ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Professor of Clinical Neurosurgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neurovascular

Editor
Kazuhiro Hongo, MD
Shinshu University, Matsumoto, Japan

SNI
Open Access

Case Report

Traumatic intracranial aneurysm in a distal posterior cerebral artery: A case report and literature review

Kengo Yamada[1], Takayuki Morimoto[1], Kenta Fujimoto[2], Toshikazu Nishioka[1], Hidemori Tokunaga[1]

[1]Department of Neurosurgery, Nara City Hospital, [2]Department of Neurosurgery, Nara Prefecture General Medical Center, Nara, Japan.

E-mail: Kengo Yamada - k196441@naramed-u.ac.jp; *Takayuki Morimoto - t.morimoto@naramed-u.ac.jp; Kenta Fujimoto - kentaf@nara-hp.jp; Toshikazu Nishioka - t-nishioka@nara-jadecom.jp; Hidemori Tokunaga - h-tokunaga@nara-jadecom.jp

***Corresponding author:**
Takayuki Morimoto,
Department of Neurosurgery,
Nara City Hospital, Nara, Japan.

t.morimoto@naramed-u.ac.jp

Received: 11 September 2023
Accepted: 18 November 2023
Published: 15 December 2023

DOI
10.25259/SNI_752_2023

Quick Response Code:

ABSTRACT

Background: Traumatic intracranial aneurysms (TICAs) are rare and known to rupture easily and have a high mortality rate.

Case Description: An 87-year-old male patient with no neurological deficits presented to our hospital after head trauma. Computed tomography (CT) revealed a tentorial acute subdural hematoma (ASDH). The patient was managed conservatively and discharged home six days after hospitalization. Two days later, the patient returned with a severe headache. CT showed that the ASDH had enlarged and extended from the tentorium to the convexity. CT angiography and digital subtraction angiography revealed a pseudoaneurysm in a branch of the left posterior inferior temporal artery. The patient was diagnosed with an enlarged ASDH due to a ruptured TICA that arose from the P3 segment. We performed endovascular intervention with parent artery occlusion (PAO) using n-butyl-2-cyanoacrylate (NBCA). The parent artery was accessed through the left posterior communicating artery because left vertebral angiography revealed an aplastic left P1 segment. After navigating the microcatheter near the aneurysm, we injected 33% NBCA into the parent artery. The pseudoaneurysm disappeared after injection. The patient was discharged on hospital day 25 despite persistent delirium.

Conclusion: This is the first report of a TICA arising from the P3 segment that was treated with PAO using NBCA. TICAs are rare; however, a TICA must be considered when an enlarged hematoma caused by head injury is detected.

Keywords: Acute subdural hematoma, N-butyl-2-cyanoacrylate, Parent artery occlusion, Traumatic intracranial aneurysm

INTRODUCTION

Traumatic intracranial aneurysms (TICAs) are very rare, accounting for <1% of all aneurysms. It is known to rupture easily and has a high mortality rate.[6,8] TICAs are even rarer in the posterior cerebral artery (PCA). Only a few case studies have reported TICAs in the distal PCA.[4,5] In this study, we report a rare case of a patient with acute subdural hematoma (ASDH) due to a ruptured TICA arising from the distal PCA. The patient was treated by endovascular parent artery occlusion (PAO) using n-butyl-2-cyanoacrylate (NBCA).

CASE REPORT

An 87-year-old healthy male patient presented to the emergency department with head trauma after a fall. The patient's Glasgow coma scale (GCS) score was 14 (E4V4M6), indicating a mild disturbance of consciousness. The patient had no neurological deficits. A head computed tomography (CT) scan showed a subcutaneous hematoma in the right parietal region and ASDH extending from a subarachnoid cyst in the left middle cranial fossa to the left tentorium and falx [Figures 1a and b]. The CT scan showed no significant subarachnoid hemorrhage or cerebral contusion. The patient was diagnosed with ASDH and cerebral concussion and was treated conservatively. A repeat CT scan on the following day revealed no remarkable change, while a CT scan performed six days after admission showed a reduction in the ASDH. The patient presented no neurological abnormalities and was discharged on day 6.

