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[Case Reports]

Acute Traumatic Posteroinferior Cerebellar Artery Aneurysms: Report of Three Cases

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Abstract

OBJECTIVE AND IMPORTANCE: Posterior fossa subarachnoid hemorrhage secondary to blunt head trauma is rarely associated with traumatic aneurysms of the posterior circulation.

CLINICAL PRESENTATION: We present three cases of posterior fossa subarachnoid hemorrhage from ruptured posteroinferior cerebellar artery (PICA) aneurysms after blunt head trauma. In each case, there was no associated penetrating injury or cranial fracture. All three patients presented with acute hydrocephalus requiring ventriculostomy. Two of the three patients had a proximal PICA aneurysm visible on emergent angiography. The remaining patient's aneurysm, although not visible on his initial angiogram, was detected on a subsequent angiogram 72 hours later.

INTERVENTION: All patients underwent successful surgical clipping of their aneurysms. Two cases required sacrificing of the parent vessels because of the friable nature of the false aneurysms. In each case, severe symptomatic vasospasm occurred, requiring angioplasty. All three patients also required a ventriculoperitoneal shunt for persistent hydrocephalus.

CONCLUSION: Features of these three cases and similar cases reported in the literature support the theory that vascular ruptures and traumatic aneurysms of the proximal PICA may be related to anatomic variability of the PICA as it transverse the brainstem. This variability predisposes individuals to vascular lesions, which occur in a continuum based on the severity of the injury. Posterior fossa subarachnoid hemorrhage after head injury requires a high index of suspicion and warrants aggressive diagnostic and therapeutic interventions.

Trauma continues to be the most common cause of subarachnoid hemorrhage (SAH); however, acute traumatic intracranial aneurysms occur very rarely (1%) (7). These aneurysms are most frequently associated with penetrating trauma or vessel injury from a contiguous cranial fracture (7). There have been several reports of distal posteroinferior cerebellar artery (PICA) aneurysms associated with displaced occipital or clival cranial fractures resulting in SAH and requiring surgical repair (10, 11, 14). Blunt head trauma has also been associated with rupture of the PICA and/or vertebral artery that resulted in rapid death (2, 4, 6). Subarachnoid hemorrhage associated with PICA dissection is also well documented (1, 9, 13). These dissections may occur with minimal or no trauma (9) and may involve a predisposing vascular pathology, such as fibromuscular dysplasia.

We present three cases of acute SAH from PICA aneurysms associated with blunt trauma in the absence of associated cranial or cervical fractures. All three patients underwent successful surgical treatment of the aneurysms. All three patients developed severe symptomatic vasospasm requiring treatment with angioplasty as well as persistent hydrocephalus requiring shunting. The literature suggests that vascular ruptures and traumatic aneurysms associated with posterior fossa SAH from blunt trauma result from a common pathophysiological process that reflects both the severity of the injury and preexisting vascular anatomic variants.

CASE REPORTS**Patient 1**

A 22-year-old man was assaulted and experienced a subsequent loss of consciousness. The Glasgow Coma Scale score at presentation was 3T. His initial computed tomographic (CT) scan demonstrated diffuse SAH with intraventricular hemorrhage in the third and fourth ventricles (Fig. 1 A). There was no evidence of cranial or cervical spine fractures. A ventriculostomy was placed for hydrocephalus. Cerebral angiography demonstrated a congenitally

absent left vertebral artery, with both PICAs arising from the right vertebral artery. A 3-mm irregular aneurysm was identified at the origin of the right PICA (Fig. 2 A). A right suboccipital craniectomy was performed. Because of the friable nature of this apparent false aneurysm, clip ligation required sacrificing the distal right PICA. The specimen sent for pathological examination was consistent with a false aneurysm. Transcranial Doppler revealed that the patient had developed severe vasospasm, with hypoperfusion confirmed by single-photon emission computed tomography. Balloon angioplasty of the bilateral supraclinoid carotid arteries, the bilateral M1 segments, and the basilar artery was performed, with resolution of the vasospasm. The patient required a ventriculoperitoneal shunt for persistent hydrocephalus but otherwise made a good recovery and was subsequently discharged to a rehabilitation facility. At 1 year after his injury, the patient was involved in vocational rehabilitation with some memory difficulties but otherwise living independently.

