 points. All patients had acute subdural hematoma (ASDH), and two had traumatic subarachnoid hemorrhage (tSAH). One patient had received antiplatelet therapy before the trauma incident. All patients received 10,000–15,000 units of heparin calcium per day for prophylaxis of venous thromboembolism (VTE). Only one patient met the existing TCVI screening criteria. TA rupture occurred between the fifth and eleventh day. Three patients had TA in the middle cerebral artery (MCA), and one had TA in the posterior cerebral artery (PCA). Three patients underwent emergency cranial decompression. Three patients died and one was discharged. The clinical course of the representative case is shown below.

CASE 3

A 77-year-old man was injured after falling backward. On admission, the patient's GCS score was 15 (E4V5M6), and a CT scan showed ASDH over the cerebellar tent (Figure 1A). Other injuries included fractures of the 1st and 2nd cervical vertebrae. The administration of heparin calcium (10,000 U/day) was administered on day 3. CT showed a low-density area in the right PCA region, and cerebral infarction was diagnosed on day 4. His consciousness decreased to GCS 3 (E1V1M1) on day 5, and a CT scan showed ASDH in the right temporal region with a midline shift. Emergency cranial decompression surgery was performed, and postoperative CT angiography revealed a TA in the right PCA (Figure 1B). However, the patient died on day 6. Three of the recommended criteria for TCVI screening were met: excessive neck flexion, cerebral infarction on CT, and cervical vertebral fracture.

DISCUSSION

In this study, all patients had clear or mildly impaired consciousness upon arrival. However, three patients died after the TA rupture. Despite the poor prognosis, only one patient

TABLE 2 Characteristics of four cases and imaging of head injury.

Case	Age	Sex	Mechanism	GCS On admission	ISS	Meeting TCVI criteria	Antiplatelet use	Administration of anticoagulant	TA rupture day	Outcome	TBI on admission	Intracranial hematoma expansion	Region of TA
1	33	F	Fall	14	13	No	Yes	Day 5	Day 7	Discharged	Parietal ASDH	No	Rt. MCA
2	61	M	Fall	15	17	No	No	Day 7	Day 11	Dead	Temporal ASDH Sylvian fissure tSAH Contusion	Sylvian fissure	Rt. MCA
3	77	M	Fall	15	21	Yes	No	Day 3	Day 5	Dead	Cerebellar tentorium ASDH	No	Rt. PCA
4	79	M	MVA	14	22	No	Yes	Day 4	Day 8	Dead	Frontal ASDH Sylvian fissure tSAH Contusion	Sylvian fissure	Lt. MCA

Abbreviations: ASDH, acute subdural hematoma; Contusion, brain contusion; GCS, Glasgow Coma Scale; ISS, injury severity score; MCA, middle cerebral artery; MVA, motor vehicle accident; PCA, posterior cerebral artery; TA, traumatic cerebral aneurysm; TBI, traumatic brain injury; TCVI, traumatic cerebrovascular injury; tSAH, traumatic subarachnoid hemorrhage.

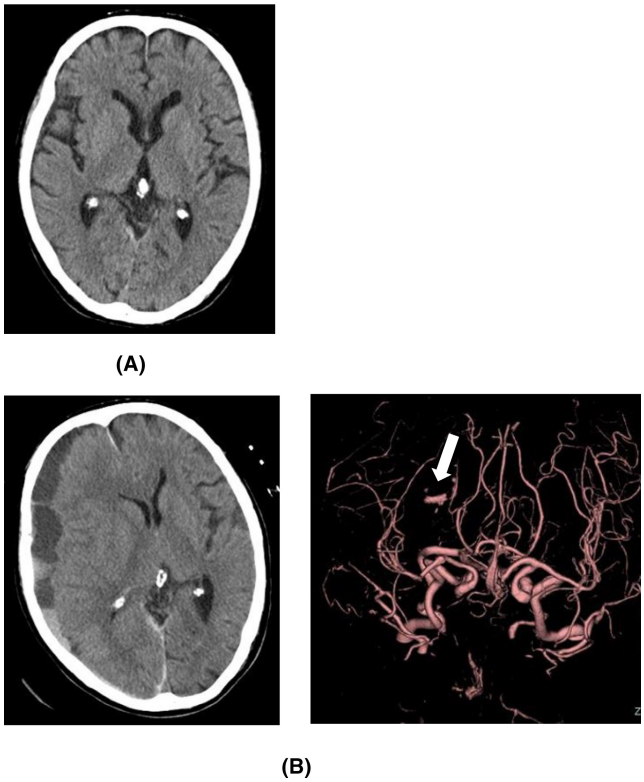

FIGURE 1 (A) Head computed tomography (CT) of Case 3 on admission, (B) Head CT and CT angiography of Case 3 on day 5. Arrow shows a traumatic aneurysm in the right posterior cerebral artery.

