

Case Report

De Novo Aneurysm on the Posterior Cerebral Artery: A Case Report and Literature Review

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De novo aneurysm formation is a rare entity of cerebral aneurysms. The authors describe a 19-year-old man presenting with spontaneous intracerebral hemorrhage of unknown etiology. The initial cerebral angiography revealed no identifiable vascular lesion. A few weeks following a surgical evacuation of the hematoma, a tiny saccular aneurysm was incidentally found on the distal posterior cerebral artery (PCA) remote from the site of the primary ictus. Several rationales indicated that it was compatible with a cerebral aneurysm of infective etiology. The aneurysm was successfully treated by antibiotic therapy alone. To the authors' knowledge, de novo aneurysm on the PCA has rarely been reported.

Keywords: Cerebral de novo aneurysm, Posterior cerebral artery, Cerebral hemorrhage

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Cerebral de novo aneurysm (CDNA), a rare entity, is defined as aneurysm arising on previously normal intracranial vasculature demonstrated by a cerebral angiography, computerized tomography angiography or surgical exploration⁽¹⁾. Only a few of cases with this kind of aneurysm have been reported. Most of them had newly developed aneurysm following exclusion of the initial aneurysm from the brain circulation⁽²⁾ or after therapeutic occlusion of the carotid artery⁽³⁾. CDNA rarely occurs in the posterior cerebral circulation, particularly on the posterior cerebral artery (PCA). The present authors describe a patient with CDNA originating from the distal PCA successfully treated by antimicrobial therapy and review of the relevant literature of CDNA originating from the posterior cerebral circulation.

Case Report

A healthy 19-year-old man presented with sudden right-sided weakness and aphasia. Physical examination revealed a conscious individual with motor aphasia. Right hemiplegia and facial palsy were elicited. The patient had no history of hypertension, heart disease, connective tissue disease, hematologic

disorder, intravenous drug or alcohol use, cigarette smoking, previous head injury and family history of stroke. Cranial CT scan displayed a sizable acute hematoma in the left frontal lobe just anterior to the precentral gyrus (Fig. 1A). Digital subtraction cerebral angiography (DSA) revealed no demonstrable intracranial aneurysm or vascular malformation (Fig. 1C, 1D, 2A). He underwent a surgical evacuation of the retained hematoma through the left frontal craniotomy. Intraoperatively, there was no any lesion such as aneurysm, vascular malformation, or neoplasm that could explain the occurrence of spontaneous hemorrhage. Pathological section revealed organized blood clots without evidence of hemorrhagic brain tumor. After the operation, motor function of the limb and motor aphasia gradually improved with time. Early postoperative cranial CT scan exhibited disappearance of the hematoma (Fig. 1B). The patient was discharged from the hospital without complication.

Six weeks later, the second cerebral angiography was performed to investigate an occult vascular lesion. A 3-mm end-on saccular aneurysm arising from the posterior parietal branch of the right PCA was unexpectedly encountered (Fig. 2B). Astonishingly, the cerebral aneurysm was situated on the contralateral side of the previously evacuated intracerebral hematoma. Echocardiography and electrocardiography were unremarkable. Two specimens of blood culture revealed no growth of organism at the seventh day. On account of rapid

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growth, distal location on the PCA and remote site from the initial intracerebral hemorrhage, a cerebral aneurysm of infectious origin was highly suspected. A 6-week course of high-dose intravenous cloxacillin and cefotaxime administration was promptly initiated. Six weeks later, the third cerebral angiography revealed regression in size of the aneurysm (Fig. 2C). The fourth cerebral angiography at ten subsequent weeks demonstrated complete resolution of the aneurysm (Fig. 2D). Currently, motor and language functions of the patient have completely returned to normal.

Discussion

CDNA is a rare entity of intracranial aneurysms. The occurrence rate is approximately 0.28-1.62% per year⁽⁴⁾. Major risk factors in novel aneurysm formation include female gender, middle age, arterial hypertension, cigarette smoking, and family history of cerebrovascular disease^(2,5). The anterior cerebral circulation is the most common site of CDNA while those originating from the posterior cerebral circulation are very rare⁽⁶⁾.

