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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
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Case Report

Successful endovascular occlusion of multiple fusiform aneurysms on the persistent primitive lateral basilovertebral anastomosis

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Received: 22 February 2025
Accepted: 14 April 2025
Published: 16 May 2025

DOI
10.25259/SNI_195_2025

Quick Response Code:

ABSTRACT

Background: The primitive lateral basilovertebral anastomosis (PLBA) is a transient embryonic vessel in the vertebrobasilar system that typically regresses during cerebellar artery development. Persistent PLBA (PPLBA), which forms a vertebrobasilar duplication, is a rare vascular anomaly. This is the first reported case of multiple fusiform aneurysms associated with a PPLBA.

Case Description: A 14-year-old girl was diagnosed with intracranial aneurysms before undergoing coronary artery bypass grafting surgery for myocardial infarction. Digital subtraction angiography showed two fusiform aneurysms on a PPLBA, connecting the left vertebral artery (VA) and the left anterior inferior cerebellar artery (AICA). After 3 years of follow-up, a new aneurysm developed at the origin of the PPLBA, proximal to the existing two aneurysms. Due to the AICA blood flow originating mainly from the basilar artery (BA) rather than the PPLBA, endovascular parent artery occlusion of the PPLBA was planned to prevent aneurysmal rupture and subarachnoid hemorrhage. Complete occlusion of all three aneurysms was achieved without complications.

Conclusion: Understanding the embryological anatomy of this rare vertebrobasilar duplication involving the PPLBA, AICA, BA, and VA facilitated the successful development of a therapeutic strategy. Aneurysms associated with PPLBA exhibit various vascular structures and can be treated effectively with tailored endovascular approaches.

Keywords: Endovascular parent artery occlusion, Fusiform aneurysms, Persistent primitive lateral basilovertebral anastomosis, Vertebrobasilar duplication

INTRODUCTION

The primitive lateral basilovertebral anastomosis (PLBA) is a transient embryonic vessel of the vertebrobasilar artery system, first described by Padget in 1948.[8] The PLBA runs parallel to the longitudinal neural arteries (LNAs) and typically regresses during cerebellar artery development.[8] Persistent PLBA (PPLBA) is a remnant of the PLBA that does not degenerate, with several anatomical variants, including vertebrobasilar duplications.[3] Vertebrobasilar

duplications arise from the fusion of two embryologically different vessels, with the lateral limb representing a persistent segment of the PLBA, sometimes forming a loop structure.[7] These duplications are rare anomalies distinct from basilar fenestrations, which result from incomplete fusion of paired LNAs. Few previous reports have shown intracranial aneurysms associated with variants involving PPLBA; however, most described aneurysms occur at the bifurcation points of the PPLBA and cerebellar arteries, rather than on the PPLBA itself, such as common bifurcation-type aneurysms.[2,4,6,9]

We report the first case of multiple fusiform aneurysms associated with a PPLBA. In this case, because the PPLBA formed a vertebrobasilar duplication, endovascular occlusion of the PPLBA with fusiform aneurysms was successfully performed.

CASE PRESENTATION

A 14-year-old girl was admitted with intracranial aneurysms detected on magnetic resonance angiography during preoperative assessment before coronary artery bypass grafting surgery for myocardial infarction. Digital subtraction angiography (DSA) revealed two fusiform aneurysms on the PPLBA, connecting the left vertebral artery (VA) and left anterior inferior cerebellar artery (AICA) [Figure 1a]. Although her medical history included acute myocardial infarction and coronary artery aneurysms, the diagnoses of Kawasaki disease and connective tissue disorders, such as Ehlers–Danlos syndrome, Marfan syndrome, and systemic lupus erythematosus, were ruled out based solely on elevated levels of SS-A antibodies. Initially, the fusiform aneurysms were closely observed without therapeutic intervention due to the uncertain natural history of these rare aneurysms.

After 3 years of follow-up, a new aneurysm developed at the origin of the PPLBA, proximal to the previous two aneurysms on DSA images [Figures 1b and c]. The two previously detected distal aneurysms were unchanged, with sizes of 4.3 mm × 3.4 mm and 4.9 mm × 2.9 mm, whereas the *de novo* aneurysm measured 3.3 mm × 2.5 mm. Blood flow to the left AICA from the basilar artery (BA) was observed before the flow from the PPLBA through the three fusiform aneurysms [Figures 1d and e]. Therefore, blood flow to the left AICA was confirmed mainly from the BA and not from the PPLBA. Moreover, no branching artery from the PPLBA, other than the AICA, was evident on high-resolution cone-beam computed tomography (CBCT) [Figure 1f]. As the fusiform aneurysms were located on the PPLBA in the loop structure of the vertebrobasilar duplication, which includes the PPLBA, AICA, BA, and VA [Figures 2a and b], parent artery occlusion of the PPLBA was planned. This intervention prevented rupture and subarachnoid hemorrhage due to the enlarging aneurysms.

