

Traumatic Intracranial Aneurysm Presenting with Delayed Subarachnoid Hemorrhage

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Traumatic intracranial aneurysm rarely occurs after a head injury. The authors report a case of a 51-year-old man in whom subarachnoid hemorrhage was developed as a result of delayed traumatic aneurysmal rupture of the distal portion of the middle cerebral artery following a minor, closed-head injury. The unruptured aneurysm had been evident on the magnetic resonance image taken two days prior to onset of the subarachnoid hemorrhage. The clinical presentation and possible underlying mechanism are discussed with a review of pertinent literature.

KEY WORDS : Aneurysm · Trauma · Subarachnoid hemorrhage.

Introduction

Traumatic intracranial aneurysms are uncommon sequelae of head injury, constituting only 0.15 to 0.4 % of all intracranial aneurysms. They are most commonly the result of a closed head injury, a penetrating injury, or may be iatrogenic^{1,16}. Traumatic intracranial aneurysms are most commonly located in the anterior circulation, involving the internal carotid artery or the middle cerebral artery (MCA)^{1,8,10}. When they rupture, they present with subarachnoid, intraparenchymal, or intraventricular hemorrhage. Less commonly, they result in subdural hematoma^{11,14}. The interval from injury to diagnosis of a traumatic aneurysm varies from several hours to several weeks and, in rare occasions, several years^{5,16}.

We describe a rare case of a mixed-type traumatic intracranial aneurysm of the distal MCA, presenting with delayed subarachnoid hemorrhage (SAH) that occurred six months after a minor, blunt head injury.

Case Report

History and Cranial Imaging

A fifty-one-year-old man was admitted with severe headache and nuchal rigidity lasting for two days. Six months prior,

he had been in a motor vehicle accident and experienced loss of consciousness for a few seconds. After that time, he suffered from persistent, chronic headaches. Just after head injury, a non-enhanced computed tomography (CT) scan of the brain showed no evidence of aneurysm or SAH (Fig. 1). Upon admission, the neurological examination was positive for neck stiffness and Kernig's sign. When he experienced an abrupt onset of severe headache two days before admission, there was no evidence of hemorrhage. However, magnetic resonance imaging (MRI) revealed a small,



Fig. 1. Axial computed tomography scan, which was taken six months ago immediately after head injury, revealing no evidence of intracranial hemorrhage.

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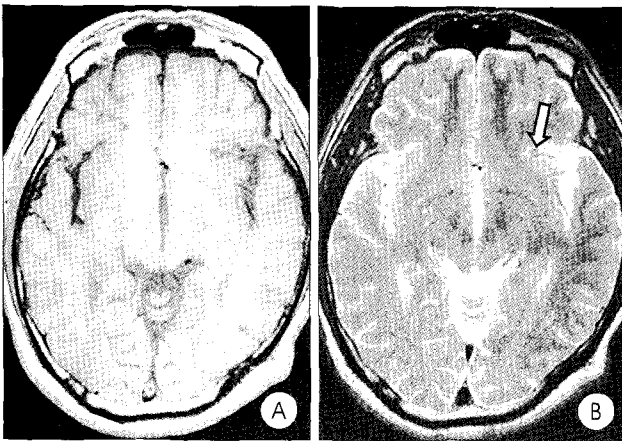


Fig. 2. Preoperative axial T1-weighted magnetic resonance image (MRI) (A), obtained two days before admission and revealing no evidence of intracranial hemorrhage; however, axial T2-weighted MRI (B) reveals a small, ovoid signal-void density on the left distal middle cerebral artery (arrow).



Fig. 3. Preoperative axial computed tomography scan showing a thick hemorrhagic density in the basal cisterns and both sylvian fissures.

round, signal-void lesion on the vascular tree of the left MCA (Fig. 2). At admission, the CT scan revealed a thick, hemorrhagic density at the basal cistern and both sylvian fissures (Fig. 3). Three-dimensional (3-D) CT angiography confirmed the presence of a saccular aneurysm along the M2 segment of the left MCA (Fig. 4).

