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Case Report

Inpatient re-rupture of a middle meningeal arteriovenous fistula after traumatic brain injury ☆

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ABSTRACT

We present a case of a spontaneous second intraparenchymal hemorrhage (IPH) *following* patient admission for a traumatic brain injury with an initial traumatic IPH. After a subsequent review of all imaging, it was concluded that the patient had a traumatic middle meningeal associated dural arterial venous fistula (MMAVF) which re-ruptured during admission, and the MMAVF was overlooked as a potential contributor to the *initial* traumatic IPH for which the patient was admitted. A 49-year old man presented with right temporal IPH following an ATV accident and was found to have a right MMAVF on cerebral angiography. The MMAVF appeared on angiography to be unruptured, and therefore was not immediately treated. Later in admission, the patient suffered a new spontaneous IPH ipsilateral to the MMAVF, suggesting a re-rupture. Endovascular transarterial embolization with ethyl vinyl alcohol resulted in complete obliteration of the MMAVF. The patient tolerated treatment well and went on to make a good recovery as of last post-operative imaging at 8 months. Hence, MMAVFs may be present in the setting of IPH following a traumatic brain injury which warrants maintaining a high level of suspicion and low threshold for intervention as they can cause secondary spontaneous intracranial hemorrhage. The absence of notable subdural or extradural hemorrhage on imaging should not exclude rupture. Transarterial embolization with an ethylene vinyl alcohol copolymer is an effective treatment modality.

Introduction

Middle meningeal arteriovenous fistulas (MMAVFs) are relatively rare dural AVFs between the middle meningeal artery and a neighboring dural venous sinus or cortical veins. Prior case reports have showed varied etiologies and natural histories, with reports of both MMAVF spontaneous formation and spontaneous resolution [1–3]. However, to our knowledge, reports of definitive MMAVF re-rupture during a single admission are scarce. Regardless, as MMAVFs pose a risk of hemorrhage, careful evaluation in the context of patient history is necessary. Here, we present a case of a patient who we originally thought to have an unruptured MMAVF during a cerebral

Abbreviations: MMAVF, middle meningeal arteriovenou fistula; IPH, intraparenchymal hemorrhage; TBI, traumatic brain injury.
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angiogram following a traumatic brain injury (TBI), that later suffered a spontaneous second intraparenchymal bleed during the same admission. A second cerebral angiogram with embolization for treatment of the fistula was performed. Review of both sets of imaging revealed that the MMAVF was likely ruptured upon admission despite the lack of extradural or subdural hemorrhage, and that in retrospect the patient likely would have benefited from early treatment. Imaging, clinical course, including treatment, and implications are discussed.

Case description

A 49-year old male with unremarkable past medical history was brought to the emergency room after being found unresponsive at the scene of a rollover ATV accident. On initial presentation to the ED, his Glasgow coma scale score was 3T. Initial head and neck imaging demonstrated acute fractures of the right inferior temporal bone that extended along the right middle cranial fossa, right zygomatic arch nondisplaced fracture, nondisplaced fractures of the right second through seventh ribs, and a partially comminuted right clavicle fracture (Fig. 1).

Non-contrast computed tomography (CT) of the head demonstrated a 0.7 mL right anteroinferior temporal lobe IPH, petechial hemorrhage of the contralateral temporal lobe, and a small amount of acute intraventricular hemorrhage in the left ventricle (Fig. 2).

These hemorrhagic findings were initially attributed to direct traumatic brain injury. CT angiography of the head was performed without any associated abnormal findings. Still, neurointerventional service was requested to rule out any arterial disease, after which cerebral angiography showed presence of a small, and what we initially presumed to be, stable MMAVF draining into a dural vein without extravasation into the parenchyma (Fig. 3). Discussion with two other neurosurgeons led to the conclusion that the MMAVF did not necessitate emergent treatment.

Over the next 10 days, the patient made a good clinical improvement and was extubated and graduated out of the intensive care unit to the surgical ward. However, he was subsequently found fallen on the ground of his room and confused. He thusly had a repeat CT of the head that revealed interval development of a new 26 mL right temporal lobe IPH with associated extension of hemorrhage into the right sided ventricles (Fig. 4).