met the TCVI screening criteria, indicating that existing criteria are insufficient for detecting TA formation and new criteria are needed.

In some cases, imaging studies have shown ASDH and delayed hematoma expansion, were potentially indicative of TA. A previous study reported that TA in the MCA commonly formed secondary to ASDH at the temporal lobe[4] and that TA in the PCA was secondary to ASDH on the cerebellar tent.[5] It is assumed that the temporal lobe collides with the sphenoid edge[4] and that occipital lobe collides with the cerebellar tent due to cervical hyperflexion,[5] resulting in direct MCA and PCA injury, and TA formation. Hematoma on the cerebellar tent could be an indirect sign of intracranial vascular injury from cervical hyperflexion. This study identified one case of ASDH in the cerebellar tent and TA in the PCA. The other three patients showed ASDH in the frontal to temporal regions, suggesting that TA rupture formed in the MCA.

Delayed expansion of the intracranial hematoma after injury may result from both the consumption of coagulation factors and TA rupture.[6] In particular, delayed expansion of tSAH was identified in two patients, suggesting TA formation.[7] Rapid expansion of the hematoma on serial images indicates TA rupture, requiring further aggressive exploration (e.g., CT or MR angiography).

In this study, all the patients received anticoagulants for thromboprophylaxis after admission. As far as we know, it is not clear whether anticoagulants are the risk for TA rupture and the appropriate timing for their administration. However, in other locations anticoagulant use is associated with the formation of pseudoaneurysms,[8] so we believe that anticoagulant use was also associated with fatal bleeding. Thus, aggressive TA exploration before anticoagulant administration is essential for at risk patients.

Typically, TA is detected a few days to weeks after head injury.[3] In this study, TA rupture occurred between days 5 and 11. A previous study reported that approximately half of patients showed a false-negative result on initial images and most TAs were detectable 6 day after injuries.[7] Considering this study's results, in cases involving bleeding risk, such as the anticoagulants use, the optimal timing for initial TA screening could be around day 5, slightly earlier than in the previous report. Even if no TA is found, screening should be repeated if TA is suspected. However, the optimal interval, duration, and choice of screening methods remain unclear.

This study had several limitations. First, TA diagnosis relied on CT imaging and intraoperative findings by a neurosurgeon based on clinical implications. Differential diagnoses include delayed traumatic intracranial hematoma (DTICH), which often appears within 48 h of injury.[9] Complete vascular imaging such as angiography, is required to differentiate between TA and DTICH,[10] the diagnosis was confirmed using these modalities in only one case. Second, non-traumatic unruptured aneurysms cannot be ruled out based on concrete evidence and depended on the neurosurgeons' discretion. Finally, we only assessed patients with ruptured TA. Therefore, we might have missed cases of undiagnosed TA that could have been managed nonoperatively. Future studies on TA morphology and localization are required to clarify the characteristics of aneurysms that require intervention.

CONCLUSION

Whether patients with traumatic brain injury meet the existing criteria for TCVI screening, it is advisable to suspect TA when serial images show ASDH on admission and intracranial hematoma expansion during the hospital course. We suggest implementing TA screening around day 5 before using anticoagulants if patients are considered at a high risk of TA.

CONFLICT OF INTEREST STATEMENT

Dr. Shoji Yokobori is an Editorial Board member of AMS Journal and a co-author of this article. To minimize bias, they were excluded from all editorial decision-making related to the acceptance of this article for publication.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available at corresponding author's request.

ETHICS STATEMENT

Approval of the research protocol: N/A.

Informed Consent: N/A.
Registry and the Registration No. of the study/Trial: N/A.
Animal Studies: N/A.

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