Pathogenesis of CDNA remains obscure. Interaction between hemodynamic stress and structural

weakness plays the major role in development of the aneurysm. CDNA can be encountered after therapeutic occlusion of the carotid artery. The ligation of the vessel results in hemodynamic changes and increased blood flow for supplying the contralateral circulation. Therefore, the aneurysm often emerges on the contralateral side, particularly on the anterior communicating and internal carotid arteries^(7,8). In patients with brain arteriovenous malformation (AVM), CDNA usually arises on the feeding artery due to abnormal high flow. This flow-related aneurysm often decreased in size after removal of AVM or aberrant hemodynamic stress⁽⁹⁾. Aneurysm clipping may cause alteration of hemodynamics leading to de novo formation of aneurysm. Abnormal high flow in the vessel results in smooth muscle damage and release of local factors related to aneurysm progression, such as nitric oxide synthase^(7,10). Furthermore, congenital defect in the tunica media and acquired factors, such as degenerative changes, arterial hypertension, is also involved in the pathogenesis⁽⁷⁾.

In the literature, there has been a small number of case reports or case series of CDNA arising from the posterior cerebral circulation (Table 1)^(2,5,6,11-31). Of

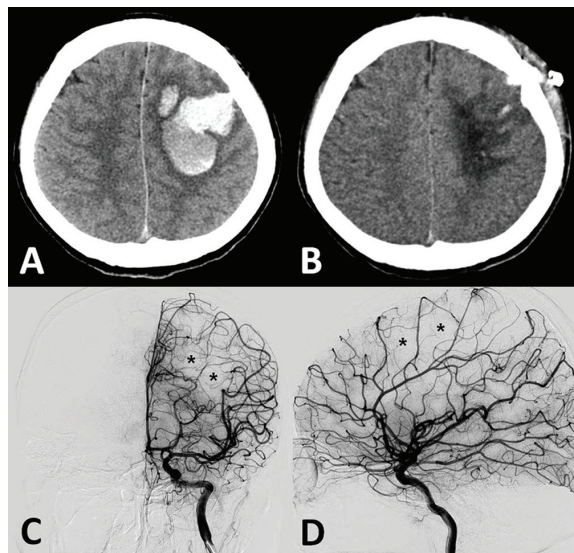


Fig. 1 Axial non-contrast enhanced cranial CT showing: (A) the left frontal lobe hematoma with minimal midline shift; and (B) disappearance of the hematoma following a surgical removal through the left frontal craniotomy. DSA of the left internal carotid artery in anteroposterior (C) and lateral (D) views before the surgical evacuation showing the avascular area (asterisk) occupied by the retained hematoma without identifiable vascular lesion

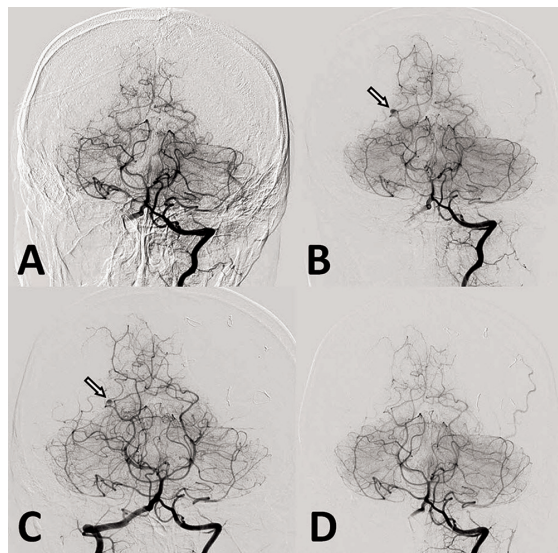


Fig. 2 Sequential DSA of the vertebral artery showing: (A) no demonstrable aneurysmal lesion on the initial angiography before the surgery; (B) the second angiography showing a de novo saccular aneurysm (arrow) emerging from the posterior parietal branch of the right PCA; (C) six weeks later, the aneurysm slightly regressed in its size (arrow); (D) the vanishing aneurysm on the sixteenth week following the diagnosis

