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Traumatic aneurysm at the superior cerebellar artery: illustrative case

Mun-Chun Yeap, MD,[1] Meng-Wu Chung, MD,[2] and Chun-Ting Chen, MD[1]

Departments of [1]Neurosurgery and [2]General Medicine, Chang Gung Memorial Hospital, Taoyuan City, Taiwan

BACKGROUND Traumatic aneurysms at the superior cerebellar arteries after head injury are extremely rare and may be overlooked. Rupture of these aneurysms can cause fatal intracranial hemorrhages; thus, early identification of the entity helps prevent detrimental outcomes.

OBSERVATIONS A patient suffered from sudden severe headache and decreased consciousness level several weeks after a blunt head injury. He received surgery to remove a progressive enlarging subdural hematoma. The diagnosis of a traumatic aneurysm at the superior cerebellar artery was delayed, made only after a recurrent subdural hemorrhage occurred. He received another surgery to obliterate the aneurysm.

LESSONS The patient could have been treated earlier if traumatic aneurysm had been suspected in the beginning. In addition to the case, the authors also reviewed the literature to clarify the pathophysiology, clinical presentation, diagnosis, and management of the disease.

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KEYWORDS traumatic aneurysm; superior cerebellar artery; delayed hemorrhage

Intracranial traumatic aneurysms (TAs) are rare, accounting for only 1% of all intracranial aneurysms, and are predominantly located within the anterior circulation, especially at the anterior cerebral arteries.[1–6] Meanwhile, only ~10% of TAs are found in the posterior circulation.[7–9] Either being nonsymptomatic or presenting with hemorrhage late after injury, the diagnosis of TA is often delayed, sometimes leading to devastating consequences.[7] So far, there is a paucity of reports of TA found at the superior cerebellar artery (SCA).[1,8,10–12] We hereby report a lesson learned from a case of SCA-TA after head injury. We also review the literature and discuss the diagnosis and treatment of this entity. We believe that better understanding of the pathophysiology and clinical presentations of TA helps avoid clinical pitfalls.

Illustrative Case

A 59-year-old man with hypertension and type 2 diabetes mellitus fell while walking and sustained blunt head injury. After the incident, he was brought to our hospital, where brain computed tomography (CT) showed mild acute subdural hemorrhage (SDH) at the left convexity and tentorium. Surgery was not indicated then because of no neurological deficit. However, he had persistent left-sided headache. Ten days later, sudden unbearable headache, nausea, vomiting, and gradual decrease level of consciousness were noted. His Glasgow Coma Scale (GCS) score was E2V3M5. CT revealed an increased amount of hyperdense subdural hematoma. He received emergency craniotomy for hematoma evacuation. Postoperatively, he regained clear consciousness but still complained of frequent headache. Repeat CT on postoperative day 2 showed residual subdural hematoma at the right tentorial area. On postoperative day 9, his GCS score again deteriorated to E1V1M5. CT revealed acute intracerebral hemorrhage (ICH) posteriorly at the base of the left temporal lobe (Fig. 1C). Cerebral angiography was arranged for this inexplicable occurrence of ICH, and a small outpouching lesion originating from left SCA was seen located superior and medial to the left tentorium edge. The lesion had an irregular shape with a delayed filling and emptying phenomenon, suggesting a traumatic SCA pseudoaneurysm (Fig. 1D and E). Recraniotomy was performed. Intraoperatively, an irregularly shaped saccular lesion was identified arising from a distal SCA branch (Fig. 1F). The lesion was excised and histologically revealed as a false aneurysm formed by organized hematoma (Fig. 2). Postoperatively, the patient regained consciousness and was eventually discharged without neurological deficit except aphasia. The

ABBREVIATIONS CT = computed tomography; ICH = intracerebral hemorrhage; SAH = subarachnoid hemorrhage; SCA = superior cerebellar artery; SDH = subdural hemorrhage; TA = traumatic aneurysm; TBI = traumatic brain injury.