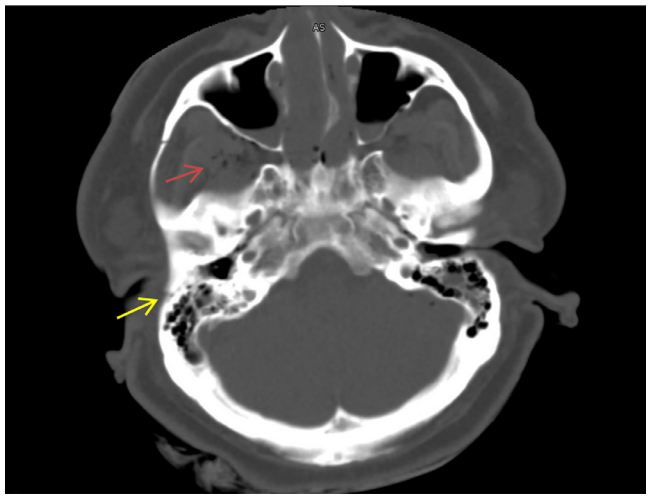

Fig. 1 – Axial CT head bone window of skull base showing fracture pattern at presentation with presence of air in middle cranial fossa, a common finding in skull base fractures (red arrow), and acute infero-temporal fracture (yellow arrow).

Another diagnostic cerebral angiography was performed that redemonstrated the finding of the right MMAVF involving the proximal intracranial segment of the middle meningeal artery, and also showed presence of a 7 mm × 6 mm venous varix (Fig. 5). The MMAVF showed early supero-lateral dural venous drainage along the dura into a cortical vein.

This was treated with endovascular transarterial embolization with ethyl vinyl alcohol (Onyx-34, Medtronic) with assistance of a Scepter C (Microvention) balloon microcatheter to prevent Onyx reflux. Embolization achieved complete obliteration of the MMAVF (Fig. 6).

The patient made a good clinical recovery and was eventually able to be discharged directly home from the hospital.

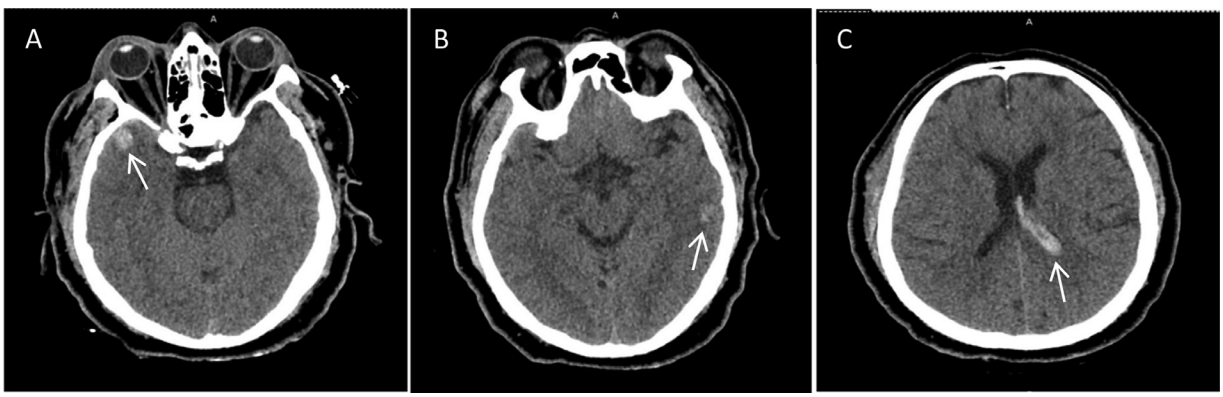

Fig. 2 – Initial non-contrast CT of the head showing anteroinferior right temporal lobe IPH (white arrow, A), petechial hemorrhage of left temporal lobe (white arrow, B), and intraventricular left ventricle hemorrhage (white arrow, C).

