




Traumatic Middle Meningeal Artery Aneurysm: A Rare Cause of Recurrent Acute Epidural Hematoma. A Case Report

Toshihide Takahashi¹  Kiyoyuki Yanaka¹ Hitoshi Aiyama¹ Minami Saura¹ Michihide Kajita¹
Nobuyuki Takahashi² Eiichi Ishikawa³

¹ Department of Neurosurgery, Tsukuba Memorial Hospital, Tsukuba, Ibaraki, Japan

² Department of Radiology, Tsukuba Memorial Hospital, Tsukuba, Ibaraki, Japan

³ Department of Neurosurgery, Institute of Medicine, University of Tsukuba, Tsukuba, Ibaraki, Japan

Address for correspondence: Eiichi Ishikawa, MD, PhD, Department of Neurosurgery, Institute of Medicine, University of Tsukuba, Tennodai 1-1-1, Tsukuba, Ibaraki 305-8577, Japan
(e-mail: e-ishikawa@md.tsukuba.ac.jp).

Asian J Neurosurg 2024;19:825–828.

Abstract

Traumatic middle meningeal artery aneurysm (TMMA) is a rare condition and a known cause of several different bleeding patterns after head injury. Once detected, they need to be treated as an emergency due to their potential for morbidity and mortality. Generally, recurrence does not occur in surgery for acute epidural hematoma if adequate hemostasis is achieved. Here, we report a case of atypical postoperative recurrence of an acute epidural hematoma, possibly due to the development and rupture of a TMMA. A 41-year-old man with left acute epidural hematoma after a head injury was referred to our hospital. Emergency craniotomy was performed immediately, and the hematoma was removed. The source of the bleeding was near the fracture site in the middle cranial fossa, and sufficient hemostasis was confirmed. However, a head computed tomography (CT) scan the next day revealed a recurrence of the acute epidural hematoma. Magnetic resonance (MR) angiogram showed an aneurysm with a diameter of approximately 4 mm in the left middle meningeal artery. The recurrence of the acute epidural hematoma appeared to be related to the formation and a rupture of a middle meningeal artery aneurysm, and to prevent subsequent rebleeding, the patient underwent reoperation, and the hematoma and aneurysm were removed. In surgery for acute epidural hematoma, recurrence can be prevented by removing the hematoma and ensuring hemostasis. Although conventional surgery was performed in this case, a repeat of epidural hematoma occurred. A postoperative middle meningeal artery aneurysm had been thought to have developed, ruptured, and caused a repeat epidural hematoma.

In treating acute epidural hematoma, a TMMA development should be considered when an atypical clinical course occurs, such as a recurrence of postoperative bleeding.

Keywords

- epidural hematoma
- head injury
- middle meningeal artery
- pseudo aneurysm
- trauma

article published online
October 4, 2024

DOI <https://doi.org/10.1055/s-0044-1791580>.
ISSN 2248-9614.

© 2024. Asian Congress of Neurological Surgeons. All rights reserved.

This is an open access article published by Thieme under the terms of the Creative Commons Attribution-NonDerivative-NonCommercial-License, permitting copying and reproduction so long as the original work is given appropriate credit. Contents may not be used for commercial purposes, or adapted, remixed, transformed or built upon. (<https://creativecommons.org/licenses/by-nc-nd/4.0/>)

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

Introduction

Traumatic intracranial aneurysms rarely occur as a result of head injury. The occurrence mechanism is suggested to be related to skull fractures. Although the incidence of traumatic intracranial aneurysms is low at 0.09 to 0.4% of all cerebral aneurysms, traumatic middle meningeal artery aneurysms (TMMAs) account for approximately one-quarter of all traumatic intracranial aneurysms.¹ TMAA rupture may cause different types of intracranial hemorrhages. The overall incidence of acute epidural hematoma is low at 3.8%, but the prognosis is unfavorable, with a mortality rate of 20%.^{2–4} Since TMAA rupture is reported to occur 1 to 30 days after injury,² early preventive treatment is recommended when the aneurysm is detected. Here, we report a case in which a TMAA developed and ruptured after surgery for acute epidural hematoma, which appeared to result in hematoma recurrence.

