A Patient with a Pseudoaneurysm of the Middle Meningeal Artery Who Developed Intracerebral Hemorrhage during Conservative Treatment for Traumatic Subarachnoid Hemorrhage

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Objective: We report a 73-year-old male in whom a traumatic pseudoaneurysm of the middle meningeal artery (PMMA) increased during conservative treatment for traumatic subarachnoid hemorrhage, an acute subdural hematoma, and brain contusion, leading to intracerebral hemorrhage related to its rupture.

Case Presentation: During decommissioning operations, he fell down from the bed of a truck, and was brought to our hospital by ambulance. Head CT revealed traumatic subarachnoid hemorrhage, left acute subdural hematoma, and brain contusion. Conservative treatment was performed. Left temporal lobe hemorrhage related to the rupture of a traumatic pseudoaneurysm of the left middle meningeal artery (MMA) was observed 20 days after onset. Emergency endovascular treatment and hematoma removal under craniotomy were conducted.

Conclusion: In cases of subarachnoid hemorrhage after head trauma, serial changes should be assessed using CTA and DSA, considering the possibility of a traumatic PMMA.

Keywords ▶ pseudoaneurysm of the middle meningeal artery, intracerebral hemorrhage, hematoma removal under craniotomy, aneurysm embolization

Introduction

Traumatic pseudoaneurysms are rare among intracranial cerebral aneurysms, but traumatic pseudoaneurysms of the middle meningeal artery (PMMAS) are rarer. Intracranial hemorrhage due to PMMA rupture develops as an epidural hematoma in many cases, and the onset of intracerebral hemorrhage is extremely rare. We report a patient in whom a traumatic PMMA increased during conservative treatment for traumatic subarachnoid hemorrhage, an acute subdural hematoma, and brain contusion, leading to intracerebral hemorrhage related to its rupture.

Case Presentation

A 73-year-old male. During decommissioning operations, he fell down from the bed of a truck, and was brought to our hospital by ambulance. Consciousness was clear. He complained of lower back pain. On arrival, the Japan Coma Scale (JCS) score was 2, and retrograde amnesia was observed, but there were no abnormal neurological findings. Imaging showed left pelvic fracture. Head CT revealed traumatic subarachnoid hemorrhage, left acute subdural hematoma, and brain contusion (Fig. 1), but there was no skull fracture. Head CTA did not show any intracranial vascular abnormalities. He was admitted, and
PMMA rupture. Under general anesthesia, 3000 units of heparin were intra-arterially injected. A 6Fr guiding catheter (Roadmaster; Goodman, Aichi, Japan) was catheterized into the left external carotid artery, and a 4Fr Cerulean (Medikit, Tokyo, Japan) to the origin of the left MMA. A Marathon (Medtronic, Irvine, CA, USA) was catheterized into the aneurysm. Through Marathon, three Barricade coils Complex Finish (Balt, Irvine, CA, USA) (5 mm × 10 cm, 3 mm × 6 cm, and 3 mm × 4 cm, respectively) were inserted and 0.2 mL of 13% n-butyl-2-cyanoacrylate (NBCA) was intra-arterially injected into the aneurysm (Fig. 3). After observation of NBCA reflux to the posterior convexity branch of the left MMA, Marathon was removed. Final angiography through the Roadmaster indicated the disappearance of the aneurysmal sac, and treatment was completed. Heparin was not reversed. After surgery, there was no marked increase in the volume of hemorrhage, and there was no exacerbation of neurological findings, such as facial nerve paralysis. As there was no hematoma-related conservative treatment was selected. Concerning pelvic fracture, conservative treatment was also selected in cooperation with the Department of Orthopedics. Head CT after admission showed a decrease in the volume of intracranial hemorrhage, and there was no exacerbation of the consciousness state or neurological findings. As mild aprosexia remained, a referral to a recovery-phase hospital was decided on. However, left temporal pain and aphasia suddenly appeared 20 days after onset. Head CT revealed fresh intracerebral hemorrhage of the left temporal lobe (Fig. 2; upper). Head MRA, including source image, did not show any vascular abnormalities. Head CTA and DSA were performed considering the necessity of further detailed cerebrovascular examination. The imaging findings suggested an aneurysm of the anterior branch of the left middle meningeal artery (MMA; Fig. 2, lower). As there was continuity between the hematoma and aneurysm, emergency embolization of the aneurysm was performed under a diagnosis of intracerebral hemorrhage due to
A Patient with a PMMA Who Developed ICH during Conservative Treatment for tSAH (Fig. 5). The patient was referred to a recovery-phase hospital 60 days after PMMA rupture, with a JCS score of 3 and persistent sensory aphasia (modified Rankin Scale [mRS] 2).