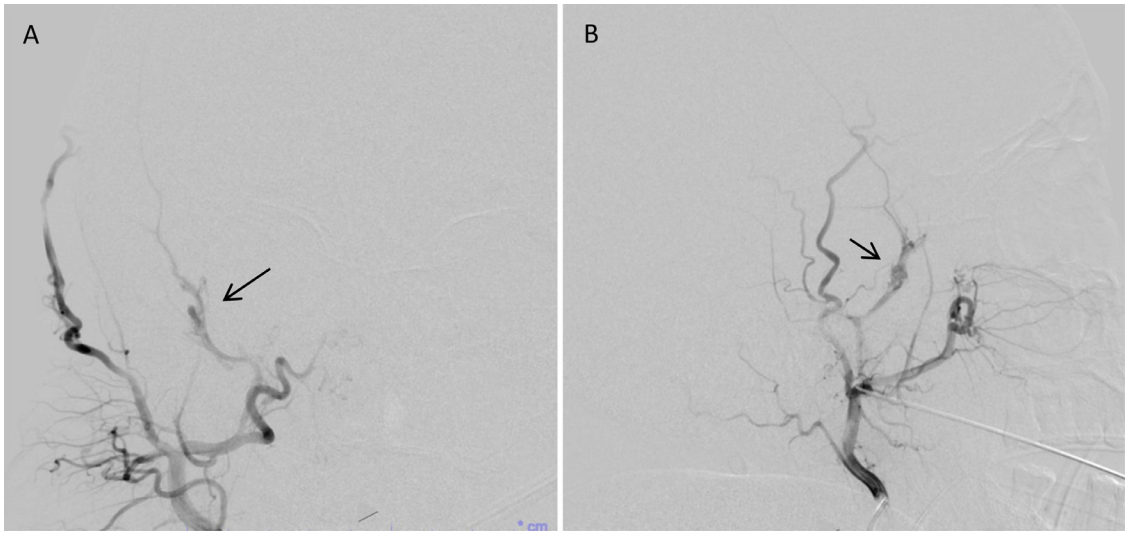

Fig. 3 – First cerebral angiogram in anterior-posterior (black arrow, A) and lateral (black arrow, B) views showing a right MMAVF that was presumed to be unruptured.

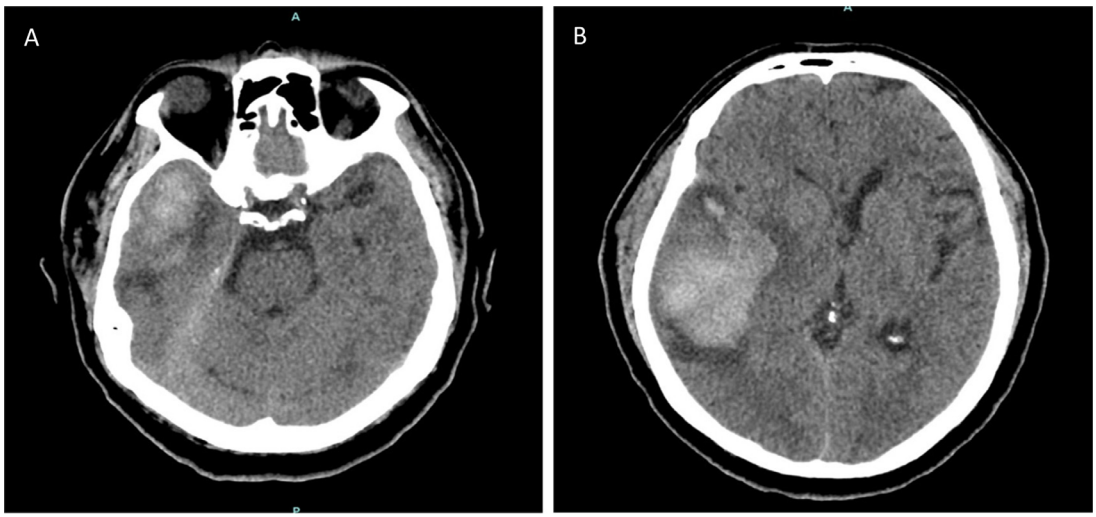

Fig. 4 – Second non-contrast CT of the head showing interval development of a new right temporal IPH (A) and impact on the right ventricles (B).

The patient's most recent postoperative imaging at 8 months showed no further abnormalities.

Discussion

We have reviewed a case of IPH due to MMAVF re-rupture during a single patient admission, and have provided one of the few reports with sequential cerebral angiograms illustrating a possible short-term evolution of MMAVFs. We believe we overlooked MMAVF rupture as a potential contributor to the patient's initial intracerebral bleed at admission following TBI. Although literature suggests that the patient's MMAVF likely developed secondary to the initial ATV trauma; it cannot be said for certain whether the MMAVF was posttraumatic or whether it pre-existed and then ruptured due to the initial ATV trauma, or even perhaps ruptured spontaneously and led to the ATV trauma. Regardless, our case report highlights that isolated IPH following trauma warrants maintaining a high level of suspicion of a ruptured MMAVF. Additionally, given the successful clinical recovery as of 8 months post-op suggests that patients may stand to benefit from early treatment with endovascular embolization.

