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A Rare Case of Dissecting Aneurysm of Extradural PICA Presenting With Subdural Hemorrhage

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Abstract

Subdural hematoma (SDH) occurs from interruption of bridging or superficial cerebral veins, most commonly from head trauma. Rupture of an intracranial aneurysm may rarely lead to SDH with or without subarachnoid or intraventricular bleed. We report a rare case of ruptured dissecting aneurysm of an extradural posterior inferior cerebellar artery (PICA) leading to spontaneous subdural hematoma and explain possible mechanisms.

Keywords: Extradural PICA • Dissecting aneurysm • SDH

Introduction

The posterior inferior cerebellar artery (PICA) is the most prominent, complex branch of the V4 segment of the vertebral artery and is notorious for its anatomic variations. The extradural origin of PICA comprises approximately 5-20% of its variations [1]. The extradural location predisposes the artery to traumatic dissection as the artery is not supported by bony calvaria. Also, there is a high chance of iatrogenic injury during open suboccipital surgeries. Dissecting aneurysms of PICA embrace about 0.5%- 0.7% of intracranial aneurysms [2]. Dissecting aneurysm of an extradural PICA is furthermore rare [3,4]. Many of these aneurysms are missed during routine imaging due to the field of view failing to include sections below the foramen magnum. When these aneurysms rupture, the blood may seep into the subdural space *via* a dural defect. Ruptured aneurysms presenting as acute subdural hematoma (SDH) are rare with an incidence ranging from 0.5 to 7.9% [5]. Rupture of posterior communicating artery aneurysm is the most common cause of SDH, however, ICA, MCA, ACA, and anterior communicating artery aneurysm rupture have also been implicated [5]. To our knowledge, no case of ruptured extradural PICA presenting with a subdural hematoma has been reported yet. We present an exceedingly rare case of ruptured dissecting aneurysm of an extradural PICA presenting with SDH managed endovascularly with a good clinical outcome on follow-up.

Case Presentation

A female patient in their late fifties presented with a sudden onset of headache and vomiting followed by loss of consciousness. The patient had a similar untreated episode one month ago with an index CT showing the presence of subdural hematoma along the right frontoparietotemporal convexity and intraventricular hemorrhage (Figure 1a). In current admission, the patient had a GCS of 15. Repeat NCCT showed an increase in SAH in posterior fossa cisterns (Figure 1b). CT angiography showed delayed opacification of an outpouching in relation to the V3-V4 junction of the right vertebral artery, measuring 11 × 5 mm and extending below the foramen magnum, just above the posterior arch of the atlas (Figures 1c,d). A diagnostic DSA confirmed a dissecting aneurysm from the right extradural PICA originating below the level of the foramen magnum (Figure 2). Severe vasospasm was also seen in the posterior circulation. Endovascular management of this extradural ruptured PICA aneurysm was performed by parent vessel occlusion using two coils with post-procedural complete occlusion and non-filling of the aneurysmal sac (Figure 2d). Modified Rankin scale at 3 months follow-up was 0.

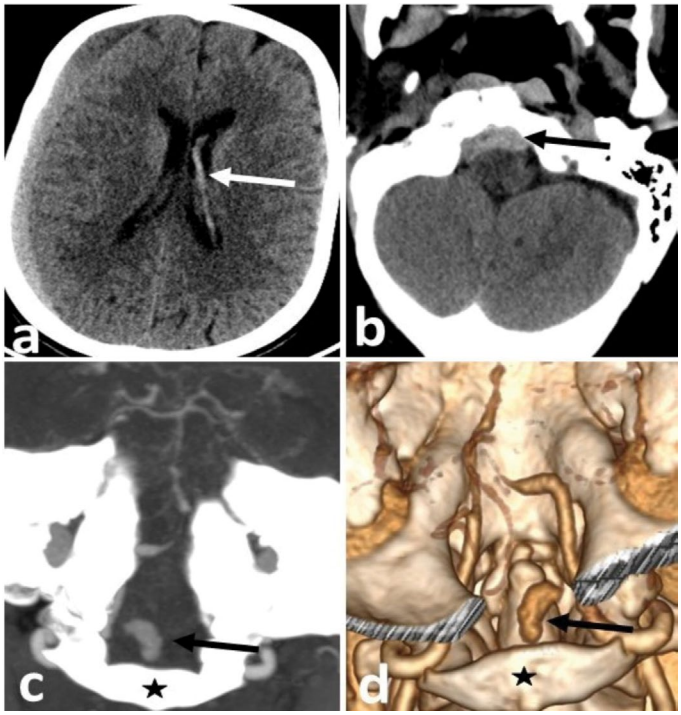

Figure 1. (a) Index NCCT head 1 month before ictus showed subacute SDH along right frontoparietal convexity and IVH in the left lateral ventricle (arrow), **(b)** Present NCCT head shows SAH in perimedullary cistern (arrow), **(c)** Coronal MIP CT angiography image shows contrast-filled outpouching (arrow) in close relation with the right vertebral artery above the posterior arch of the atlas (asterisk). Note vasospasm in vertebrobasilar axis and **(d)** Volume rendered CT angiography image showing contrast-filled outpouching in close relation with the right vertebral artery (arrow) above the posterior arch of the atlas (asterisk).