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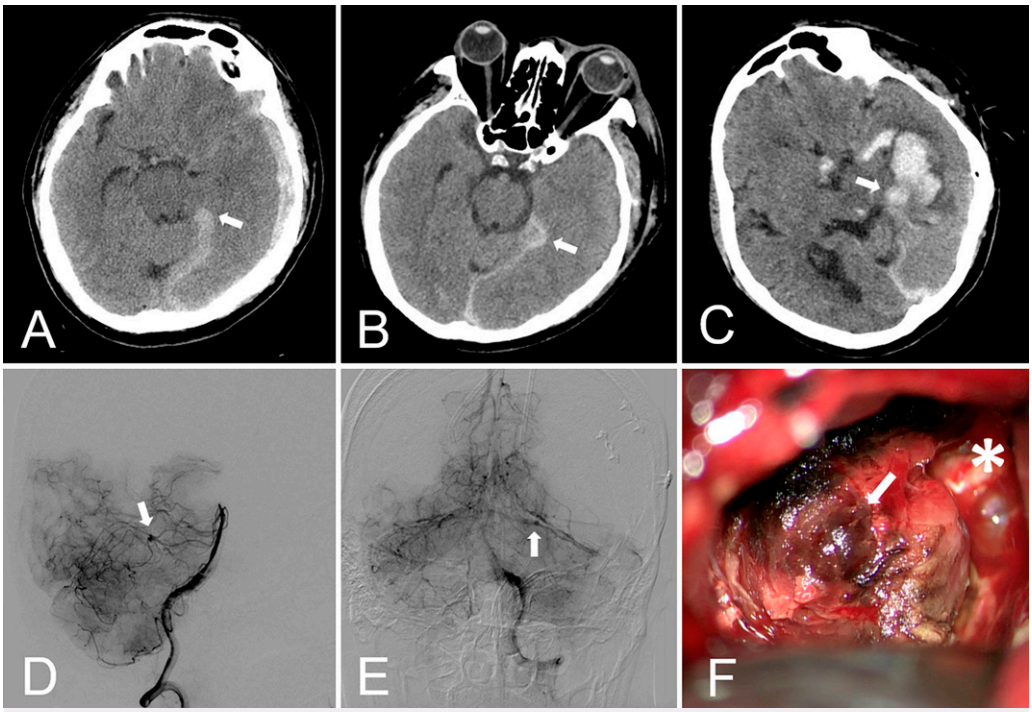

FIG. 1. Diagnostic assessments of the case. Cerebral CT showed progressive SDH at (**A**) 10 days after the fall and (**B**) 2 days and (**C**) 9 days after the first craniotomy. The aneurysm presented as a hyperdense lesion adjacent to the tentorial edge (*arrows*). **D and E:** Cerebral angiography showed an aneurysm with delayed contrast filling and emptying (*arrow*) at the left distal SCA. **F:** Intraoperative finding of the aneurysm (*arrow*) adjacent to tentorial edge (*asterisk*).

informed consent to treatment was signed by a family member on behalf of the patient.

Discussion

Intracranial TAs are aneurysms secondary to traumatic brain injury (TBI), accounting for $<$1% of all intracranial aneurysms.[3] TAs are usually pseudoaneurysms resulting from disruption of arterial walls and subsequent organization of surrounding hematomas.[13] Commonly seen in younger patients,[3,14] 90% of TAs are found within the anterior circulation, making these aneurysms a rare entity at the posterior blood vessels.[5] SCA-TAs are even rarer, with only a few case reports available.[15] To obtain a better understanding of SCA-TA, we reviewed the literature and compared the parameters such as patient characteristics, clinical presentations, management, and treatment outcomes. The results are summarized in Table 1.

Frequently associated with closed head injuries, ruptured TAs can go unnoticed and eventually cause massive intracranial hemorrhage. Overall, the mortality rate can be as high as 30% to 50% if left untreated.[3] However, this figure could be underestimated when one considers the misdiagnoses due to incomplete imaging studies.[16]

Several mechanisms have been proposed for the pathophysiology of TA. Penetrating injuries such as bone fragments or weapons may potentially cause direct injury to blood vessel walls.[17] High impact blunt injury may result in the vessels being lacerated or contused by adjacent bony structure.[18] TA can also be the result of forceful stretching or avulsion by rigid dural edges during rapid-deceleration head injury.[4,11] These mechanisms may have attributed to the higher incidence of TA within the anterior circulation because of the juxtaposed anterior falx and anterior cerebral arteries. Our patient suffered from a SCA-TA, which we believed was probably due to the latter mechanism, considering the proximity of the

