Ruptured traumatic posterior inferior cerebellar artery pseudoaneurysm: A case report and literature review

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INTRODUCTION

Traumatic intracranial aneurysms (TICAs) comprise <1% of all cerebral aneurysms[19] and can occur because of skull fractures or penetrating injuries.[14] TICA of the posterior circulation is rare (<10%).[11] TICA limited to the posterior inferior cerebellar artery (PICA-TICA) is even rarer and has only been reported in approximately 20 cases to date. TICA often grow and rupture late, implying that they are easily missed during initial imaging.[9] Previous cases of PICA-TICA rupture have often resulted in intraventricular hemorrhage (IVH) and subarachnoid hemorrhage (SAH) in the posterior fossa. Some studies report that PICA-TICA is often observed with...
vascular anomalies in the posterior fossa.\textsuperscript{[12,13]} Endovascular treatment was performed in five of the previously reported 20 cases of PICA-TICA.\textsuperscript{[5,14]}

This case report outlines the steps taken to treat the PICA-TICA, patient outcomes, and lessons learned for effective treatment of future PICA-TICA patients. We also reviewed previous reports on the characteristics and treatment of PICA-TICA.

CASE PRESENTATION

History and examination

A 69-year-old woman fell 3 m into a shallow river, bruising her head and chest. She was admitted to our hospital as an emergency case with no relevant medical history. In the emergency department, her Glasgow Coma Scale score was determined to be 11. She had a headache, nausea, and amnesia at the time of injury, but no apparent paralysis. She had a bruise on the right side of her head and swelling on the precordium.

Computed tomography (CT) showed subarachnoid hemorrhage in the posterior fossa and left Sylvian fissure, intraventricular hemorrhage with slight ventriculomegaly, and contusion of the left temporal lobe [Figures 1a and b]. Fractures of the right temporal bone and zygoma were also observed [Figure 1c]. A cerebral CT angiogram revealed no aneurysm and vascular abnormalities without right dominant PICA [Figures 1d and e]. Chest and abdominal CT showed pneumothorax and multiple rib fractures on the right side. A tube thoracostomy was performed to treat the traumatic hemothorax and pneumothorax. Laboratory examination revealed coagulation disorders, including a d-dimer elevation of 175 μg/mL and international normalized ratio of 1.35. The platelet count was 14.1 μg/mL. We suspected disseminated intravascular coagulation due to severe trauma. Six units of fresh frozen plasma and four units of red blood cells were administered to correct the coagulopathy, and 1 g of tranexamic acid was administered twice. We decided to perform conservative treatment and careful follow-up for intracranial hemorrhage. However, the patient suddenly fell into a semi-comatose condition 1 h later. Cerebral CT showed increased subarachnoid hemorrhage, IVH, and ventricular enlargement [Figure 2a].

Treatment and posttreatment course

Digital subtraction angiography (DSA) was performed to identify the source of the hemorrhage. Under local anesthesia, a 5-French introducer sheath was placed in the right femoral artery (FA). A six-vessel selective cerebral angiogram was performed using a 5-French JB2 125-cm catheter (Medikit, Tokyo, Japan). A right vertebral artery (VA) angiogram revealed an 11 mm aneurysm in the right PICA anterior medullary segment (AMS). It was considered...
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Figure 2: (a) One hour later cerebral computed tomography (CT) images showed increased subarachnoid hemorrhage, intraventricular hemorrhage, and ventricular enlargement. (b) A right vertebral artery (VA) angiography three-dimensional image revealed an 11 mm aneurysm in the right posterior inferior cerebellar artery (PICA) anterior medullary segment (AMS). (c) The enlarged view of B. Green line is 11 mm and the orange line is 9 mm. It was considered a pseudoaneurysm because of its small and unclear aneurysmal neck. (d) The Marathon microcatheter (Medtronic, Minneapolis, Minnesota, USA) was approached to the aneurysm from the right VA using the Tenrou 1014 (Kaneka Medics, Kanagawa, Japan). However, with this approach, it was difficult to guide the microcatheter because the direction of the microcatheter and the aneurysm neck was almost at 180° with each other. (e) A Marathon catheter was approached to the aneurysm from the left VA beyond the union using the Tenrou 1014. (f) Microcatheter angiography revealed an aneurysm and backflow to the VA and PICA. The diameter of the small space at the entrance of the aneurysm where the Marathon microcatheter could be placed was approximately 1.5 mm. G: n-butyl-2-cyanoacrylate (NBCA) (12.5%) was injected into the aneurysm while blocking the proximal part of the right VA with a SHOURYU HR balloon (7 mm × 7 mm) (Kaneka Medics, Kanagawa, Japan) and suppressing the backflow to the VA. (h) Using NBCA, we were able to embolize a small part of the aneurysm neck. Although the Marathon microcatheter was carefully removed, its removal from the aneurysm caused NBCA to scatter into the PICA. (i) The right VA angiography revealed that the aneurysm had disappeared, but blood flow in the PICA was very slow.