Two days after discharge, the patient presented to the emergency department with a headache and disorientation. The assessment indicated mild disturbance of consciousness, a GCS score of 14 (E4V4M6), and no focal signs. Head CT imaging revealed ASDH extending from the left tentorium to the left convexity, with effacement of the sulci and a midline shift [Figures 1c and d]. Three-dimensional CT (3D-CT) showed a 7.0-mm mass lesion in the ASDH of the tentorium, which was contiguous with the left posterior inferior temporal artery (PITA) branching from the P3 segment of the PCA [Figures 1e and f]. Three-dimensional digital subtraction angiography and vertebral artery angiography demonstrated a pseudoaneurysm in the PITA from the P3 segment with irregular contours, delayed filling, and no obvious neck [Figures 2a and b]. Based on these findings, the patient was diagnosed with ASDH regrowth due to a ruptured TICA. PAO with endovascular treatment was planned to avoid re-rupture of the pseudoaneurysm.

Endovascular treatment was performed under general anesthesia. We approached through the left posterior communicating artery as the left P1 segment was hypoplastic. A 6 Fr FUBUKI catheter (Asahi Intec, Aichi, Japan) was guided into the origin of the left internal carotid artery using a 4-Fr JB2 catheter and a 0.035-inch guiding wire. An intermediate catheter (Guidepost; Tokai Medical Products, Aichi, Japan) was, then, advanced into the left internal carotid artery. A microcatheter (DeFrictor BULL; Medicos Hirata, Tokyo, Japan) was inserted proximal to the aneurysm in the left PITA using a microwire (TENROU 1014; Kaneka Medix, Osaka, Japan; [Figures 3a and b]). The artery was embolized with 0.13 mL of 33% NBCA. A left vertebral artery angiography revealed the disappearance of the pseudoaneurysm [Figure 3c].

Head magnetic resonance imaging on the following day showed a partial acute infarction at the base of the left temporal lobe. However, the patient experienced no symptoms such as visual apraxia or loss [Figures 4a and b]. Follow-up magnetic resonance angiography and 3D-CT angiography demonstrated complete occlusion of the pseudoaneurysm with no recanalization [Figures 4c and d]. On the 6th postoperative day, a CT scan showed a chronic subdural hematoma, which was evacuated by a burr-hole surgery. Despite persistent delirium, the patient was discharged home on day 25 of hospitalization with a Glasgow outcome scale score of 4.

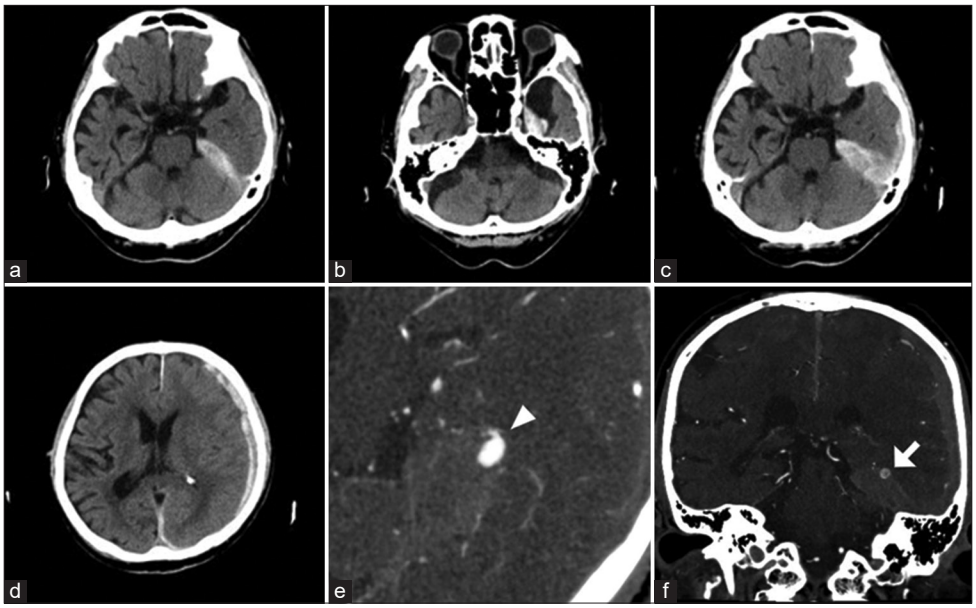

Figure 1: (a and b) Initial computed tomography (CT) scan showing left tentorial acute subdural hemorrhage (ASDH). (c and d) A CT scan during the second admission showed enlarged ASDH. (e and f) CT angiography showing left pseudoaneurysm in the P3 segment of the posterior cerebral artery (arrowhead) (arrow).