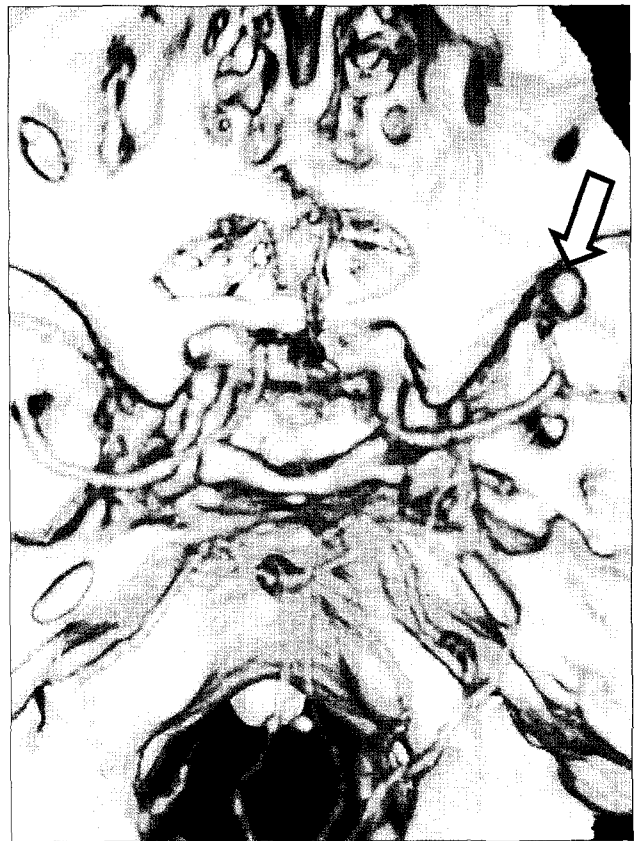


Fig. 4. Preoperative 3-dimensional computed tomographic angiography demonstrating a saccular aneurysm (arrow) on the non-branching portion of the left distal M2 closed to the bony ridge of the sphenoid.

Postoperatively, the patient's recovery was uneventful.

Postoperative 3-D CT angiography showed complete obliteration of the aneurysm with no compromise of the parent circulation (Fig. 6). He was discharged without neurological deficits and has returned to his work.

Pathological Findings

Histopathological examination of an avulsed, thrombosed mass revealed a typical traumatic aneurysm, consisting of fibroblasts and inflammatory cells with thrombi. The resected aneurysm wall contained none of the normal arterial wall structures covered with organized thrombus and fibrous tissue (Fig. 7).

Discussion

Traumatic intracranial aneurysms were first described by Guibert in 1895, and they are commonly associated with major head injuries^{9,11,16}. However, traumatic aneurysms caused by minor head injuries are rarely reported^{13,14}. Traumatic, intracranial aneurysms are predominantly located in the anterior circulation and commonly occur in the distal cerebral vascular

Operation and Postoperative Course

A left posterior pterional craniotomy was performed for obliteration of the aneurysm from the cerebral circulation. Dissection of the distal sylvian fissure easily exposed a friable, dark-colored aneurysm, measuring about 4 × 6 mm on the unbranched portion of the left M2 segment. Half of the sac was buried in the frontal lobe (Fig. 5A). Fortunately, the temporal branch, and the lateral lenticulostriate artery originated from the side opposite the origin of the aneurysm. After dissection of the neck of the aneurysm, two straight clips were applied to the neck of the aneurysm. After applying the clips, the distal thrombosed hematoma was pushed out from the neck immediately (Fig. 5B).