... a pseudoaneurysm because of its small and unclear aneurysmal neck [Figures 2b and c]. No other vascular lesions causing SAH were observed. Therefore, the patient was diagnosed with SAH due to a ruptured traumatic PICA pseudoaneurysm.

Under local anesthesia, 3000 units of heparin were injected intravenously, and activated coagulation time (ACT) was extended to 250 s. A 5-French JB2 125 cm catheter was placed in the left VA for angiography to confirm blood flow. A 6-French introducer sheath was placed in the left FA, and a 6-French FUBUKI guiding catheter of 90 cm (Asahi Intecc, Aichi, Japan) was placed in the right VA. A Marathon microcatheter (Medtronic, Minneapolis, Minnesota, USA) was inserted into a 6-French FUBUKI with a Tenrou 1014 (Kaneka Medics, Kanagawa, Japan) to approach the aneurysm. However, with this approach, it was difficult to guide the microcatheter because the direction of the microcatheter and the aneurysm neck was almost at 180° with each other [Figure 2d]. A Marathon catheter and a Tenrou 1014 were inserted into the 5-Fr JB2 125 cm catheter in the left VA and guided beyond the union to the right PICA [Figure 2e]. We managed to guide the Marathon microcatheter and a Tenrou 1014 to the entrance of the aneurysm but were unable to guide them into the aneurysm. Microcatheter angiography revealed an aneurysm and backflow to the VA and PICA [Figure 2f]. The diameter of the small space at the entrance of the aneurysm where the Marathon microcatheter could be placed was approximately 1.5 mm. It was predicted that the Marathon microcatheter would be extremely unstable. The coil could not be placed inside the aneurysm; hence, it was embolized using n-butyl-2-cyanoacrylate (NBCA). NBCA (12.5%) was injected into the aneurysm while blocking the proximal part of the right VA with a SHOURYU HR balloon...
(7 mm × 7 mm) (Kaneka Medics, Kanagawa, Japan) and suppressing the backflow to the VA [Figure 2g]. Using NBCA, we were able to embolize a small part of the aneurysm neck. The Marathon microcatheter was carefully removed, but its removal from the aneurysm caused NBCA to scatter into the PICA [Figure 2h]. An angiogram revealed that the aneurysm had disappeared, but blood flow in the PICA was extremely slow [Figure 2i]. The puncture site was manually pressed to stop the bleeding.

Immediately after the surgery, the level of consciousness was somnolent due to sedation, but no anisocoria was observed. Cerebral CT revealed dilated ventricles and contusive hemorrhage in the left temporal lobe with uncal herniation [Figure 3a]. After CT imaging, the respiratory condition deteriorated and intubation was performed. The mydriasis of the left pupil was observed. We then decided to perform decompression craniectomy on the left side of the head. However, it was difficult to stop the bleeding during the surgery, likely due to considerable swelling of the brain and abnormal coagulation. Although decompression craniectomy and hematoma removal were performed, postoperative CT showed an enlarged contusion hemorrhage and uncal herniation [Figures 3b and c]. Brain death was confirmed on the 2nd day after the surgery, and the patient died on the 4th day.

DISCUSSION

In this case report, we described the diagnosis and treatment of PICA-TICA in a patient with TBI after a fall. Although we embolized the TICA, the patient did not survive. These results demonstrate the importance of early detection and appropriate treatment when treating TICAs due to their unpredictable nature.