Epidural and subdural hematomas are a common sequalae of traumatic brain injury, while the etiology of an isolated intraparenchymal hemorrhage can be more varied, including rupture of cerebral vascular malformations [4]. Although the lack of epidural or subdural hemorrhage in our patient is atypical, traumatic IPH can certainly occur with this pattern or

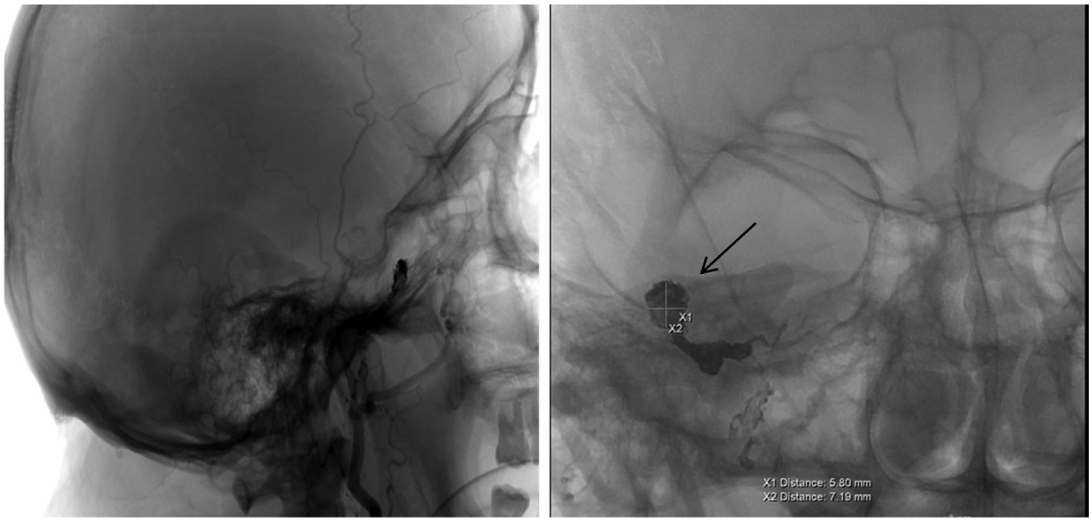

Fig. 5 – Cerebral angiogram showing an infero-anterolateral right MMAVF with a 7 mm x 6 mm venous varix (black arrow).