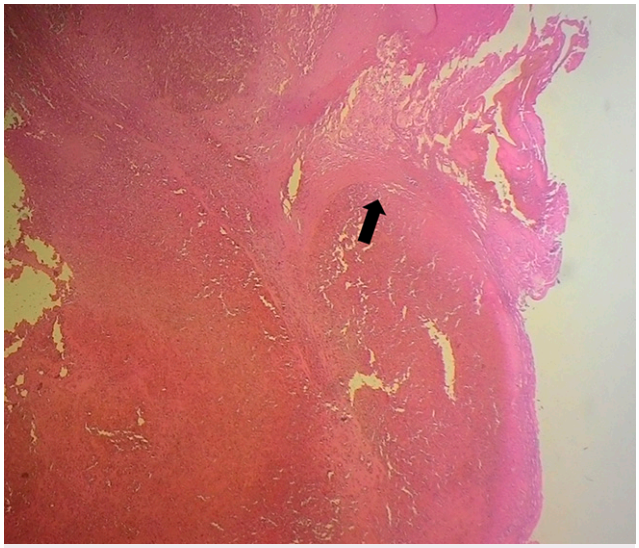

FIG. 2. Pathological findings of the resected aneurysm. Hematoxylin and eosin stain revealed an arterial wall defect (*black arrow*) surrounded by hematoma, inflammatory cells, and connective tissue. Original magnification $\times$100.

TABLE 1. Summary of literature review on traumatic aneurysms located at the SCA

Authors & Year	Age (yrs), Sex	Injury Mechanism	Location at SCA	Diagnostic Imaging	Symptom	Timing of Symptom After Injury	Radiographic Hemorrhage	Treatment	Outcome
Ferry & Kempe, 1972[13]	23, M	Penetrating	Left	Angiography	Facial pain, diplopia, audible bruits	Immediate	NA*	Surgical ligation of SCA	Mild right hemiparesis due to surgical complications
McDonald et al., 1976[23]	44, M	Penetrating	Right distal	Angiography	Lethargy, left hemiparesis	Hours	NA*	Conservative	Dense left hemiparesis
Cockrill et al., 1977[1]	15, M	Blunt	Right distal	Angiography	Headache, diplopia, ataxia	6 yrs	NA*	Surgical clipping	Diplopia, improved ataxia
Quattrocchi et al., 1990[12]	26, M	Penetrating	Left proximal	CTA, angiography	GCS 4	Immediate	Basal cistern SAH; recurrent IVH	EVD	Mortality
Amirjamshidi et al., 1996[24]	23, NA	Penetrating	Left	Angiography	NA	NA	NA	Conservative	Spontaneous aneurysmal healing
Proust et al., 1997[11]	22, F	Blunt	Right proximal	CT, MRI, angiography	Headache	15 days	No hemorrhage; nodular lesion at brainstem cistern	SCA trapping	Asymptomatic
Gjertsen et al., 2007[10]	40, M	Blunt	Left proximal	CT, CTA, angiography	GCS 3, left anisocoria	1 day	Basal cistern SAH	EVD + coiling	GCS 3
Ong et al., 2010[7]	3, M	Blunt	Left superior vermian branch	CTA, angiography	Nonarousable	2 wks	Basal cistern SAH	Parent artery occlusion with Onyx	Mortality
Paiva et al., 2012[8]	31, M	Blunt	Left distal	CT, angiography	Decreased GCS from 11 to 7, left anisocoria	Immediate	Basal cistern SAH, IVH, cerebellar ICH	Endovascular occlusion; ventricular shunt	GOS 3
Present study	59, M	Blunt	Left distal	CT, angiography	Headache, decreased GCS to 7	19 days	SDH, then IVH + ICH	Aneurysm excision	Aphasia

CTA = CT angiography; EVD = extraventricular drainage; GOS = Glasgow outcome scale; IVH = intraventricular hemorrhage; NA = not available.
* Pre-CT era in which angiography was the main diagnostic method.

aneurysm and tentorial edge. Anatomically, some SCA branches may pass through the perimesencephalic cistern and course by the posterior tentorial edge.[19,20] These branches may give off supplying arteries to the dura mater, and these small arteries are vulnerable to being injured by the adjacent rigid tentorium.[21] Laceration or rupture of these blood vessels may thus result in SAH, SDH, or even ICH prominently in the posterior cerebrum.