Table 1. Published reports of de novo aneurysms arising on the posterior cerebral circulation

Authors (year)(ref)	Sex	Initial aneurysm or other lesion		De no aneurysm	
		Age	Location	Age	Location
Roski et al. (1981) ⁽¹¹⁾	F	31	R ICA	33	BA, L ICA
Miller et al. (1985) ⁽¹²⁾	F	38	L MCA	49	BA-SCA, L PCoA*
	F	37	R MCA	41	Basilar tip, ACoA*
	F	28	R ICA-PCoA	48	Basilar tip
Koeleveld et al. (1991) ⁽¹³⁾	F	13	L ICA tip, L ICA-AChA	26	Basilar tip, R ICA tip*, L ACA*, L MCA*
Kojima et al. (1996) ⁽¹⁴⁾	F	61	R VA-PICA, L MCA, basilar tip	67	L distal PICA
Sugiura et al. (1997) ⁽¹⁵⁾	F	65	ACoA	78	Basilar tip
Kanemoto et al. (1997) ⁽¹⁶⁾	M	46	R MCA	54	R VA-PICA
Johnston et al. (1998) ⁽¹⁷⁾	M	10	L cervical to cavernous ICA	12	VB (both distal VAs to proximal BA)
	M	6	L cervical to supraclinoid ICA	11	VB (R distal VA to proximal BA)
	F	10	L cervical-petrous ICA	21	VB (VA union to distal BA)
Tsutsumi et al. (2001) ⁽⁵⁾	M	57	R ICA	63	BA, R MCA
Wolf et al. (2002) ⁽¹⁸⁾	F	58	R cavernous ICA, midbrain AVM	66	L PCA
Osawa et al. (2002) ⁽¹⁹⁾	M	29	L MCA	35	R VA-PICA, R ACA*, L PCoA*, both ICAs & MCAs*
Brock et al. (2003) ⁽²⁰⁾	M	48	Cerebellar AVM	48	BA
Lee and Brophy (2003) ⁽²¹⁾	M	19	R MCA, L ICA tip	42	Basilar tip
Suri and Mehta (2003) ⁽²²⁾	M	22	Left AICA	22	L VB junction
Yoshioka et al. (2003) ⁽²³⁾	F	43	R ICA-PCoA	52	L distal PCA (P3)
Yoneoka et al. (2004) ⁽²⁴⁾	F	64	L ICA tip	66	R VA-PICA
	F	54	L PCA (P2-P3)	67	R proximal PCA (P1)
Kang et al. (2005) ⁽²⁵⁾	F	30	R petrous-cavernous ICA	30	BA
Fujimoto et al. (2005) ⁽²⁶⁾	F	72	R MCA, L cavernous ICA, L ICA-PCoA	77	Basilar tip
Shimokawara et al. (2007) ⁽²⁷⁾	F	58	ACoA, L ICA-PCoA	65	L BA-SCA
Kim et al. (2007) ⁽²⁸⁾	F	50	ACoA	66	R proximal PCA (P1)
	F	69	R MCA	70	Basilar tip
	F	41	L ICA-PCoA	58	Basilar tip
	F	36	R MCA	46	Basilar tip
Schebesch et al. (2008) ⁽²⁹⁾	M	37	ACoA	37	Basilar tip
Hirota et al. (2009) ⁽³⁰⁾	M	64	L distal PICA	67	L distal PICA
Kawahara et al. (2010) ⁽³¹⁾	F	48	L MCA	53	Basilar tip
	F	49	R proximal ACA (A1)	52	R BA-SCA
Rahmah et al. (2011) ⁽²⁾	F	69	L MCA	80	BA
	F	71	Both ICAs & MCAs	81	BA
Suzuki et al. (2011) ⁽⁶⁾	F	62	R ICA, L cavernous ICA	62	Basilar tip
Current case	M	19	L frontal hematoma	19	R distal PCA