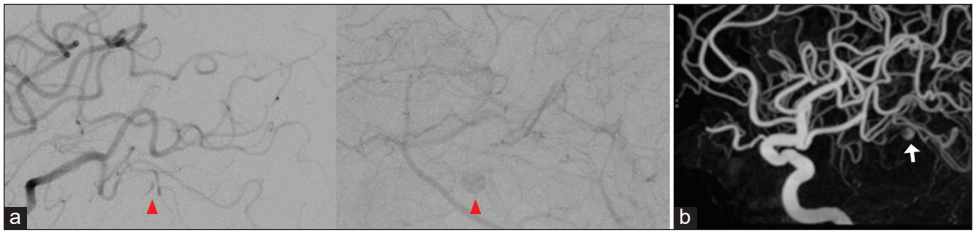

Figure 2: (a) Left internal carotid artery angiography in the early (left panel) and late phase (right panel) showing a pseudoaneurysm (arrowhead). (b) Three-dimensional rotational angiography of the internal carotid artery showing the pseudoaneurysm (arrow).

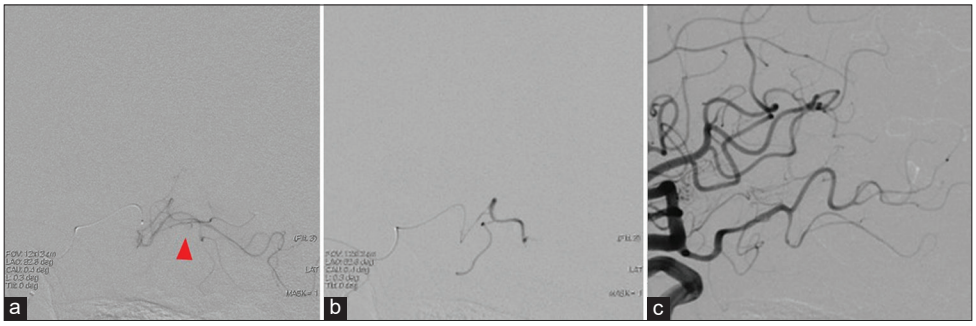

Figure 3: (a) A microcatheter was introduced into the posterior inferior temporal artery. The arrowhead points at the pseudoaneurysm. (b) Injection of 33% *n*-butyl cyanoacrylate (NBCA). (c) Internal carotid artery angiography after injection of NBCA.

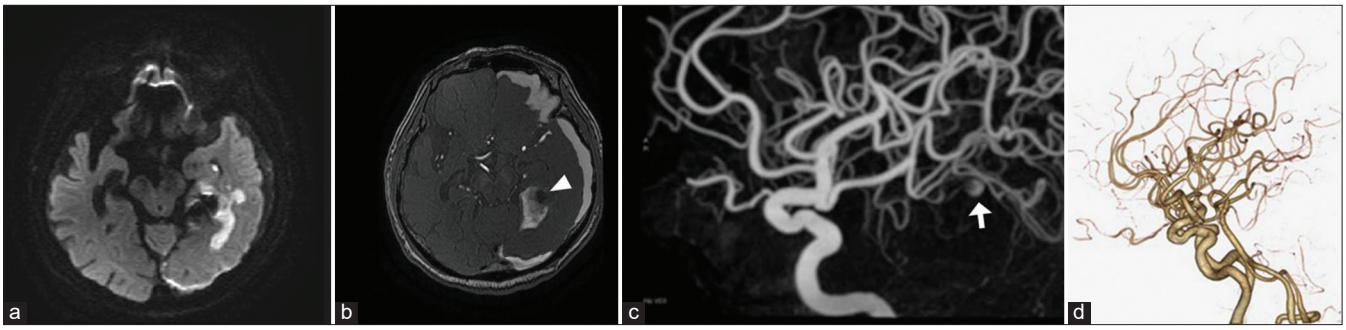

Figure 4: (a) Magnetic resonance (MR) imaging showing a small infarction in the temporal lobe. (b) MR angiography revealed the absence of intra-aneurysmal flow (arrowhead). (c and d) Computed tomography angiography revealed the disappearance of the aneurysm (arrow).