Case History

A 41-year-old man with no specific medical history or heavy alcohol consumption fell at work and bruised the left side of his head. At first, he was conscious and articulate, but gradually, his conversation became incoherent, and his colleague took him to a local doctor. A head computed tomography (CT) scan revealed acute epidural hematoma and

temporal bone fracture on the left side, and the patient was urgently brought to our hospital due to progressive disturbance of consciousness and drowsiness. On admission, there was no apparent paresis, but his consciousness was obscured, and he was restless, with a Glasgow Coma Scale score of 12 (E3V3M6). General blood tests showed no abnormalities, and there was no tendency to bleeding. A previously performed CT scan indicated a fracture of the left temporal bone and a left acute epidural hematoma (►Fig. 1A, B). An emergency craniotomy was performed due to progressive impairment of consciousness.

After craniotomy and hematoma removal, persistent bleeding from the middle cranial fossa was noted, and hemostasis was obtained by applying pressure to the cellulose oxide. Dural surface examination did not reveal any vascular malformations. The compressed brain returned to its original position when the hematoma was removed. After confirming adequate hemostasis, dural tack-up sutures were placed around and in the center of the craniotomy to prevent re-accumulation of blood in the epidural space, and the procedure was completed.

The next day, a CT scan showed epidural hematoma recurrence (►Fig. 1C); however, reoperation was not considered at that time because the patient's impaired consciousness was improving. It was thought that the recurrent hematoma may have formed due to inadequate hemostasis or delayed brain deviation recovery, with a large space remaining and re-accumulation of blood.