A seven-French sheath was inserted into the right femoral artery, followed by systemic heparinization under general anesthesia. A seven-French balloon guiding catheter (Optimo; Tokai Medical, Aichi, Japan) was placed in the V1 segment of the left VA. A distal access catheter (AXS Vecta 46; Stryker Neurovascular, Fremont, CA, USA) was then positioned in the V4 segment of the left VA. Although the PPLBA between the fusiform aneurysms had a small diameter, the microcatheter (Excelsior SL-10 Straight; Stryker, Kalamazoo, MI, USA) successfully reached the interior of the most distal aneurysm [Figure 3a]. The PPLBA and aneurysms were completely occluded using 18 coils from the distal to the proximal aneurysm without any intraprocedural complications [Figures 3b and c]. Postoperative diffusion-weighted imaging revealed no cerebral infarction in the brainstem or cerebellum and no neurological defects [Figure 3d]. She was discharged with a modified Rankin scale score of 0. No recurrence of fusiform aneurysms in the PPLBA was observed 6 months after the intervention.

DISCUSSION

We report the first successful treatment of multiple fusiform aneurysms of the PPLBA through parental artery occlusion. The duplicated vertebrobasilar system, comprising the BA and PPLBA, facilitated endovascular parent artery occlusion of the PPLBA.

Aneurysm formation has rarely been observed in the PPLBA system. Six reports, including the present case, of intracranial aneurysms associated with the PPLBA are listed in Table 1. Four (67%) aneurysms were saccular and formed at the bifurcation of the PLBA system. The locations of these aneurysms vary from case to case and include bifurcations associated with the PPLBA, BA, VA, AICA, and the persistent primitive hypoglossal artery in the PLBA system. The mechanism of aneurysm formation in the PLBA system remains unknown because there are no pathological reports on aneurysms associated with PPLBA; however, a previous autopsy study observed gaps in the tunica media structure at the vascular junction of segmental duplications of the BA where the fusion of the primitive LNAs stopped.[1] Since the partially vulnerable vascular structure of the embryological junction resembles normal arterial bifurcations, previous reports have argued that similar to BA fenestration or other arterial bifurcations, hemodynamic stress due to abnormal vascular structures also contributes to aneurysm formation on the bifurcations associated with PPLBA.[4,9] Three of the four saccular aneurysms (75%) ruptured. They successfully treated ruptured saccular aneurysms with endovascular coiling assisted by balloons or intracranial stents, similar to other endovascular therapies for common bifurcation type aneurysms.[2,4,9]