Discussion

PMMAs account for 27% of all traumatic intracranial aneurysms, aneurysm, below 1% of all aneurysms. PMMA rupture appears as acute or delayed epidural hematomas in many cases. It is sometimes associated with subdural hematomas or subarachnoid hemorrhage, but the onset of intracerebral hemorrhage is extremely rare. To our knowledge, nine patients have been reported (Table 1). Several studies indicated that temporal bone fracture crossing over the MMA was present in approximately 70%–90%. Of seven patients with PMMAs, two patients developed intracerebral hemorrhage...
MMA might be etiologically involved in pseudoaneurysms, suggesting that PMMAs are not always located at an area adjacent to or below a fracture line. Bozzetto-Ambrosi et al. 7) did not describe the severity of head trauma in a PMMA patient who developed intracerebral hemorrhage. In the absence of fracture. As for mechanism of fracture-free PMMAs, Lim et al. 5) speculated that closed head injury with traction injury of the vascular wall might be involved in the pathogenesis. Holland et al. 13) assumed that the presence of a defect of the medial layer of the intracranial meninges was a factor in the pathogenesis of PMMAs. Kimura et al. 4) confirmed the presence of a defect of the meningeal layer of the intracranial dura mater in a PMMA patient who developed intracerebral hemorrhage. These findings suggest that the presence of a defect of the meningeal layer of the intracranial wall might be a factor in the pathogenesis of PMMAs. Fig. 3  A 6Fr guiding catheter was guided into the left external carotid artery, and a 4Fr Cerulean was guided to the origin of the left MMA. (A) A Marathon was guided into the aneurysm, and three Barricade coils Complex Finish were inserted. (B) Embolization with 13% NBCA at 0.2 cc was performed, and the regurgitation of NBCA to the posterior convexity branch of the left MMA was observed. Confirmative angiography through a Roadmaster indicated the disappearance of the aneurysmal shadow. MMA: middle meningeal artery; NBCA: n-butyl-2-cyanoacrylate.

Fig. 4  (A) On the dura mater of the left temporal lobe, the NBCA-related dark-red, hypertrophic left MMA (arrow) was observed. (B) When incising the dura mater in a U-shape and reversing it, the left MMA (arrowhead) had submerged in the left temporal lobe. (C) When examining the lesion site after total hematoma removal, a pseudoaneurysm (arrow) involving a coil used during endovascular treatment was suspected at the site of adhesion between the MMA and brain parenchyma. MMA: middle meningeal artery; NBCA: n-butyl-2-cyanoacrylate.
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Fig. 5  EVG staining showed vascular-wall rupture and granulomatous blood clots with inflammatory cell infiltration, leading to a diagnosis of a pseudoaneurysm. Black arrow: Granulomatous blood clots with inflammatory cell infiltration, White arrow: Ruptured elastic plate. EVG: Elastica van Gieson

hemorrhage in the absence of fracture. In the patient presented by Lim et al.,5) the severity is also unclear, but it is described that the patient was transported to their hospital with head trauma related to a traffic accident. In our patient, a PMMA occurred in the absence of fracture, but the patient was severely injured in a falling accident, with pelvic fracture. Even if skull fracture is absent, severe high-energy trauma may induce PMMAs. Even patients without fracture must be followed-up after head trauma, considering the risk of PMMA development. Concerning the pathogenesis of PMMAs, hemostasis at the site of hemorrhage may be achieved by blood clots after MMA injury, contributing to PMMA formation through false-lumen formation related to subsequent hematoma fusion.4) The risk of PMMA rupture is high, and its outcome is unfavorable, with a mortality rate of 50%.14) Furthermore, the interval until rupture is 1–30 days; therefore, early preventive treatment is necessary.4) In the present case, there was no PMMA on arrival, but the presence of a PMMA could be confirmed at the time of its rupture (20 days after injury). Under a diagnosis of delayed PMMA formation/rupture, neuroendovascular treatment was performed to prevent re-rupture. Hematoma removal under craniotomy was conducted the following day, leading to a favorable course.