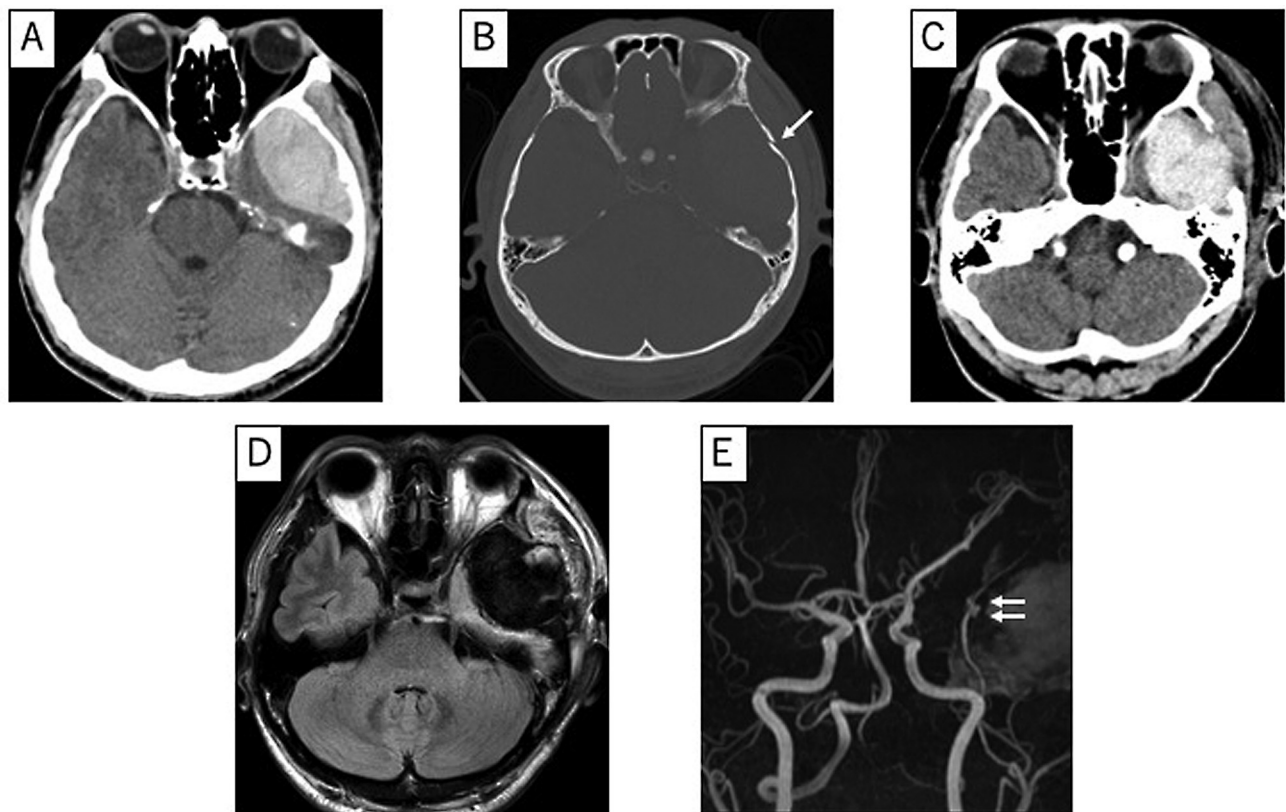


Fig. 1 On admission, computed tomography (CT) showed (A) acute epidural hematoma and (B) skull fracture (white arrow), both on the left side. (C) CT on the next day after surgery showed hematoma recurrence. Seven days after injury, (D) a fluid-attenuated inversion recovery magnetic resonance (MR) imaging showed no hematoma enlargement, and (E) MR angiography showed an aneurysm of the left middle meningeal artery (two white arrows).

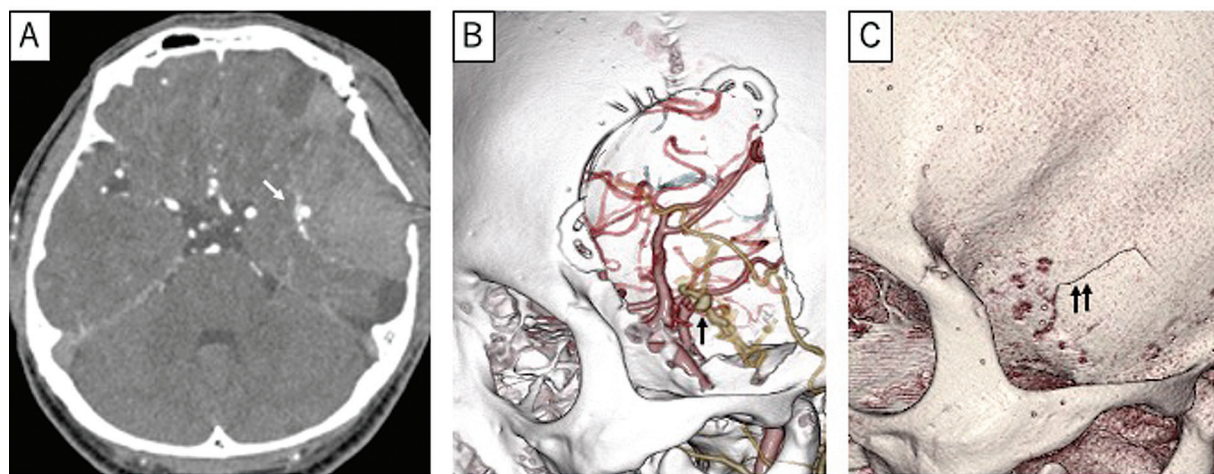


Fig. 2 (A) Contrast-enhanced computed tomography (CT) performed 8 days after the initial surgery confirmed the presence of the aneurysm of the middle meningeal artery located at the deepest part of the hematoma (white arrow). Three-dimensional CT angiography showed the (B) middle meningeal artery aneurysm (black arrow) corresponding to the (C) temporal bone fracture (double arrow) during head injury.

Seven days after surgery, magnetic resonance (MR) imaging was performed to assess brain damage and changes in hematoma volume. No enlargement of hematoma was observed (►Fig. 1D). Nevertheless, a small TMMA was visible on MR angiogram (►Fig. 1E). Three-dimensional CT angiography was performed for a detailed assessment of the TMMA, which was 4 mm in diameter in the thickest (deepest) part of the hematoma (►Fig. 2A). Referring to the initially taken CT, the TMMA was located directly below the temporal bone fracture (►Fig. 2B, C).