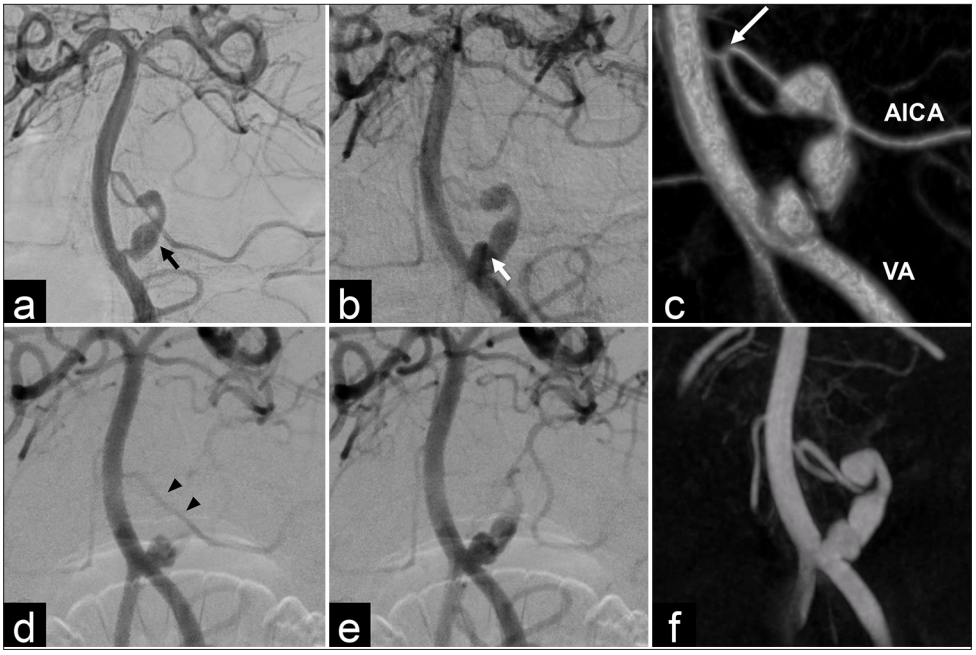

Figure 1: DSA images at the time of diagnosis. (a) Two fusiform aneurysms on a PPLBA connecting the left VA and left anterior inferior cerebellar artery at the first diagnosis. A black arrow indicates the fusiform aneurysms. (b) An additional *de novo* aneurysm appeared at the origin of the PPLBA just proximal to the previous two aneurysms after 3 years of follow-up (a white arrow). (c) A 3D-DSA image of the multiple aneurysms. The PPLBA, AICA, BA, and VA form a loop structure of the vertebrobasilar duplication. A white arrow indicates the connection between the PPLBA and AICA. (d and e) Blood flow of left AICA was observed in the earlier phase through the BA than the PPLBA. Early (d) and late phase (e). Black arrowheads indicate the blood flow of the AICA from the BA. (f) High-resolution CBCT showed no obvious perforators on the aneurysms and PPLBA. DSA: Digital subtraction angiography, PPLBA: Persistent primitive lateral basilovertebral anastomosis, VA: Vertebral artery, AICA: Anterior inferior cerebellar artery, BA: Basilar artery, CBCT: Cone-beam computed tomography.

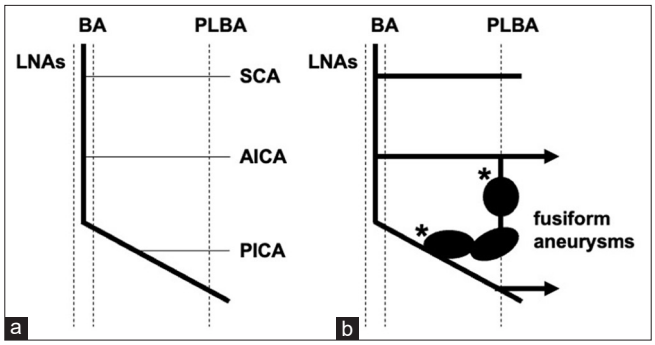

Figure 2: Normal embryonic development of the arteries in the posterior circulation and schema of the present case. (a) Normal development. (b) The schema of the present case. The three aneurysms were on the PPLBA, forming the loop structure of the vertebrobasilar duplication. Two asterisks indicate the distance of the coil embolization. LNAs: Longitudinal neural arteries, PLBA: Primitive lateral basilovertebral anastomosis, SCA: Superior cerebellar artery, PICA: Posterior inferior cerebellar artery

The other two aneurysms (33%), including this case, were fusiform. Ota *et al*.[6] reported a ruptured fusiform aneurysm at the origin of the posterior inferior cerebellar artery (PICA). The PPLBA connects the AICA and the aneurysm to the PICA. Microsurgical clipping at the origin of the aneurysm on the PICA and dome clipping were performed while preserving the vascular supply from the AICA to the PICA hemispheric branch through part of the fusiform aneurysm. Considering that occlusion of the entire aneurysm with the parent artery occlusion was structurally impossible, their strategy of flow alteration could have resulted in incomplete occlusion of the aneurysm. Notably, they observed no recurrence of the aneurysm for 2 years. As in the previous case, complete occlusion of the fusiform aneurysms in the present case was not achieved without occlusion of the parent artery. The PPLBA is usually associated with the superior cerebellar artery, AICA, or PICA, because it embryologically connects the three developing cerebellar arteries from the LNAs. Therefore, the preservation of blood supply to the cerebellar arteries is crucial in the treatment of aneurysms associated with PPLBA. In the present case, the PPLBA connected the AICA and the left VA, forming

Table 1: Literature review of aneurysms associated with PPLBA.

Case	Article	Age	Sex	Aneurysm	Symptom	Location of aneurysm	Treatment
1	Yagi *et al*.[9]	62	M	Saccular	Ruptured	Bifurcation of the BA and AICA	Coiling
2	Joshi *et al*.[4]	62	M	Saccular	Ruptured	Bifurcation the VA and BA	Coiling
3	Gregg and Gailloud[3]	45	F	Saccular	Unruptured	Bifurcation of the VA and PPLBA	NA
4	Genkai *et al*.[2]	66	F	Saccular	Ruptured	Bifurcation of the BA and PPHA	Coiling
5	Ota *et al*.[7]	48	M	Fusiform	Ruptured	On the proximal PICA	Microsurgical trapping
6	Present case	16	F	Fusiform	Unruptured	On the PPLBA	Parent artery occlusion

PPLBA: Persistent primitive lateral basilovertebral anastomosis, BA: Basilar artery, AICA: Anterior inferior cerebellar artery, VA: Vertebral artery, PPHA: Persistent primitive hypoglossal artery, PICA: Posterior inferior cerebellar artery, NA: Not Available