Age in years; ACA = anterior cerebral artery; AChA = anterior choroidal artery; ACoA = anterior communicating artery; AICA = anterior inferior cerebellar artery; AVM = arteriovenous malformation; BA = basilar artery; F = female; ICA = internal carotid artery; L = left; M = male; MCA = middle cerebral artery; PCA = posterior cerebral artery; PCoA = posterior communicating artery; PICA = posterior inferior cerebellar artery; R = right; SCA = superior cerebellar artery; VA = vertebral artery; VB = vertebrobasilar; * indicates concurrent de novo aneurysm of the anterior cerebral circulation

the 34 patients, 24 (70.6%) were female. All individuals had the CDNA developing after the treatment of the initial aneurysms, therapeutic occlusion of the carotid artery or embolization of brain AVM, whereas CDNA of the present case showed uniqueness that was not associated with such kinds of cerebral events or the major risk factors. Among CDNA of the posterior circulation, the most common location was the basilar artery bifurcation (12 of 34 cases). To the authors knowledge, newly developing aneurysms on the PCA were extremely rare. Only four cases of de novo PCA aneurysm have been previously reported in existing medical literature^(18,23,24,28). The presented patient who had aneurysm emerging from the PCA is considered to be an unusual case of CDNA in terms of the location as well.

Even though evidence of infection, especially bacterial endocarditis, had not been discovered (cryptogenic origin) in the presented patient, an unruptured PCA aneurysm of infectious origin was highly suspicious because the aneurysm developed rapidly within only 6 weeks and it was situated on peripheral location of the cerebral vasculature. Retrospectively, the frontal lobe hematoma might be also caused by occult small mycotic aneurysm although it could not be encountered intraoperatively. From a therapeutic point of view, based on expert opinion, management of unruptured infective intracranial aneurysm should be initiated with appropriate antimicrobial therapy with sequential angiography to document amelioration or resolution⁽³²⁾. A number of studies demonstrated resolution of small-sized (10 mm or less) infective intracranial aneurysm with antibiotic therapy alone^(33,34). The presented case with a small-sized one also displayed angiographic improvement and subsequent resolution of the aneurysm following medical therapy alone. Importantly, however, long-term neuroradiographic follow-up is mandatory for aneurysm surveillance in this young individual.

Conclusion

CDNA rarely presents on the PCA. Rapid spontaneous formation and peripheral distribution of cerebral aneurysm may imply infective etiology, even though source of infection has not been positively identified. Small unruptured intracranial infectious aneurysm can be successfully treated by antimicrobial therapy with serial angiography. Interval angiographic follow-up is essential to determine response to the treatment.

Potential conflicts of interest

None.

References

1. van der Schaaf IC, Velthuis BK, Wermer MJ, Majoie C, Witkamp T, de Kort G, et al. New detected aneurysms on follow-up screening in patients with previously clipped intracranial aneurysms: comparison with DSA or CTA at the time of SAH. *Stroke* 2005; 36: 1753-8.
2. Rahmah NN, Horiuchi T, Kusano Y, Sasaki T, Hongo K. De novo aneurysm: case reports and literature review. *Neurosurgery* 2011; 69: E761-6.
3. Wang YY, Rosenfeld JV, Lyon SM, O'Brien BJ. Rapid development of a de novo intracranial aneurysm following carotid occlusion. *J Clin Neurosci* 2008; 15: 324-30.
4. Cheong JJ, Ghinea N, van Gelder JM. Estimating the annual rate of de novo multiple aneurysms: three statistical approaches. *Neurosurg Focus* 2004; 17: E8.
5. Tsutsumi K, Ueki K, Morita A, Usui M, Kirino T. Risk of aneurysm recurrence in patients with clipped cerebral aneurysms: results of long-term follow-up angiography. *Stroke* 2001; 32: 1191-4.
6. Suzuki MT, Aguiar GB, Jory M, Conti ML, Veiga JC. De novo basilar tip aneurysm. Case report and literature review. *Neurocirugia (Astur)* 2011; 22: 251-4.
7. Fujiwara S, Fujii K, Fukui M. De novo aneurysm formation and aneurysm growth following therapeutic carotid occlusion for intracranial internal carotid artery (ICA) aneurysms. *Acta Neurochir (Wien)* 1993; 120: 20-5.
8. Briganti F, Cirillo S, Caranci F, Esposito F, Maiuri F. Development of "de novo" aneurysms following endovascular procedures. *Neuroradiology* 2002; 44: 604-9.
9. Batjer H, Suss RA, Samson D. Intracranial arteriovenous malformations associated with aneurysms. *Neurosurgery* 1986; 18: 29-35.
10. Fukuda S, Hashimoto N, Naritomi H, Nagata I, Nozaki K, Kondo S, et al. Prevention of rat cerebral aneurysm formation by inhibition of nitric oxide synthase. *Circulation* 2000; 101: 2532-8.
11. Roski RA, Spetzler RF, Nulsen FE. Late complications of carotid ligation in the treatment of intracranial aneurysms. *J Neurosurg* 1981; 54: 583-7.
12. Miller CA, Hill SA, Hunt WE. "De novo" aneurysms. A clinical review. *Surg Neurol* 1985;