DISCUSSION

TICA are rare, accounting for under 1% of all intracranial aneurysms. Approximately 30% of TICAs occur in patients younger than 20 years old and are often associated with blunt head trauma.[2,10] The average time from initial trauma to aneurysm rupture is approximately 21 days, and the mortality rate is high, at up to 50%.[8] TICAs usually occur in the skull base and falx cerebri. A review of 171 cases by Komiyama *et al.* found that most occurred in the anterior circulation, and only a few cases have been reported in the PCA.[7] Zeal and Rhoton anatomically classified the PCA into four regions, defining the P3 segment as "posteriorly from the pulvinar in the lateral aspect of the quadrigeminal cistern and ends at the anterior limit of the calcarine fissure."[13] Most TICAs are located in the P1 and P2 segments of the PCA. To the best of our knowledge, only two TICA cases have been reported in the PCA distal to the P3 segment. This is the first report of a TICA in the distal PCA treated with NBCA [Table 1].[4,5] Furthermore, only two case reports have reported pseudoaneurysm occurring in the PITA branching from the P3 segment, resulting in ASDH without subarachnoid hemorrhage or cerebral contusion. This was a very rare case of TICA in terms of the rupture mechanism. TICAs can be difficult to diagnose. In our case, we took eight days to make the diagnosis, which is fast compared to other cases. To avoid delays in diagnosis, it is important to suspect TICA and perform 3D CT angiography or magnetic

Table 1: A literature review of patients presenting with traumatic distal posterior cerebral artery aneurysm.

Age/sex	Location	Timing of TICA diagnosis	Presentation	Treatment	References
30 M	P3/4	16 days	EDH, SDH	Endovascular (PAO using coil)	Ciochon *et al.*
42 M	P3	3 weeks	SDH only	Endovascular (PAO using coil)	Fuga *et al.*
88 M	P3	8 days	SDH only	Endovascular (PAO using NBCA)	Present case

EDH: Epidural hematoma, NBCA: N-butyl-2-cyanoacrylate, PAO: Parent artery occlusion, SDH: Subdural hematoma, TICA: Traumatic intracranial aneurysm, M: Male

resonance angiography in cases of ASDH with atypical hematoma location or clinical course.

Direct vascular injury or stretching was reported to cause TICA.[8] TICAs occurring in the PCA are most commonly seen in the P1 and P2 segments due to vascular injury at the tentorial free edge when intracranial pressure increases.[11] However, in the present case, the pseudoaneurysm was in an atypical location, the PITA branching from the P3 segment. The absence of cerebral contusion on the ipsilateral temporal base made it unlikely that the TICA formed due to damage to direct vascular injury at the skull base or tentorial free edge. Although ASDHs are typically formed due to cerebral contusion or the disruption of a bridging vein, it is important that the ASDH occurred in this case without cerebral contusion.

The mechanisms of forming an ASDH without cerebral contusion include (1) disruption to the adhesion between the cortical artery and the dura mater, (2) disruption to the dural branch of the cortical artery, and (3) spontaneous rupture of a cortical artery at the small arterial branch origin, particularly at points of potential weakness, such as right-angled branches.[1] The TICA rupture in the present case occurred by any of the above mechanisms and resulted in ASDH without subarachnoid hemorrhage or cerebral contusion.