The incidence of TICA is rare and accounts for less than 1% of all intracranial aneurysms. TICA has a high rupture and growth rate, resulting in a high mortality rate. Half of all TICAs have delayed ruptures; reports range from hours to 10 years later, with an average of 14–21 days. The mortality rate for untreated TICAs is 32%–54%, and treatment reduces it to 18%–24%.

TICA is more common in the anterior circulation, and less than 10% of TICAs occur in the posterior circulation. Rupture of a TICA of posterior circulation should be ruled out in the presence of IVH or massive SAH of the posterior fossa. In this case, TICA was detected in the PICA. PICA aneurysms account for 0.5%–3.0% of all intracranial aneurysms, of which 0.28%–1.4% are localized in the PICA. Tokimura et al. reported that aneurysms confined to the PICA are often associated with some posterior vascular anomalies, such as defects in the contralateral and ipsilateral PICA. Approximately 90% of PICA aneurysm ruptures show concomitant IVH with hydrocephalus observed on CT.

There are few reports of traumatic PICA aneurysms such as the one described in this case study. A review of the English and Japanese language literature performed on December 3, 2021, revealed 12 reports describing 15 cases of PICA-TICA. The clinical characteristics and outcomes of these patients are summarized in Table 1. Among the 15 cases (including our patient), the mean patient age was 32.4 (15–69) years. Regarding the injury mechanism, seven cases were from fighting, four cases were falls, and two cases were traffic accidents. Eight patients with more severe injuries were in a coma at the time they were admitted. There were 15 cases of SAH and 11 cases of IVH with ventricular enlargement. Five patients had vascular anomalies of the posterior fossa. If a patient with TBI has thick posterior fossa SAH or IVH with hydrocephalus, there is a possibility of TICA rupture in the posterior fossa.

The timing of TICA diagnosis was after the 7th day in nine cases, and aneurysms in six cases were identified within 1 day. Regarding the location of PICA-TICA, 12 of 15 cases were AMS to lateral medullary segment (LMS) near the brain stem. Direct surgery, such as a trapping bypass, was performed in eight cases, and endovascular treatment was performed in four cases. Overall, 5 patients (33%) had good outcomes (Glasgow Outcome Scales 4 or 5) and seven patients died. The untreated case (two patients) had a poor prognosis. There was no difference in prognosis depending on the time of rupture or the site of the aneurysm. The reason may be that there are only a few reports and publication bias.

In our review of PICA-TICA, there were five cases with vascular anomalies in the posterior fossa. There are also reports of the PICA starting immediately after the dural penetration of VA, as in the present case. PICA fixed to the dura mater may be subject to rotational acceleration, and a pseudoaneurysm may form at its origin.
Table 1: Literature review of patients with nonpenetrating injury related pseudoaneurysm of the posterior inferior cerebellar artery.