Our results demonstrated that the mortality rate of SCA-TA was 25% (Table 1). Of note, the fatalities were associated with poor initial consciousness. For the remaining patients who survived, the symptoms varied, with the most common being cranial nerve palsies followed by headache. The timing of symptom development was either immediate or delayed, ranging from hours to 6 years. These findings are comparable to TAs of other locations reported in literature, in which aneurysmal hemorrhages most frequently occur at 1 to 3 weeks (average 21 days) after injury but can be as long as years after.[2,3,5,15]

Radiographically, TAs of proximal major arteries usually present with SAH, whereas distal aneurysms are frequently associated with ICH.[22] Based on our review, SCA-TA frequently demonstrated SAH, probably because the location of vascular injury is within the perimesencephalic cistern, as described above. These SAHs in turn may result in cranial nerve palsies. Proust et al. reported a patient who had symptoms even without hemorrhage.[11] Our patient had constant headache, and a careful retrospective review of his CT without contrast showed a rounded hyperdense lesion at the left tentorial edge adjoining the hematoma. The lesion was easily

overlooked because of its resemblance to hematoma density. Thus, for patients who suffer from persistent and inexplainable symptoms, normal noncontrast CTs warranted additional examinations.

Cerebral angiography is the diagnostic procedure of choice because TA demonstrates as an irregular outpouching arising from a nonbranching arterial site. Besides, the classic characteristic of TA is filling late in arterial phase and emptying slowly.[11] In fact, during the pre-CT era, when angiograms were used as first-line surveys, TAs were diagnosed early.[10,16] Currently, noncontrast CT is instead routinely used to screen TBI; thus, TA tends to be overlooked at primary surveys. Our review of the older reports showed that TAs are probably present immediately after injury.[1,13,23] This is in contrast to some findings that TAs take time to develop and are invisible if cerebral angiography is performed too early (within 3 days).[3,16] There are also suggestions that angiography is best performed 2 weeks after injury.[24] One should be aware of several indications for angiographic evaluation after TBI: unexplained neurological deficits, cranial base fracture, penetrating injuries, delayed neurological deterioration, or delayed intracranial hemorrhages.[1,25] Alternatively, CT angiography is a speedy diagnostic option with good accessibility that should be used if the diagnosis is in doubt.[3,23,25] As in our case, an SDH still in its acute stage 2 weeks postinjury should have raised suspicion.

Although spontaneous healing of TA has been reported,[24] appropriate treatment prevents catastrophic outcomes.[9,26] If the aneurysms are located at accessible areas, surgical resection or clipping provides the highest obliteration rate, as in our case in which the aneurysm was readily visible and surgically approachable after evacuation of hematoma. Meanwhile, bypass surgery can be performed in case of difficult accessibility. Endovascular interventions such as aneurysmal embolization, parent arterial trapping, and covered stent placement are also safe options when available.[4,16,27,28] In literature, patients with SCA-TA have been treated surgically or via endovascular methods. Nevertheless, most patients still suffered from certain degrees of neurological deficits, with some being disabled (Table 1). Our patient received surgical excision of the aneurysm and survived with aphasia as sequela.

Observations

The clinical course of delayed hemorrhage associated with SCA-TA was described in this patient. The patient suffered from sudden severe headache and decreased level of consciousness 10 days after head injury. His cerebral CT then did not demonstrate SAH, which would have otherwise hinted at vascular insult. Instead, we overlooked the event and treated the patient under the impression of merely an enlarging hematoma. Only when recurrent hemorrhage occurred did we perform cerebral angiography for clarification.

Lessons

Traumatic aneurysms located at the superior cerebellar arteries are rare but can lead to fatal and catastrophic outcomes if misdiagnosed and untreated. These aneurysms can rebleed any time after head trauma. Physicians should be aware of any unusual clinical presentation or image finding after traumatic head injury; when in doubt, the threshold for performing cerebral angiography should be low.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Chen. Acquisition of data: all authors. Analysis and interpretation of data: Yeap, Chung. Drafting the article: Yeap, Chung. Critically revising the article: Chen. Reviewed submitted version of manuscript: Chen. Approved the final version of the manuscript on behalf of all authors: Chen.

Correspondence

Chun-Ting Chen: Chang Gung Memorial Hospital, Taoyuan City, Taiwan. b9002055@cgmh.org.tw.