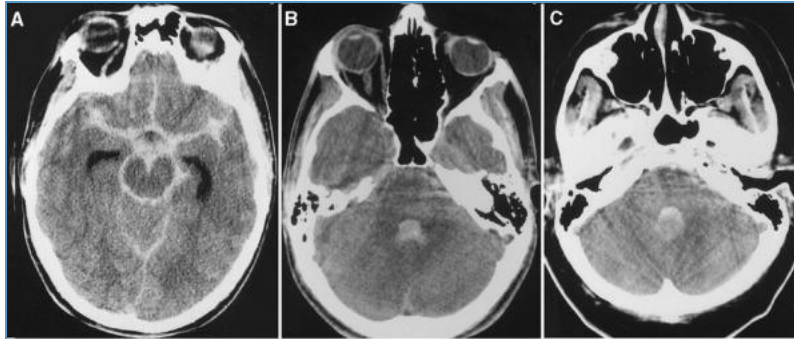


FIGURE 1. Initial head CT scans. A, Patient 1; B, Patient 2; C, Patient 3.

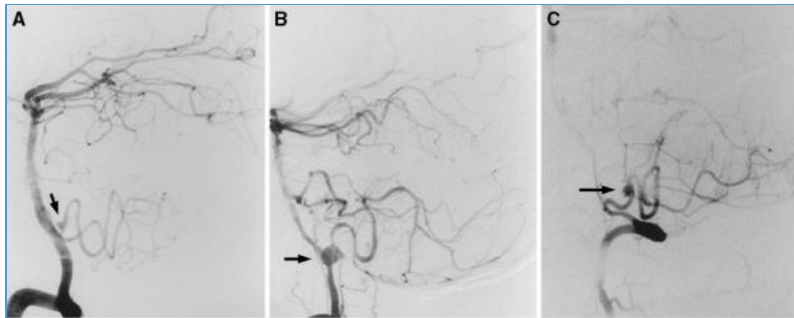


FIGURE 2. Arteriograms showing PICA aneurysms. A, Patient 1; B, Patient 2; C, Patient 3.

Patient 2

A 16-year-old boy was struck in the back of the head with a baseball and had an initial 5-minute loss of consciousness, followed by a short lucid interval; he then became comatose. On admission, his Glasgow Coma Scale score was 3T. His CT scan demonstrated SAH with intraventricular hemorrhage (Fig. 1B), and he required an emergent ventriculostomy for hydrocephalus. The initial arteriogram showed no evidence of vascular abnormality. Because of the suspicious nature of the SAH, the angiogram was repeated at 72 hours postinjury. This examination revealed a 5-mm aneurysm arising from the right PICA origin (Fig. 2B). The patient underwent craniotomy for clip ligation of the aneurysm. The lesion had the appearance of a false aneurysm at the time of surgery, and this impression was verified by pathological analysis. The postoperative angiogram demonstrated no residual aneurysm. The patient developed severe symptomatic vasospasm of the right middle cerebral artery and distal basilar artery, and he subsequently underwent successful angioplasty of these vessels. The patient required ventriculoperitoneal shunting for persistent hydrocephalus and was discharged to a subacute rehabilitation facility. At 6 months after his injury, he still required placement in an assisted living facility.

Patient 3

A 33-year-old woman was thrown from a horse and trampled. On presentation, her Glasgow Coma Scale score was 7T. A CT scan demonstrated SAH, with intraventricular hemorrhage and hydrocephalus (Fig. 1C). There was no evidence of cranial or cervical spine fractures. In addition to the intracranial injuries, the patient also had a fractured humerus. She underwent an emergency ventriculostomy, followed by an angiogram that showed an anomalous left vertebral origin from the arch, a 5-mm left PICA aneurysm arising from the lateral medullary segment, and a fetal origin of the right posterior cerebral artery (Fig. 2C). The patient subsequently underwent successful clipping of her left PICA aneurysm. It appeared to be a true berry aneurysm at the time of surgery; however, no specimen was available for pathological confirmation. She subsequently developed severe right vertebral artery and left posterior cerebral artery spasm by transcranial Doppler criteria; this was confirmed by single-photon emission computed tomography. After the development of clinical symptoms, she underwent successful balloon angioplasty of these vessels. Her hospital course was complicated by a perforated duodenal ulcer requiring

surgical repair, lower-extremity deep vein thrombosis, a pulmonary embolism requiring an inferior vena cava filter, and hydrocephalus requiring a ventriculoperitoneal shunt. She was discharged to a subacute rehabilitation facility and continues to require assisted living 2 years after discharge.