| Author          | Age  | Trauma mechanism | Neurological symptoms                       | Initial CT finding                   | Aneurysmal segment of PICA | Timing of TICA diagnosis | Vascular anomaly of posterior cranial fossa | Management          | Outcome |
|-----------------|------|------------------|--------------------------------------------|--------------------------------------|---------------------------|-------------------------|---------------------------------------------|---------------------|---------|
| Shuster (1999)  | 22   | Assault          | GCS 3T                                     | SAH, IVH, ventriculomegaly           | AMS                       | 0 days                  | Congenitally absent left VA, both PICA arising from right VA | PA ligation       | GOS 4   |
|                 | 16   | Baseball         | GCS 3T                                     | SAH, IVH, ventriculomegaly           | AMS                       | 3 days                  | N/A                          | PA ligation       | GOS 3   |
|                 | 33   | Horse accident   | GCS 7T                                     | SAH, IVH, ventriculomegaly           | LMS                       | 0 days                  | A fetal origin of the right PCA, an anomalous left vertebral origin from the arch | Clip               | GOS 3   |
| Kibayashi (2000)| 20   | Fight            | Comatose                                   | Massive basal SAH                    | AMS                       | 6 days                  | N/A                          | No                  | GOS 1   |
| Nishioka (2002)| 20   | Karate kick      | Comatose                                   | SAH, IVH, ventriculomegaly           | AMS                       | 11 days                 | N/A                          | No                  | GOS 1   |
|                 | 33   | Fight            | Headache and nausea                        | SAH localized at the preoptic cistern, IVH, ventriculomegaly | AMS                       | 21 hours                | N/A                          | Clip               | GOS 5   |
| Aronson (2008)  | 65   | Unknown          | Nausea                                     | SAH in the posterior fossa           | PMA                       | N/A                     | Extradural origin of the left PICA from the ipsilateral VA Anomalous origin of the PMA from the PICA | Resection          | GOS 5   |
| Binning (2009)  | 15   | Assault          | Comatose                                   | SAH of the posterior fossa, ventriculomegaly | AMS                       | 21 months               | N/A                          | Trapping and bypass | GOS 5   |
| Purgina (2015)  | 22   | Fight            | GCS 7T                                     | SAH, IVH, ventriculomegaly           | AMS                       | 7 days                  | N/A                          | No                  | GOS 1   |
| Lee (2016)      | 55   | Slip down        | GCS 3T                                     | SAH, IVH, ventriculomegaly           | CS                        | 30 days                 | N/A                          | Resection          | GOS 5   |
| Desouza (2016)  | 21   | Assault          | GCS 14                                     | SAH, IVH, ventriculomegaly           | AMS                       | 2 hours                 | N/A                          | Coil and Stent      | GOS 2   |
|                 | 35   | Assault          | Loss of consciousness                      | SAH of the posterior fossa, IVH, ventriculomegaly | N/A                       | 7 days                  | N/A                          | Coil               | GOS 1   |
| Sakamoto (2019) | 35   | Assault          | GCS 13                                     | SAH, IVH, ventriculomegaly           | LMS                       | 0 days                  | Left PICA dominant, proximal origin of VA | Trapping and bypass | GOS 1   |

(Contd)
Our patient also had SAH which was mostly confined to the posterior fossa, IVH, and mild ventriculomegaly on the initial CT. It was suspected that an intrinsic cerebral hemorrhage had occurred or that a traumatic cerebral aneurysm had ruptured. A cerebral CT angiogram was performed, but no vascular abnormality that could be a hemorrhage source of SAH or IVH was detected. Therefore, conservative treatment and imaging follow-up were performed for intracranial hemorrhage, and systemic treatment was administered for complicated pneumothorax, multiple fractures, and hypothermia.

The DSA performed 2 h after the cerebral CT angiogram showed that PICA-TICA had expanded to 11 mm. Because of the dominant PICA, it was likely under hemodynamic stress, and PICA might partially fix to the dura mater; therefore, it is possible that the external force of the trauma was applied to the origin of the PICA, resulting in a pseudoaneurysm. In general, direct surgery such as clipping and trapping bypass and endovascular treatment such as parent artery occlusion, intra-aneurysm coil embolization, and stent-assisted coil, have been reported for the treatment of PICA-TICA. In this case, we decided to perform intra-aneurysmal embolization using NBCA to avoid parent artery occlusion because the right PICA was dominant. There are reports on the use of arterial glue, such as NBCA, for the embolization of ruptured aneurysms. Arterial glue is useful for preserving the parent artery during aneurysm embolization. Cognard et al. reported that NBCA could be used to preserve the parent artery during embolization of PICA aneurysms. In this case, the embolization was successful without a kickback of the microcatheter. However, when the microcatheter was removed, a portion of the NBCA leaked to the main trunk of the PICA, and the distal side of the PICA became occluded. Attention should be paid to the behavior of the NBCA when removing the microcatheter.

**CONCLUSION**

We encountered a case in which a patient with a vascular anomaly in the posterior fossa had IVH and SAH after a fall, and a late PICA-TICA, which was difficult to diagnose and treat. PICA-TICA is often accompanied by IVH and SAH, and there are some reports of cases with a vascular anomaly of the posterior circulation. Since TICA is at risk of rapid growth and rupture, an early and appropriate diagnosis is important.

**Declaration of patient consent**

Patients’ consent not required as patient’s identity is not disclosed or compromised.

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**Conflicts of interest**

There are no conflicts of interest.

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