The treatment strategies for common aneurysms in the PCA includes direct surgery and endovascular treatment. Direct surgery, such as aneurysm trapping, is a high-risk procedure with a complication rate of 13% and a mortality rate of 19%.[12] Endovascular treatment, on the other hand, has a reported complication rate of 9.1% and a mortality rate of 0%.[9] Therefore, endovascular treatment has become the main treatment option, especially for PCA aneurysms. Ciceri *et al*. reported seven PAO cases of cerebral aneurysms in the PCA, including two with aneurysms in the P2/3 segment and a brainstem infarction due to perforator occlusion from the P2 segment.[3] They suggested that PAO should be avoided in the P1/2 segment due to its large perfusion area. Still, it can be safely performed in the P3/4 segment due to the retrograde collateral flow from the superior temporal branch of the middle cerebral artery. In the present case, internal trapping was necessary to reduce the risk of rebleeding. If only proximal occlusion were used, TICA recurrence could occur due to collateral flow. After considering coils and NBCA as embolization materials, we selected low-concentration NBCA because the microcatheter could not be navigated to the distal part of the pseudoaneurysm. No pseudoaneurysm recurrence was observed postoperatively, and the patient recovered well.

CONCLUSION

We reported a rare case of ruptured TICA in the PITA branching from the P3 segment and presenting only with ASDH. Cerebrovascular assessment should be performed for patients with head trauma presenting with ASDH and an atypical hematoma location or with an atypical clinical course. When an enlarged hematoma caused by head injury is detected, the possibility of TICA must be considered.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Ethical approval

Not applicable.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Borzone M, Altomonte M, Baldini M, Rivano C. Pure subdural haematomas of arteriolar origin. Acta Neurochir (Wien) 1993;121:109-12.
2. Buckingham MJ, Crone KR, Ball WS, Tomsick TA, Berger TS, Tew JM Jr. Traumatic intracranial aneurysms in childhood: Two cases and a review of the literature. Neurosurgery 1988;22:398-408.
3. Ciceri EF, Klucznik RP, Grossman RG, Rose JE, Mawad ME. Aneurysms of the posterior cerebral artery: Classification and endovascular treatment. AJNR Am J Neuroradiol 2001;22:27-34.
4. Ciochon UM, Steuble Brandt EG, Stavngaard T. Acute tentorial subdural hematoma caused by rupture of the posterior cerebral artery after minor trauma-a case report. Diagnostics (Basel) 2020;10:175.
5. Fuga M, Tanaka T, Nogami R, Tachi R, Teshigawara A, Ishibashi T, *et al.* Delayed tentorial subdural hematoma caused by traumatic posterior cerebral artery aneurysm: A case report and literature review. Am J Case Rep 2021;22:e933771.
6. Holmes B, Harbaugh RE. Traumatic intracranial aneurysms: A contemporary review. J Trauma 1993;35:855-60.
7. Komiyama M, Morikawa T, Nakajima H, Yasui T, Kan M. "Early" apoplexy due to traumatic intracranial aneurysm--case report. Neurol Med Chir (Tokyo) 2001;41:264-70.
8. Larson PS, Reisner A, Morassutti DJ, Abdulhadi B, Harpring JE. Traumatic intracranial aneurysms. Neurosurg Focus 2000;8:e4.
9. Li XY, Li CH, Wang JW, Liu JF, Li H, Gao BL. Endovascular management of cerebral aneurysms of the posterior cerebral artery. Front Neurol 2021;12:700516.
10. Loevner LA, Ting TY, Hurst RW, Goldberg HI, Schut L. Spontaneous thrombosis of a basilar artery traumatic aneurysm in a child. AJNR Am J Neuroradiol 1998;19:386-8.
11. Meyer FB, Sundt TM Jr., Fode NC, Morgan MK, Forbes GS, Mellinger JF. Cerebral aneurysms in childhood and adolescence. J Neurosurg 1989;70:420-5.
12. Terasaka S, Sawamura Y, Kamiyama H, Fukushima T. Surgical approaches for the treatment of aneurysms on the P2 segment of the posterior cerebral artery. Neurosurgery 2000;47:359-64.
13. Zeal AA, Rhoton AL Jr. Microsurgical anatomy of the posterior cerebral artery. J Neurosurg 1978;48:534-59.

How to cite this article: Yamada K, Morimoto T, Fujimoto K, Nishioka T, Tokunaga H. Traumatic intracranial aneurysm in a distal posterior cerebral artery: A case report and literature review. Surg Neurol Int. 2023;14:428. doi: 10.25259/SNI_752_2023