To prevent TMMA re-rupture, hematoma removal and resection of the aneurysm were performed. A relatively hard blood clot was found on the dural surface, which was continuous with the middle meningeal artery and appeared to be an aneurysm. The aneurysm was removed, and the middle meningeal artery was coagulated.

The resected specimen had no apparent vascular wall structure and was basically a blood clot, and therefore it was diagnosed as a pseudoaneurysm. Postoperative MR angiography confirmed the disappearance of TMMA. The patient had an excellent postoperative course without hematoma recurrence and was discharged 30 days after the injury.

Discussion

The development of TMMA after head trauma is a relatively rare phenomenon. The middle meningeal artery, usually accompanied by two veins,⁵ runs within the dura mater on the inner skull surface. When the dura is damaged by head trauma, the middle meningeal artery is also susceptible to damage.⁶ A specific association with fractures has been suggested, with one report showing that 70 to 90% of TMMA cases described fractures across the middle meningeal artery.¹ TMMA is thought to be a pseudoaneurysm formed by hemostasis of the bleeding site due to thrombosis after middle meningeal artery injury and subsequent resorption of the hematoma, which leads to the formation of a false lumen.² The middle meningeal artery has histological features of an intracranial mesonephric defect,³ which is

also a predisposing factor for trauma-induced TMMA.⁴ Compared with the wall of a true aneurysm, the fibrous tissue of a pseudoaneurysm is more fragile, increasing the risk of aneurysm enlargement and delayed bleeding.¹

Although some cases of spontaneous resolution of TMMA have been reported, TMMA can rupture and cause various intracranial hematomas. It has also been reported that acute epidural hematoma occurred in 3.8% of cases.^{2,5} Its prognosis is poor, and the mortality rate is as high as 20%.⁶ TMMA are often small, 2 to 5 mm in diameter,² and the time from injury to aneurysm formation ranges from a few hours to 30 days.^{7,8} Most TMMA are assumed to rupture within 30 days after injury and require prompt preventive treatment.

Recurrence of epidural hematomas within the spinal canal is occasionally observed, but intracranial epidural hematoma recurrence is rare. A study of recurrent hematoma cases with traumatic intracranial hemorrhages reported that 59 out of 850 patients (6.6%) required a second craniotomy.⁹ About a quarter of them had epidural hematomas. Even when repeated craniotomy was required, it was reported that hemostasis during the initial surgery was sufficient in more than 80% of cases.⁹ The reason for hematoma recurrence was suggested to be related to coagulopathy, brain atrophy, and excessive alcohol consumption rather than insufficient hemostasis.⁹ It was also suggested that blood may reaccumulate in a large space, which forms after hematoma removal.⁹ As we previously reported, coagulopathy is thought to cause epidural hematoma in the spinal canal.¹⁰ Finally, if an atypical course such as hematoma recurrence occurs, it is considered necessary to thoroughly investigate the cause of recurrent bleeding.

In our case, recurrent hematoma was observed in the same area after the initial surgery for acute epidural hematoma. The dura mater was carefully examined during that surgery, and no abnormal vessels were found. It seemed strange, as hematoma recurrence occurred despite sufficient hemostasis and meticulous dural tack-up suturing to prevent recurrent bleeding. A later MR angiography confirmed TMMA, and its formation and rupture appeared to have caused epidural hematoma recurrence. We cannot exclude

the possibility that the middle meningeal artery on the surface of the dura mater was iatrogenically damaged during the initial surgery, resulting in aneurysm formation. However, the aneurysm developed along the fracture line, suggesting that the middle meningeal artery was damaged during head trauma. In other words, the hematoma itself may have compressed the damaged blood vessel, slowing the formation of the aneurysm, and when the hematoma was removed, the compression was released, and the aneurysm formed.

Because vascular assessment is not routinely performed in trauma cases, TMMA may be underdiagnosed. Also, because TMMA is often treated soon after diagnosis, its natural history remains unknown. The reports of Biffi et al¹¹ and Miller et al¹² are useful as screening criteria for head and neck vascular injuries in cases of blunt head and neck trauma. However, these reports do not emphasize the importance of repeated vascular injury assessment. This case may enlighten us about its importance.