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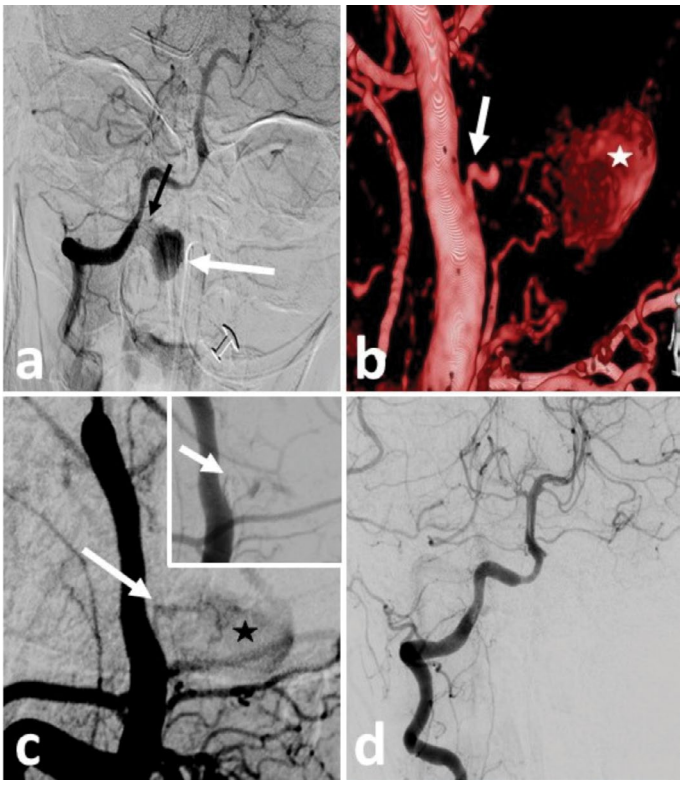

Figure 2. (a) Right vertebral angiogram showing dissecting aneurysm (white arrow) arising from extradural PICA originating from right vertebral artery below the level of the foramen magnum (black arrow), **(b)** 3D rotational angiography image demonstrating the origin of PICA (white arrow) with opacification of the aneurysm sac (asterisk), **(c)** Origin of extradural PICA is seen (arrow) with opacification of aneurysm sac (asterisk). Parent artery occlusion was done by deploying 2 microcoils of size 1mm × 1cm (white arrow in inset) in the proximal PICA with resultant near-total obliteration of the aneurysm and **(d)** Post coiling right vertebral angiogram shows complete obliteration of the aneurysm.

Discussion

PICA – Anatomical importance

The posterior inferior cerebellar artery (PICA) is the most prominent branch of the V4 segment of the vertebral artery. It supplies the inferior aspect of the cerebellum, vermis, choroid plexus of the fourth ventricle, tonsils, and medulla. PICA usually originates from the V4 segment of the vertebral artery, approximately 8.6 mm above the foramen magnum and 1.6-1.7 cm proximal to the vertebrobasilar junction [6]. Like any other artery in the cerebral circulation, PICA also has a variety of variations in origin and course. Extradural origin of PICA arising from the V3 segment is seen in approx. 5- 20% of cases [1]. It usually arises within 1 cm of the point where VA penetrates dura [7]. There is important clinical relevance to this variation in neurosurgery. As the vessel is extra-durally located, it is not secured by bony calvaria and is enclosed under the neck muscles. This connotes a high incidence of vascular injury in trivial neck trauma. Injury to extradural PICA may also occur after chiropractic maneuvers [8]. During neurosurgical soft tissue dissection in suboccipital posterior fossa surgeries, awareness of this variation is of extreme importance to avoid an iatrogenic injury.

The extradural course of PICA is generally tortuous and is subject to unusual hemodynamic stress which may predispose it to dissection and aneurysm formation. The vessel may come in close proximity to the arch of C1, which can inadvertently damage the walls of the artery from neck manipulation [9]. Clinically, a dissected PICA may present with ischemic symptoms, if the proximal segment is involved or, with hemorrhagic manifestations, if the distal segment is involved [10]. Prognosis in both scenarios is different with a better prognosis in the former, as there is typically a favorable cerebellar arterial anastomotic network. Hemorrhagic manifestations, on the other hand, have a poor prognosis and transpire in form of subarachnoid or intraventricular bleed.