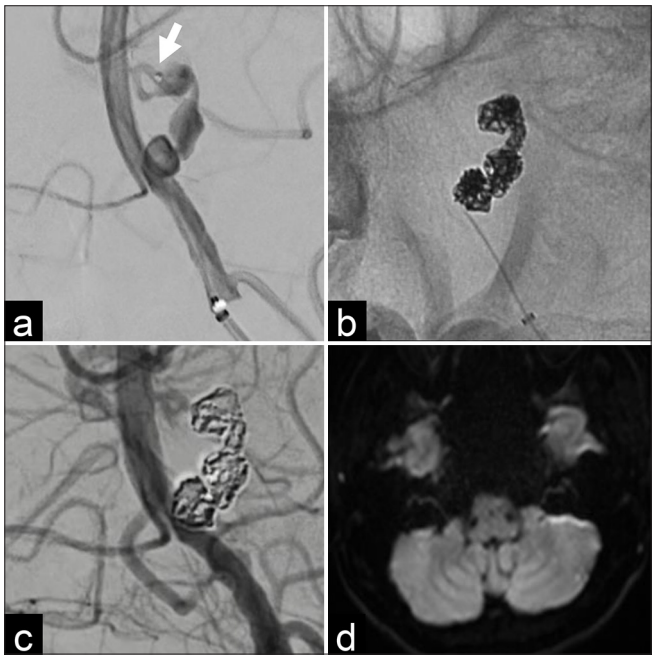

Figure 3: DSA and MRI images of perioperative periods. (a) The tip of the microcatheter was inserted into the distal fusiform aneurysm. A white arrow indicates the tip of the microcatheter. (b) Coil mass after complete occlusion of the multiple aneurysms. (c) DSA showed complete occlusion of the multiple aneurysms. (d) A DWI image after endovascular therapy. DSA: Digital subtraction angiography, MRI: Magnetic resonance imaging, DWI: Diffusion-weighted imaging.

a vertebrobasilar duplication. DSA revealed that the blood flow to the AICA was primarily from the BA rather than from the PPLBA. Although a balloon occlusion test was not performed, parental artery occlusion with multiple fusiform aneurysms on the PPLBA was achieved, leaving blood flow to the AICA.

In the case of the BA fenestration, perforating arteries are usually involved because it is consisted of the LNAs.[7] Thus, a therapeutic strategy for parental artery occlusion could not be established. However, whether the perforating arteries would branch off from the PPLBA remains unknown. Previous reports have suggested that there are many transverse channels between the LNA and PLBA.[7] Moreover, the PLBA and posterior lateral spinal artery feeding the spinal cord represent the cranial and spinal portions of the same vascular channels.[5] Based on these descriptions, the existence of perforators in the PPLBA could not be ruled out in the present case. Since the natural history of aneurysms on the PPLBA was also unknown due to their rarity, the patient was initially followed up without therapeutic intervention. However, after rapid aneurysm growth, parent artery occlusion was planned to prevent rupture. Although high-resolution CBCT showed no obvious perforating artery from the PPLBA, informed consent was obtained before the occlusion of the parent artery regarding the risk of cerebral infarction due to occlusion of the perforating artery. Multiple fusiform aneurysms were successfully treated without cerebral infarctions, indicating the absence of a perforating artery in the PPLBA in the present case. In a previous case of a PPLBA-related aneurysm treated with microsurgery, no branch of the perforating artery was observed around the aneurysm or from the PPLBA in the operative field.[6] These two cases imply that parent artery occlusion, including a fusiform aneurysm on the PPLBA, can be achieved without cerebral infarction in some cases with a loop structure of vertebrobasilar duplication.

The etiology of fusiform and bifurcated aneurysms may differ, potentially involving factors such as hemodynamic conditions, genetic predisposition, or systemic vascular fragility factors. Given the patient's history of multiple coronary aneurysms of unknown etiology, both hemodynamic stress and systemic vascular fragility may have contributed to aneurysm formation in the intracranial PPLBA.

CONCLUSION

In this case, the rare looped vertebrobasilar duplication, encompassing the PPLBA, AICA, BA, and VA, facilitated endovascular occlusion of the PPLBA and associated fusiform aneurysms. Aneurysms associated with PPLBA

exhibit diverse vascular morphologies and can be effectively treated by understanding their embryological development.

Ethical approval: The Institutional Review Board approval is not required.
Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.
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.

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How to cite this article: Ikedo T, Itazu T, Imamura H, Inoue Y, Tabata S, Shimonaga K, *et al*. Successful endovascular occlusion of multiple fusiform aneurysms on the persistent primitive lateral basilovertebral anastomosis. Surg Neurol Int. 2025;16:185. doi: 10.25259/SNI_195_2025