- 24: 173-80.
13. Koeleveld RF, Heilman CB, Klucznik RP, Shucart WA. De novo development of an aneurysm: case report. *Neurosurgery* 1991; 29: 756-9.
 14. Kojima A, Nakamura T, Takayama H, Harada S, Takamiya Y. A case of de novo aneurysm of the distal posterior inferior cerebellar artery with intraventricular hemorrhage. *No Shinkei Geka* 1996; 24: 469-73.
 15. Sugiura Y, Miyamoto T, Takehara S, Hiramatsu H, Akamine S, Uchiyama H. Basilar artery aneurysm associated with agenesis of unilateral internal carotid artery: two case reports. *No Shinkei Geka* 1997; 25: 385-90.
 16. Kanemoto Y, Hisanaga M, Bessho H. De novo vertebral artery-posterior inferior cerebellar artery aneurysm: a case report. *Surg Neurol* 1997; 47: 473-5.
 17. Johnston SC, Halbach VV, Smith WS, Gress DR. Rapid development of giant fusiform cerebral aneurysms in angiographically normal vessels. *Neurology* 1998; 50: 1163-6.
 18. Wolf RL, Imbesi SG, Galetta SL, Hurst RW, Sinson GP, Grossman RI. Development of a posterior cerebral artery aneurysm subsequent to occlusion of the contralateral internal carotid artery for giant cavernous aneurysm. *Neuroradiology* 2002; 44: 443-6.
 19. Osawa H, Fukui K, Otsuka G, Hattori K, Satake T, Miyazaki M. De novo cerebral aneurysms manifesting as repeated subarachnoid hemorrhage and cerebral ischemic stroke—case report. *Neurol Med Chir (Tokyo)* 2002; 42: 391-5.
 20. Brock S, Giombini S, Ciceri E. Development and rupture of a de novo basilar artery aneurysm after surgical removal of a cerebellar arteriovenous malformation. *Acta Neurochir (Wien)* 2003; 145: 1117-20.
 21. Lee GY, Brophy BP. Recurrent subarachnoid haemorrhage from a de novo basilar bifurcation aneurysm: a case report. *J Clin Neurosci* 2003; 10: 250-2.
 22. Suri A, Mehta VS. Giant vertebrobasilar junction aneurysms: unusual cases. *Neurol India* 2003; 51: 84-6.
 23. Yoshioka H, Hotta T, Taniguchi E, Hashimoto N, Kinoshita Y, Ohba S, et al. De novo distal posterior cerebral artery aneurysm. *Surg Neurol* 2003; 60: 534-9.
 24. Yoneoka Y, Takeda N, Akira I, Ibuchi Y, Kumagai T, Sugai T, et al. Ruptured de novo intracranial aneurysms. *Acta Neurochir (Wien)* 2004; 146: 979-81.
 25. Kang HS, Oh CW, Han MH, Byun HS, Han DH. Treatment of a sequential giant fusiform aneurysm of the basilar trunk. *Korean J Radiol* 2005; 6: 125-9.
 26. Fujimoto K, Kimura R, Iida J, Kawaguchi S, Sakaki T, Nakagawa H, et al. De novo basilar top aneurysm in an elderly patient treated with Guglielmi detachable coils. *AJNR Am J Neuroradiol* 2005; 26: 915-6.
 27. Shimokawara T, Shimomura T, Okumura Y, Sakaki T. Three cases of de novo aneurysms. *No Shinkei Geka* 2007; 35: 365-70.
 28. Kim dH, Jung JY, Lee JW, Huh SK, Lee KC. A clinical analysis of twelve cases of ruptured cerebral de novo aneurysms. *Yonsei Med J* 2007; 48: 30-4.
 29. Schebesch KM, Doenitz C, Zoepfel R, Finkenzeller T, Brawanski AT. Recurrent subarachnoid hemorrhage caused by a de novo basilar tip aneurysm developing within 8 weeks after clipping of a ruptured anterior communicating artery aneurysm: case report. *Neurosurgery* 2008; 62: E259-60.
 30. Hirota Y, Ikeda N, Tamura Y, Yokoyama K, Yamada Y, Kuroiwa T, et al. A de novo distal PICA aneurysm occurring 3 years after clipping of another distal PICA aneurysm: a case report. *No Shinkei Geka* 2009; 37: 905-11.
 31. Kawahara I, Nakamoto M, Matsuo Y, Tokunaga Y. De novo basilar head aneurysms—two case reports. *Neurol Med Chir (Tokyo)* 2010; 50: 54-6.
 32. Peters PJ, Harrison T, Lennox JL. A dangerous dilemma: management of infectious intracranial aneurysms complicating endocarditis. *Lancet Infect Dis* 2006; 6: 742-8.
 33. Morawetz RB, Karp RB. Evolution and resolution of intracranial bacterial (mycotic) aneurysms. *Neurosurgery* 1984; 15: 43-9.
 34. Meena AK, Sitajayalakshmi S, Prasad VS, Murthy JM. Mycotic aneurysm on posterior cerebral artery: resolution with medical therapy. *Neurol India* 2000; 48: 276-8.