SDH in ruptured extradural PICA aneurysm

Ruptured aneurysms can present with SAH (~60%), ICH (~30-40%), IVH (~12-17%) & SDH (~0.5-7.9%) [5,11]. A subdural hemorrhage usually ensues from a traumatic head injury, although spontaneous SDH has been associated with rupture of an arteriovenous malformation, cerebral atrophy, use of antithrombotic agents, coagulopathy, and cocaine abuse. Tear in bridging or cortical veins lead to egress of blood in the subdural space. SDH is a rare occurrence in the rupture of an intracranial aneurysm. Pure SDH without SAH is furthermore rare. All previous descriptions of aneurysm rupture leading to spontaneous SDH have shown that these aneurysms are intradurally located with the most common locations being ACA, MCA, and ICA (posterior communicating artery) [5]. Encroachment of the perianeurysmal environment (dura, bone, and cranial nerves) by the aneurysm sac may predict rupture of the aneurysm beyond subarachnoid space. Mechanisms include adherence of aneurysm adventitia to the overlying arachnoid membrane in response to previous subclinical microbleeds leading to tearing and extension of blood in the subdural space, high pressures generated during rupture penetrating through unprecedented weak points in the arachnoid membrane, or, erosion of cavernous sinus from aneurysmal growth in aneurysms in such locations [12]. Occasionally, a peripherally located aneurysm (e.g. mycotic aneurysms) can directly lead to SDH on rupture. Whether rupture of an extradural aneurysm can lead to SDH is an unexplored entity. To our knowledge, our index case is the first report in literature harboring an extradural aneurysm that ruptured and led to supratentorial SDH. This may presumably occur from small anatomical dural defects (as in the case of vertebral artery and PICA, where the loop of the vessel pierces dura in the region of the foramen magnum) with eventual separation of dural folds from repetitive microbleeds or shear force from the jet of the aneurysm. The direction of the jet may predict the location of subdural blood, which should typically be located in the infratentorial compartment, or more rarely, in the supratentorial region, as in our case. The presence of SAH and IVH can be explained by concomitant tearing of arachnoid and extension of blood into subarachnoid spaces.

Therapeutic strategy

The presence of SDH is managed with surgical evacuation to control rising intracranial pressures. Dissecting aneurysms of PICA can be treated by microsurgery or endovascularly by parent artery occlusion (PAO). Microsurgical methods include clipping or trapping of the aneurysm or resection and end-to-end anastomosis. However, there have been case reports where post-op dislodgement of the clip has been seen with recurrence and rupture of the aneurysm [13]. Dislodgement of the clip can occur because of neck movements or trivial trauma to the neck, as the aneurysm is not secured by bony calvaria. Even in intradural PICA aneurysms, clipping and, resection and anastomosis are shown to be associated with complications due to the proximity of brainstem and lower cranial nerves [14].

Endovascular management is well known & relatively safe and less morbid treatment option in these patients. Endovascular parent artery occlusion of intradural PICA in the proximal segment may cause brainstem ischemia because brainstem perforators arise from the proximal two segments. In distal aneurysms, PAO can be easily performed without much concern. Incidence of brainstem ischemia in occlusion of extradural PICA is increasingly scarce as perforators arise from the expected location of PICA from VA [15]. PAO may lead to the development of cerebellar infarcts, however, these are not significant due to extensive anastomosis between cerebellar arteries. It is important to identify the origin of the spinal artery which occurs close to an extradural PICA before resorting to definitive management. Endovascular techniques are well-established and safe methods for intracranial aneurysm occlusion. We performed PAO of the extradural PICA in our case, however, the tortuosity of PICA prevented the microcatheter to reach distal to the aneurysm, and trapping was not possible. This is one of the technical limitations of endovascular procedures. However, after achieving the farthest distal control, complete occlusion of the aneurysm and parent artery was performed and post-procedure angiography showed no other anastomotic channel feeding the dissecting aneurysm. The patient was discharged with no neurological deficits.

Conclusion

Dissecting aneurysm of an extradural PICA is a rare entity and more so, rare presentation with subdural hematoma may occur. During imaging evaluation of a spontaneous SDH and posterior fossa SAH and IVH, the field-of-view coverage should include the area below the foramen magnum to detect ruptured aneurysms below the skull base. Dissecting aneurysms have a high incidence of rebleeding, as seen in our patient. Timely diagnosis and management can change patient outcomes.

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