Concerning imaging procedures, in our patient, the PMMA could be confirmed for the first time on head CTA and cerebral angiography at the time of its rupture. Usually, delayed traumatic cerebral aneurysms may develop after head trauma, and follow-up using head MRA or CTA may be useful. However, PMMAs are rare, and much attention may not have been paid to their development in many cases. Montanari et al.11) recommended head CTA and DSA PMMA diagnosis. In their case report, a PMMA was clearly visualized on CTA and DSA images. In the present case, head CTA and DSA were performed after the onset of hemorrhage; the presence of a PMMA could be retrospectively indicated. Concerning CTA, the external carotid artery system may be frequently omitted at the time of image processing. However, if the possibility of PMMA development is suspected, detailed examination involving the external carotid artery system may be necessary. Park et al.15) reported a patient in whom the rupture of a traumatic PMMA caused a dural arteriovenous fistula, leading to cerebral parenchymal and traumatic subarachnoid hemorrhage. In this patient, the PMMA was also diagnosed using CTA and DSA. These procedures may be useful for detailed examination of traumatic vascular lesions following head trauma.

Montanari et al.11) indicated fracture and temporal hematomas at an area adjacent to the site of fracture as risk factors for PMMA formation. They recommended that close follow-up of neurological examination needed in patients with these risk factors. Evaluation of PMMA formation should be done within 7–10 days after head trauma using CTA. In our patient, there was no skull fracture, but head CT on arrival showed a small-volume hematoma above the temporal MMA. Even among patients without fracture, CTA and DSA should be performed a few days after injury in those with severe high-energy trauma. Thus, PMMA formation could be detected before the onset of hemorrhage.
Table 1  Previously reported patients with pseudoaneurysms of the middle meningeal artery complicated by intracerebral hemorrhage

<table>
<thead>
<tr>
<th>Case</th>
<th>Authors</th>
<th>Age (years)</th>
<th>Sex</th>
<th>A history of head bruise, symptoms on the initial consultation and at the time of rupture</th>
<th>CT on the initial consultation and at the time of rupture</th>
<th>Fracture</th>
<th>Treatment method</th>
<th>Outcome on discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bruneau et al.</td>
<td>64</td>
<td>Female</td>
<td>A history of head bruise was unclear. The patient was transported by ambulance with consciousness disorder (GCS 3).</td>
<td>ICH of the frontal and temporal lobes and intraventricular perforation on arrival</td>
<td>+</td>
<td>Craniotomy</td>
<td>mRS 6</td>
</tr>
<tr>
<td>2</td>
<td>Rambaugh et al.</td>
<td>63</td>
<td>Female</td>
<td>No description.</td>
<td>ICH of the right temporal lobe</td>
<td>+</td>
<td>Craniotomy</td>
<td>Good recovery</td>
</tr>
<tr>
<td>3</td>
<td>Lim et al.</td>
<td>70</td>
<td>Male</td>
<td>He had a history of head bruise related to a traffic accident. He was transported by ambulance with consciousness disorder (GCS 13) and hemiplegia.</td>
<td>ICH of the right temporal lobe on arrival</td>
<td>−</td>
<td>Craniotomy</td>
<td>Discharge on day 58 with a GCS score of 14</td>
</tr>
<tr>
<td>4</td>
<td>Bozzetto-Ambrosi et al.</td>
<td>39</td>
<td>Male</td>
<td>A history of head bruise was unclear. He was transported by ambulance with consciousness disorder (GCS 12) and hemiplegia.</td>
<td>ICH of the right temporal lobe on arrival</td>
<td>−</td>
<td>After craniotomy, a PMMA was indicated, and IVR (NBCA) was performed.</td>
<td>Discharge on day 5 with an improvement</td>
</tr>
<tr>
<td>5</td>
<td>Paiva et al.</td>
<td>33</td>
<td>Male</td>
<td>He was transported by ambulance with head bruise related to a traffic accident (GCS 13).</td>
<td>ICH of the left temporal lobe on arrival</td>
<td>+</td>
<td>IVR (NBCA) alone</td>
<td>Discharge with a GCS score of 15</td>
</tr>
<tr>
<td>6</td>
<td>Kumar et al.</td>
<td>53</td>
<td>Male</td>
<td>Falling down related to alcohol consumption. After 4 days, he consulted our hospital (GCS 12).</td>
<td>ICH of the right temporal lobe on arrival</td>
<td>+</td>
<td>Craniotomy</td>
<td>After a few days, he was discharged, with a GCS score of 14</td>
</tr>
<tr>
<td>7</td>
<td>Wu et al.</td>
<td>49</td>
<td>Male</td>
<td>He was transported by ambulance due to a traffic accident. After 14 days, vomiting and headache were noted at the time of rupture.</td>
<td>ASDH on arrival, ICH of the temporal lobe after 14 days</td>
<td>+</td>
<td>Craniotomy</td>
<td>A favorable prognosis was achieved, and he was discharged on day 25</td>
</tr>
<tr>
<td>8</td>
<td>Wu et al.</td>
<td>53</td>
<td>Male</td>
<td>He was transported by ambulance due to a traffic accident (GCS 10).</td>
<td>ICH of the left frontal lobe on arrival</td>
<td>+</td>
<td>Craniotomy</td>
<td>A favorable prognosis was achieved, and he was discharged. mRS 6</td>
</tr>
<tr>
<td>9</td>
<td>Montanari et al.</td>
<td>66</td>
<td>Male</td>
<td>He was transported by ambulance due to falling down from the stairs. Transient loss of consciousness and consciousness disorder (GCS 12) were observed. Paralysis of the right upper and lower limbs and consciousness disorder were noted 15 days after admission.</td>
<td>On arrival, slight hemorrhage of the left temporal region and traumatic subarachnoid hemorrhage were noted. ICH of the left temporal lobe occurred 15 days after admission.</td>
<td>−</td>
<td>After IVR (coil + NBCA), craniotomy was performed.</td>
<td>He was referred to a recovery-phase hospital on day 60, with an mRS score of 2.</td>
</tr>
<tr>
<td>10</td>
<td>Our case</td>
<td>73</td>
<td>Male</td>
<td>He was transported by ambulance due to a falling accident. Amnesia was noted. After 20 days, headache and aphasia were observed.</td>
<td>ASDH, TSAH on arrival, ICH of the left temporal lobe after 20 days</td>
<td>−</td>
<td>Craniotomy</td>
<td>He was referred to a recovery-phase hospital on day 60, with an mRS score of 2.</td>
</tr>
</tbody>
</table>