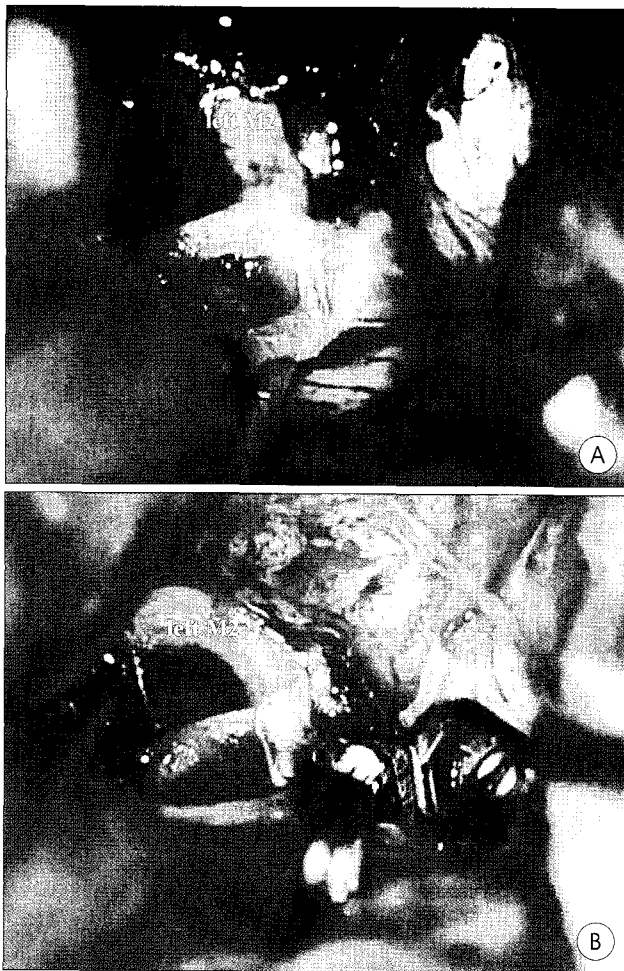


Fig. 5. Intraoperative photographs, demonstrating a friable, pseudo-capsulated and dark-colored aneurysmal sac before clipping (A), and autoamputated aneurysmal sac after neck clipping (B).

tree. The most frequent sites are the peripheral branches of the MCA, followed by branches of the pericallosal artery¹⁰.

The development of a traumatic aneurysm is believed to be caused by a direct injury to the arterial wall. However, indirect injury can produce a traumatic aneurysm as a result of the different velocities of the brain and skull. The brain and its vessels may strike bony or connective tissue protuberances such as the falx, tentorium, or sphenoid ridge¹⁵. In our case, the traumatic aneurysm occurred on the distal portion of the MCA after a minor, blunt head injury. The aneurysm may have been the result of axial rotational movement of the brain at the moment of impact, causing trauma to the MCA, which might have struck the sphenoidal ridge.

The time between the occurrence of trauma and the diagnosis of a traumatic aneurysm varies from several hours to several months and, on rare occasions, several years^{5,12,16}. However, most of the presentations occur within 2-3 weeks of injury¹². CT or CT angiography have replaced cerebral angiography as the first line of neuroradiological procedure in the acute

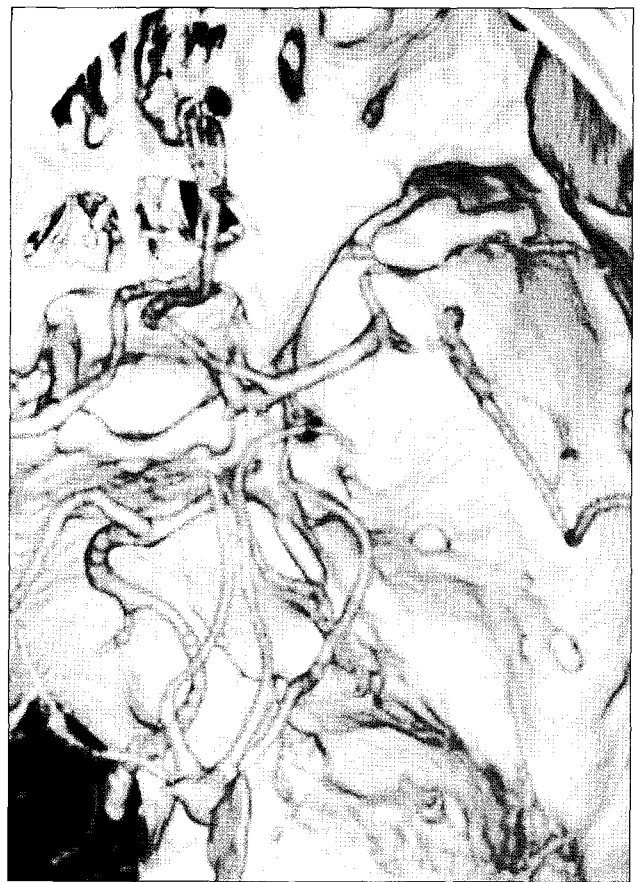


Fig. 6. Postoperative 3-dimensional computed tomographic angiography demonstrating applied clips and no visualization of remnant aneurysm sac.

phase of head injury. Because CT scans do not reveal vascular structures directly, the early detection of traumatic aneurysm may be delayed. In our case, six months have elapsed between the head injury and detection of the aneurysm. Such a long delay is unusual. The angiographic hallmarks of traumatic aneurysms include delayed filling and emptying of the aneurysm, irregular shape, unusual location, and a poorly defined neck^{5,15}. We performed MRI before aneurysm rupture and CT angiography after the SAH. The 3-D CT angiographic findings showed an aneurysm on the unbranched side of the distal MCA, located very close to the sphenoid ridge.