This case also highlights the need to consider TMMA in atypical clinical scenarios (e.g., recurrent epidural hematoma). Important clinical implications for enhancing patient care in these cases include heightened vigilance; that is, if the postoperative course is atypical, clinicians should suspect rare causes such as TMMA and pay close attention to postoperative imaging. Additional imaging, such as MR or CT angiography, may be required to identify complications. Future research directions include understanding the mechanisms of TMMAs, conducting systematic case analyses to identify risk factors, and potentially revising clinical guidelines based on new evidence.

Additional research is needed to determine the true incidence of this entity, its natural history, and treatment guidelines. This case suggested that hematoma recurrence was associated with TMMA development and rupture after surgery for acute epidural hematoma. Our case illustrates the importance of a careful search for the source of bleeding in the management of head injuries. In particular, in cases with atypical clinical course, such as unexpected hematoma recurrence, the source of bleeding should be investigated, considering the possibility of TMMA formation.

Conclusion

We reported a recurrence of acute epidural hematoma, probably caused by the formation and rupture of a TMMA. In the management of head trauma, especially in acute epidural hematoma, TMMA formation should be considered in atypical clinical course cases, such as recurrent postoperative bleeding.

Ethical Approval

This study was approved by the Ethics Committee of Tsukuba Memorial Hospital. Need for written patient consent form was waived by the Ethics Committee because data were deidentified.

Funding

None.

Conflict of Interest

None declared.

Acknowledgment

The authors thank Dr. Alexander Zaboronok of the University of Tsukuba Institute of Medicine Department of Neurosurgery for language revision.

References

- 1 Montanari E, Polonara G, Montalti R, et al. Delayed intracerebral hemorrhage after pseudoaneurysm of middle meningeal artery rupture: case report, literature review, and forensic issues. *World Neurosurg* 2018;117:394–410
- 2 Bruneau M, Gustin T, Zekhnini K, Gilliard C. Traumatic false aneurysm of the middle meningeal artery causing an intracerebral hemorrhage: case report and literature review. *Surg Neurol* 2002;57(03):174–178, discussion 178
- 3 Hassler O. Medial defects in the meningeal arteries. *J Neurosurg* 1962;19:337–340
- 4 Nakamura K, Tsugane R, Ito H, Obata H, Narita H. Traumatic arterio-venous fistula of the middle meningeal vessels. *J Neurosurg* 1966;25(04):424–429
- 5 Meder JF, Gaston A, Merienne L, Godon-Hardy S, Fredy D. Traumatic aneurysms of the internal and external carotid arteries. One case and a review of the literature. *J Neuroradiol* 1992;19(04):248–255
- 6 Biesbroek JM, Rinkel GJ, Algra A, van der Sprenkel JW. Risk factors for acute subdural hematoma from intracranial aneurysm rupture. *Neurosurgery* 2012;71(02):264–268, discussion 268–269
- 7 Holmes B, Harbaugh RE. Traumatic intracranial aneurysms: a contemporary review. *J Trauma* 1993;35(06):855–860
- 8 Kawaguchi T, Kawano T, Kaneko Y, Ooasa T, Ooigawa H, Ogasawara S. Traumatic lesions of the bilateral middle meningeal arteries: case report. *Neurol Med Chir (Tokyo)* 2002;42(05):221–223
- 9 Bullock R, Hanemann CO, Murray L, Teasdale GM. Recurrent hematomas following craniotomy for traumatic intracranial mass. *J Neurosurg* 1990;72(01):9–14
- 10 Sakamoto N, Yanaka K, Matsumaru Y, Nose T. Cervical epidural hematoma causing hemiparesis. *Arch Neurol* 2003;60(05):783
- 11 Biffi WL, Moore EE, Elliott JP, Brega KE, Burch JM. Blunt cerebrovascular injuries. *Curr Probl Surg* 1999;36(07):505–599
- 12 Miller PR, Fabian TC, Croce MA, et al. Prospective screening for blunt cerebrovascular injuries: analysis of diagnostic modalities and outcomes. *Ann Surg* 2002;236(03):386–393, discussion 393–395