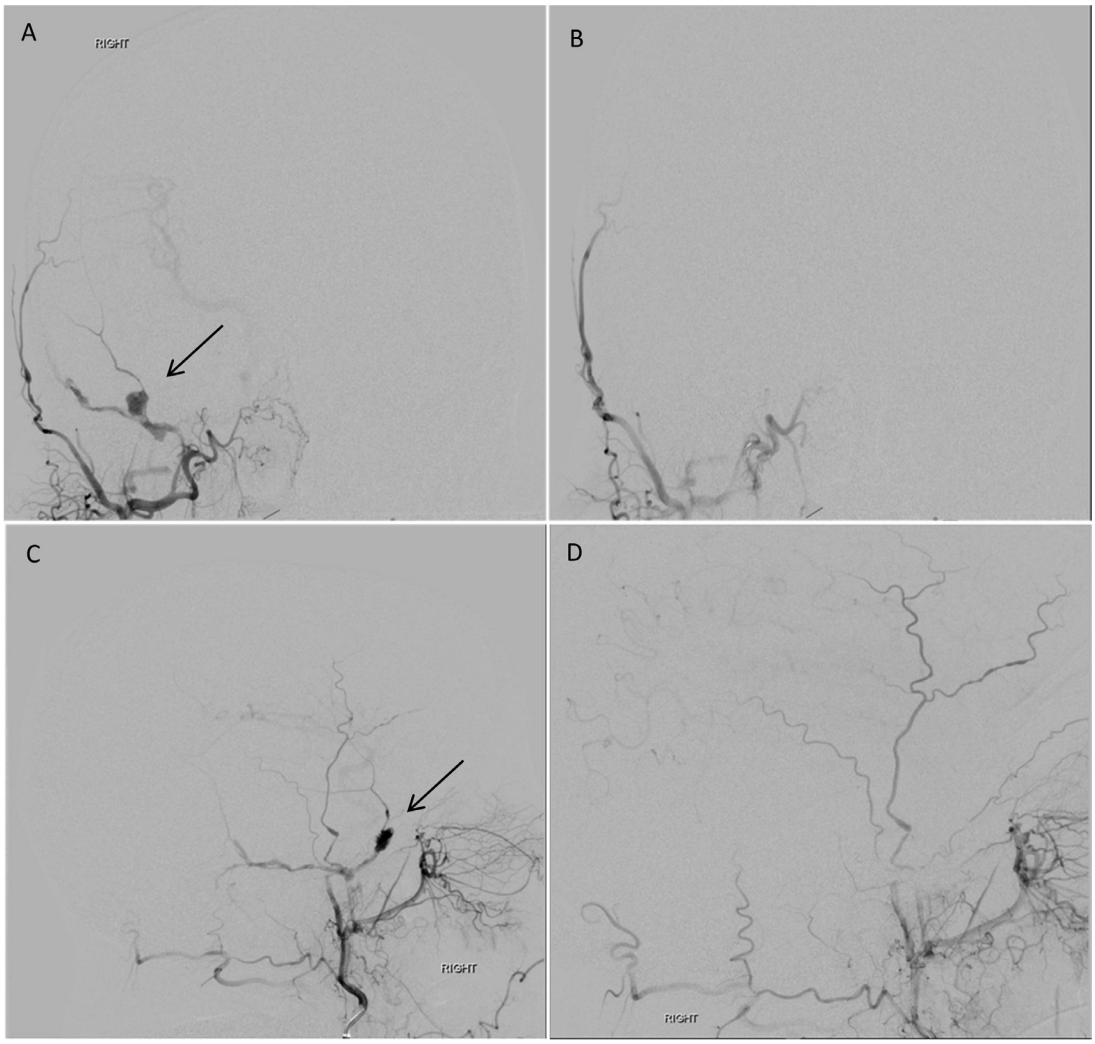

Fig. 6 – MMAVF pre-embolization (black arrow, A and C) and post-embolization (B and D) angiograms in anterior-posterior (A and B) and lateral (C and D) views showing complete obliteration of the MMAVF.

skull fracture and possible coup-contrecoup injury. Next, dural arteriovenous fistulas (dAVFs) account for almost 15% of all cerebral vascular malformations [5,6]. A dural AVF is an, often acquired, pathological connection with tributaries between a dural artery and a dural venous sinus or between a dural artery and cortical veins within the leptomeninges. Depending on the fistula anatomy, they can range from being benign to being prone to hemorrhage, particularly in variants that prevent normal venous outflow [7]. As a fistula's anatomical classification can change over time, treatment to prevent bleeding is still a matter of some debate.

A middle meningeal AVF is a type of dural AVF with the main feeding tributaries directly from the middle meningeal artery. The middle meningeal artery runs along the superficial layer of the dura mater, while dural venous sinuses lie between the endosteal and meningeal layer of the dura mater. Thus rupture of a MMAVF can present with a variety of intracranial hemorrhage types, complicating clinical diagnosis. Furthermore, MMAVFs can occur secondary to trauma as reported in about 2% of cases, or develop spontaneously and identified incidentally [1]. Nonetheless, these lesions have been described as having high potential for hemorrhage in majority of case reports thus far. CT angiography may not provide sufficient resolution to identify fistulae without large drainage or those in initial stages of formation, thus 6-vessel cerebral angiography is warranted for complete investigation [8].

The majority of high-risk dAVFs, including MMAVFs, are now treated endovascularly through transarterial embolization, and occasionally through transvenous embolization. Surgery, and rarely radiosurgery can also be employed for lesions not amenable, or refractory to endovascular treatment [5].

Nazari et al. and Tavakkoli et al. have reported both spontaneous thrombosis and spontaneous formation of MMAVFs respectively, both case reports advocating for close patient follow-up and prompt treatment once a MMAVF is detected. Moreover, Almefty et al. provided a case illustration where an untreated MMAVF resulted in pseudoaneurysm formation and rupture approximately 2 weeks after MMAVF diagnosis. In our case we had no pseudoaneurysms, no subarachnoid or epidural hemorrhage, and a much shorter interval between MMAVF diagnosis and IPH hemorrhage. Therefore, our case underscores these findings and recommends MMAVFs be considered a high-risk dAVF necessitating prompt treatment.

The present case demonstrated how traumatic MMAVFs can present with IPH with lack of a notable subdural or extradural hematoma. Further, spontaneous thrombosis with complete healing of a MMAVF is rare and serial angiography is recommended [9]. Thus, we add to the existing literature that patients with IPH in the setting of a traumatic MMAVF stand to benefit from early treatment with transarterial embolization using a liquid embolic.

Conclusion

Although trauma can result in de novo formation of MMAVF, the lack of notable extradural or subdural hemorrhage should not preclude rupture of a preexisting or newly formed MMAVF. Untreated lesions may result in secondary spontaneous intracranial hemorrhage. Given that the MMA is readily accessible via endovascular approaches, embolization of the MMAVF is a safe and effective treatment option.

Patient consent

This is to confirm that written, informed consent for publication of our case report "Inpatient re-rupture of a middle meningeal arteriovenous fistula after traumatic brain injury" was obtained from the patient(s).

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