Histologically, traumatic aneurysms have been classified by Burton according to the types of vascular trauma and types of aneurysms produced. The classifications include true aneurysm (partial disruption of arterial wall), false aneurysm (cavity of encapsulated hematoma communicating with the lumen of the artery) and mixed aneurysm (rupture of the true aneurysm, giving rise to a secondary false aneurysm)⁴. In our case, there was a disruption of the full-thickness of the arterial wall, and this was occluded by a blood clot. Subsequent fibrous organization and dynamic excavation of a hematoma resulted

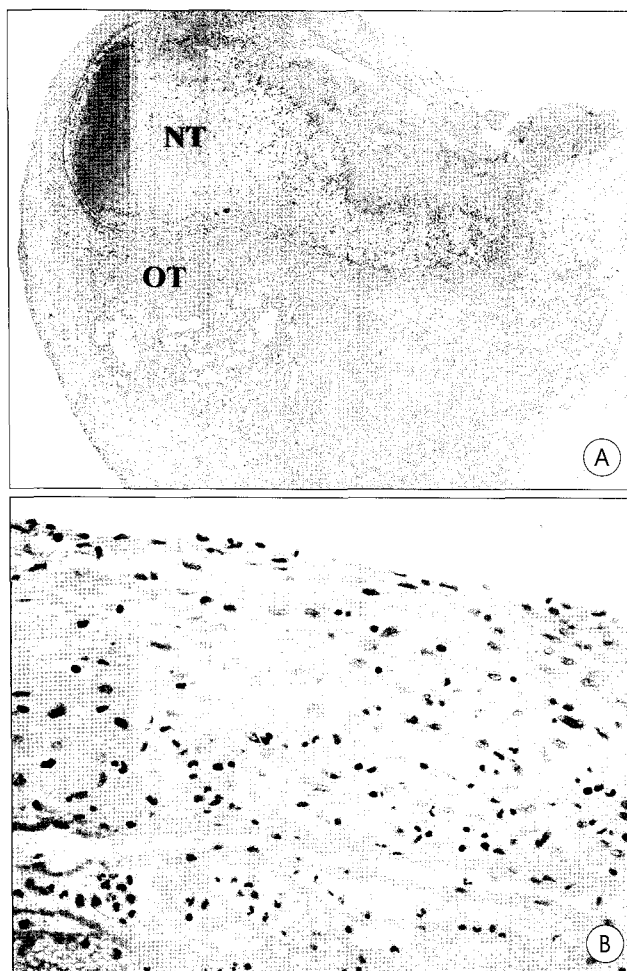


Fig. 7. Photomicrographs of the surgical specimen, demonstrating the pseudoaneurysm composed of the non-organized thrombus (NT), absence of endothelial lining cells, and the false aneurysm wall. The aneurysm wall consisted of organized thrombus (OT), chronic inflammatory cells, and fibroblasts. H & E. original magnification, 40x (A), 400x (B).

in the rupture of the aneurysm. The aneurysm was a mixed-type, which had a thin arterial wall of the neck region, and the remaining part was covered by organized thrombus and fibrous tissue.

The treatment of choice for traumatic aneurysms is surgical elimination by clip ligation, trapping, or endovascular embolization^{2,6,7}. Traumatic aneurysms usually have no neck or a broad neck. In false aneurysms, there is no definite aneurysmal wall. Treating traumatic aneurysms by clipping may be impossible, and alternative surgical strategies may be required^{2,3,7,11,16}. We didn't expect the possibility of traumatic aneurysm prior to the surgery. Because of the successful eradication of the aneurysmal sac with clipping technique,

we didn't consider about the by-pass surgery. The mortality rates following rupture of traumatic aneurysms have been reported to be between 31% and 54%^{1,3,4}. However, in surgically treated patients, the mortality rates are much lower and range from 18% to 20%^{3,8}. Therefore, prompt and definitive treatment is essential once the diagnosis is made.

Conclusion

A mixed type traumatic aneurysm of the distal MCA, presenting with delayed SAH, is rare. The possibility of a traumatic aneurysm should be considered when new symptoms develop in a patient with even minor head trauma. Early surgical intervention often yields improved outcomes.

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