ASDH: acute subdural hemorrhage; GCS: Glasgow Coma Scale; ICH: intracerebral hemorrhage; IVR: interventional radiology; mRS: modified Rankin Scale; NBCA: N-butyl-2-cyanoacrylate; TSAH: traumatic subarachnoid hemorrhage; PMMA: pseudoaneurysm of the middle meningeal artery
According to previous case reports on PMMAs with intracerebral hemorrhage, craniotomy had been performed in seven of nine patients, whereas interventional radiology (IVR) alone and both had been conducted in two patients. The seven patients included two who died. However, the course was favorable in the patients in whom craniotomy was combined with IVR; the prognosis may be relatively favorable. When performing craniotomy alone, the intracranial pressure can be reduced in the early phase, but it may be difficult to manage rare PMMAs. Endovascular treatment combined with direct surgery may prevent PMMA re-rupture in the early phase; therefore, it may be useful if time permits. In our patient, preoperative imaging procedures suggested that the PMMA was located in the brain parenchyma adjacent to the skull base, and we considered frontotemporal craniotomy without endovascular treatment possible. However, PMMAs are rare, and hemostasis by IVR was initially performed, considering the advantage of safe re-rupture prevention and lifesaving. Subsequently, elective hematoma removal under craniotomy was conducted, considering the risk of late developing consciousness disorder related to hematoma compression. As hemostasis by IVR had been achieved, hematoma removal could be safely performed regardless of skills for craniotomy. There was no postoperative hemorrhage, leading to a favorable course. If the hematoma volume is small, differing from that involved in cerebral herniation, the combination of IVR and craniotomy may be effective.

No study has examined the pathogenesis of PMMAs with intracerebral hemorrhage. In the present case, head CT and CTA on arrival showed brain contusion, and head CTA at the time of rupture and intraoperative examination confirmed a pseudoaneurysm submerging in the left temporal lobe. Adhesion and blood-clot-related hemostasis may have occurred at the hemorrhagic site of the ruptured MMA and site of brain contusion, leading to PMMA formation in the brain parenchyma through subsequent hematoma fusion. PMMAs, as observed in the present case, are rare, but may develop. Their rupture may be prevented by indicating their presence using follow-up head CT, CTA, and DSA. Even among patients without skull fracture, follow-up should be continued in those with high-energy trauma, considering PMMA development.

### Conclusion

We encountered a patient in whom a PMMA increased during conservative treatment for traumatic subarachnoid hemorrhage, an acute left subdural hematoma, and brain contusion, resulting in intracerebral hemorrhage due to its rupture. As it is a rare external carotid artery lesion, it is difficult to predict its onset based on diagnostic imaging findings. However, recognizing PMMAs as a condition that may occur, follow-up by head CT, CTA, and DSA may be necessary. If PMMA rupture is observed, detailed cerebrovascular imaging examination should be promptly performed considering the risk of re-rupture. After diagnosis, early treatment is necessary. Direct surgery or its combination with endovascular treatment may be effective.

### Disclosure Statement

There is no conflict of interest for the first author and coauthors.

### References