DISCUSSION

Traumatic SAH in the posterior fossa has been associated with several clinical entities, including rupture of a vertebral artery or its branches, the formation of aneurysms (true and false) associated with blunt or penetrating trauma, and dissections of a vertebral artery or associated branches. The mechanism for aneurysm formation after penetrating injury or depressed cranial fracture is well understood. The etiology of dissections that occur spontaneously or with minimal trauma often is related to an underlying vascular pathological condition, such as fibromuscular dysplasia or atherosclerosis. However, the three cases in our series and the supporting literature suggest that the vascular lesions associated with blunt trauma (vascular disruptions and aneurysms) may share a common pathophysiology based on the severity of the injury and predisposing vascular anatomy.

There have been several autopsy studies of fatal SAH resulting from disruption of the intradural vertebral artery or PICA after blunt craniocervical trauma (2, 4, 6). Other than ecchymosis and occasional muscle hematomas, the injuries seemed to be relatively minor and were not associated with overlying cranial fractures. With regard to the vertebral artery ruptures, the lesions were in the transition zone, as the vertebral artery entered the intracranial compartment, and were thought to be due to tethering of the artery by the dura and/or changes in muscular/elastic makeup of the artery (3). In the patients with ruptured PICAs, the vascular disruptions occurred within approximately 1 cm of the origin of the PICA. In each case, the aneurysms were in close proximity to a penetrating brainstem branch. Bostrom et al. (2) speculated that these penetrating vessels from the proximal PICA served as an anchor, focusing shearing forces on the associated artery. Dolman (5) reported two cases of delayed death from traumatic PICA rupture associated with SAH (one at 3 d and one at 3 wk). Autopsy studies demonstrated necrosis and fragmentation of the vessel wall within 1 cm of the origin. The three traumatic aneurysms reported in our series had similar locations (two at the origin and one at the anterolateral medullary segment) along the PICA as the above-noted frank ruptures. Two of the aneurysms had pathology consistent with false aneurysms. By convention, the false traumatic aneurysm represents disruption of the entire vascular wall, including the intima, internal elastic lamina, media, and adventitia, with containment of hematoma by surrounding tissues (12). Owing to the similar location of our traumatic PICA aneurysms and the previously described fatal vascular disruptions, they most likely represent varying degrees of severity of the same pathological process.

True traumatic aneurysms show disruption of the intima, internal elastic lamina, and media with preservation of the adventitia. Although the aneurysm in Patient 3 had a definable neck at the time of surgery, no pathological specimen was submitted for confirmation; it therefore remains unclear whether this represented a true or false aneurysm. Sahjpal et al. (12) recently reported a fatal SAH from a vertebral artery aneurysm rupture associated with a hockey puck striking the patient in the neck. The pathological studies demonstrated that it was a true aneurysm, but the question remains as to whether this was a rupture of an acute aneurysmal dilation or of a preexisting berry aneurysm. As in our cases, Sahjpal et al. had no reason to believe that the SAH preceded or led to the injury.

In our series, the initial CT appearance of SAH and intraventricular hemorrhage was consistent with PICA aneurysm rupture. The high incidence of persistent hydrocephalus and symptomatic vasospasm in these patients was unusual in comparison to our experience with spontaneous PICA aneurysm ruptures, as well as to a recently published series of spontaneous PICA aneurysm ruptures in which symptomatic vasospasm and hydrocephalus occurred in only 5 and 12% of the patients, respectively (8). The occurrence of associated traumatic injuries, as in Patient 3, also certainly affects outcome.

In summary, SAH associated with blunt occipital-cervical trauma should arouse a high degree of suspicion for vascular pathologies, including aneurysms, ruptures, and dissections of the vertebral basilar system. The three patients reported here, as well as the cited literature, support the hypothesis that anatomic variations may predispose patients to injury to the proximal PICA, with the resulting clinical outcomes dependent not only on the severity of the trauma but perhaps also on preexisting vascular abnormalities. The resultant traumatic false aneurysms present a surgical challenge and may require sacrifice of the parent vessel. Our experience indicates that these patients warrant aggressive diagnostic work-up and intervention, as for nontraumatic SAH, with particular attention to the apparent high risk of hydrocephalus and symptomatic vasospasm.