หลอดเลือดแดงโป่งพองซึ่งเกิดขึ้นใหม่ในตำแหน่งหลอดเลือดแดงโพสทีเรียซีรีบริด: รายงานผู้ป่วยและบททวนวรรณกรรมทางการแพทย์

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หลอดเลือดแดงโป่งพองซึ่งเกิดขึ้นใหม่เป็นหลอดเลือดแดงโป่งพองในสมองที่พบได้น้อย คณะผู้นิพนธ์รายงานผู้ป่วยชายอายุ 19 ปี ซึ่งมารับการรักษาด้วยภาวะเลือดออกในสมองโดยไม่ทราบสาเหตุ การตรวจหลอดเลือดสมองทางรังสีครั้งแรกไม่พบสาเหตุของภาวะเลือดออกดังกล่าว หลังจากนั้นไม่กี่สัปดาห์หลังการผ่าตัดนำก้อนเลือดในสมองออกได้ตรวจพบหลอดเลือดแดงโป่งพองขนาดเล็กในตำแหน่งหลอดเลือดแดงโพสทีเรียซีรีบริดส่วนปลายซึ่งอยู่ในตำแหน่งที่ไกลจากตำแหน่งของเลือดออกในสมองครั้งแรก หลายเหตุผลบ่งชี้ว่าหลอดเลือดโป่งพองดังกล่าวเข้าได้กับหลอดเลือดแดงโป่งพองในสมองซึ่งมีสาเหตุจากการติดเชื้อหลอดเลือดโป่งพองในผู้ป่วยรายนี้ได้รับการรักษาสำเร็จโดยการให้ยาปฏิชีวนะ จากการค้นคว้าของคณะผู้นิพนธ์พบว่า มีรายงานผู้ป่วยที่มีหลอดเลือดโป่งพองซึ่งเกิดขึ้นใหม่ในตำแหน่งหลอดเลือดแดงโพสทีเรียซีรีบริดจำนวนน้อยมาก