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COMMENTS

Traumatically induced false aneurysms of the cerebral vasculature are usually identified by the following hallmarks: delayed filling and emptying, irregular contour, absence of a neck, and peripheral location not at a branching point. Patients 1 and 2 in this study fulfill most of these criteria. However, I am somewhat uncomfortable with the concept of a traumatic aneurysm undergoing clip ligation in the traditional way. Whether or not Patient 3 had a traumatic aneurysm or an aneurysm associated with trauma may never be known. The central theme of this article is the evaluation of the cause of a subarachnoid hemorrhage in the aftermath of trauma. I am afraid that, without a readily available noninvasive mechanism to accurately image the cerebral circulation, these lesions will continue to be undetected.

Steven L. Giannotta

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This is an interesting report of what must be a very unusual situation. As the authors point out, we are beginning to recognize with increasing frequency dissections of the intracranial portion of the vertebral artery, which sometimes involve the posteroinferior cerebellar artery (PICA) and can occur either spontaneously or after relatively minor head injuries or manipulations such as chiropractic interventions. Incidentally, I am in slight disagreement with the authors about their statement that the etiology of intracranial vertebral dissections is often related to an underlying vascular pathology, such as fibromuscular dysplasia or atherosclerosis. In my limited experience with perhaps 10 of these cases, I have not been impressed with either atherosclerosis or fibromuscular dysplasia as having had any association with the etiology of dissections in this particular location.

These aneurysms that the authors describe, however, are clearly different from dissections. In fact, they demonstrated pathologically that these were traumatic aneurysms in two cases in which they excised the aneurysm. The third patient may not have had a traumatic aneurysm at all, as the authors point out. They state that this third aneurysm had the appearance of a "true berry aneurysm," and the fact that they were able to clip it without sacrificing the parent vessel would support the idea that this was, indeed, not a traumatic aneurysm. It may well be that, in this particular patient, an ordinary berry aneurysm ruptured and the fall from a horse was actually the result of that rupture, rather than vice versa.

In the first two patients, it appears that the authors sacrificed the PICA at its origin; they do not indicate that these patients sustained a lateral medullary infarct. I find this interesting because I have always advocated some form of distal revascularization whenever the origin of the PICA has to be sacrificed, provided that it is practical and feasible to perform such revascularization, in view of the patient's clinical condition at the time and technical factors. These two patients were very ill, and perhaps they did have a medullary infarct that was masked by other problems related to vasospasm or hydrocephalus. If, however, they did not experience a lateral medullary infarction, I may need to revise my thinking in terms of the danger of taking the PICA at its origin. Nevertheless, until convinced otherwise, I would always recommend at least attempting some form of distal revascularization, which in my experience is easier to accomplish, when the anatomy is appropriate, by a side-to-side anastomosis of a more distal branch of the ipsilateral PICA with a distal branch of the contralateral PICA or, alternatively, by reimplanting the origin of the ipsilateral PICA into a branch of the contralateral PICA.

Roberto C. Heros

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This article reviews an unusual clinical and anatomic situation: the traumatic development and rupture of intracranial aneurysms unrelated to penetrating injury. The authors convincingly present three similar cases in which sudden acceleration-deceleration head trauma resulted in massive subarachnoid bleeding associated with disruption of the PICA wall at (Patient 2) or very near (Patient 1) the arterial origin from the parent vertebral vessel. It would seem most likely that the vertebral artery's rigid tether by the dural tunnel, compounded by sudden sagittal movement of the brainstem, cerebellum, and attached PICA, must focus shearing forces on the PICA origin sufficient to effect false aneurysm formation. The risk of obligatory PICA occlusion associated with the treatment of such lesions should be recognized preoperatively, and provisions should be made for implantation bypass if necessary.

Duke Samson

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Key words: Aneurysm; PICA; Subarachnoid hemorrhage; Trauma

IMAGE GALLERY

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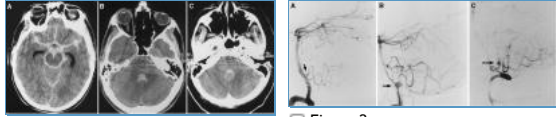


Figure 1

Figure 2

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