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Case Reports

# *De novo* development of a cerebral arteriovenous malformation following radiation therapy: Case report and an update to classical arteriovenous malformation nomenclature



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#### ABSTRACT

Cerebral arteriovenous malformations (AVM) are traditionally considered primary congenital lesions that result from embryological aberrations in vasculogenesis. Recent insights, however, suggest that these lesions may be secondary to a vascular insult such as ischemia or trauma. Herein, the authors present a rare case of a secondary cerebral AVM, occurring in a young girl who received prior cranial radiation therapy. At age 3 years, she underwent surgical resection, chemotherapy, and photon radiation therapy for treatment of a fourth ventricular ependymoma. At age 19 years, she developed new onset seizures and was found to have a left medial temporal lobe AVM. Her seizures were managed successfully with anti-epileptic medications and the AVM was treated with proton radiation therapy. This case highlights a rare but possible vascular sequela of radiation therapy and adds to the growing body of evidence that cerebral AVM may arise as secondary lesions.

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#### 1. Introduction

Cerebral arteriovenous malformations (AVM) are traditionally considered congenital lesions arising as a result of incomplete fetal vascular maturation [1,2]. Apart from known genetic syndromes such as hereditary hemorrhagic telangiectasia (HHT) or cerebrofacial arteriovenous metameric syndromes, it has yet to be definitively shown that AVM are strictly congenital phenomena [3,4].

In contrast, there is a small, but growing, body of literature supporting "spontaneous formation" of these lesions, with 19 cases at the time of writing [5–19]. *De novo* formation of cerebral AVM can be primary or idiopathic, but there is mounting evidence that these lesions may be secondary to cerebral traumatic or ischemic insult [2]. In the present report, we present a case of cerebral AVM formation 13 years following photon radiation therapy for an ependymoma, and highlight the growing distinction between primary and secondary cerebral AVM. We also use this new insight as a platform to elaborate novel nomenclature for AVM. To our knowledge this case is the first full report in the

literature of an AVM developing secondary to photon radiation therapy.

#### 2. Case presentation

A 16-month-old girl born prematurely at 29 weeks gestation, but with an otherwise uncomplicated developmental history without family history of AVM presented to the neurosurgery service with developmental regression manifested by a progressive inability to sit upright without falling, loss of balance, and rapid growth in head circumference. MRI of the brain revealed a lesion within the fourth ventricle causing outflow obstruction and ventriculomegaly. She underwent a midline suboccipital craniotomy with gross total resection of the lesion. Pathological examination demonstrated an ependymoma.

Post-operatively, she required a ventriculo-peritoneal shunt for persistent ventriculomegaly and hydrocephalus. She underwent chemotherapy with cisplatin, vincristine, and cyclophosphamide prior to consideration of radiation treatment. After completing chemotherapy at age 3 years, she underwent photon radiation therapy to the posterior fossa at a dose of 4680 Gy with a 720 Gy boost over 30 fractions (Fig. 1). Radiation and chemotherapy were tolerated well and follow-up yearly MRI were stable for 16 years.



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**Fig. 1.** Sagittal plain radiograph photon radiation plan demonstrating the area targeted to receive 4680 Gy as well as the area of 720 Gy boost, illustrating inclusion of the middle fossae in the treatment plan.

At age 19 years, she experienced a partial complex seizure, with seizure semiology described as decreased responsiveness, right hand rhythmic grasping, and right facial twitching. No lesion was identified on MRI of the brain (Fig. 2A, B); however, she was closely monitored with regular biennial imaging. Routine follow-up MRI of the brain at 24 years of age demonstrated a  $1.5 \times 1.2 \times 0.7$  cm lesion in the left choroidal fissure above the left hippocampal body with flow voids concerning for an AVM (Fig. 2C, D). Digital subtraction angiography further elucidated a 1.5 cm left choroidal AVM (0.63 cm<sup>3</sup>) with a supplemented Spetzler-Martin grade of 6 (S1E1V1/A2B1C0) and a Pollock-Flickinger score of 0.94 (score = 0.1 \* volume + 0.02 \* age + 0.5 \* location) (Fig. 2E) [20–22]. Given the location, supplemented Spetzler–Martin grade, and the Pollock-Flickinger score, she underwent 1600 centiGray (relative biological effectiveness) proton radiosurgery in two fractions [23].

#### 3. Discussion

The genesis of AVM is classically viewed as a prenatal event during embryological vasculogenesis [24]. This paradigm is supported by the presence of these lesions in both the pediatric and adult populations [25]. Due to the widespread use of cranial imaging over the past decade, case reports of "spontaneous" rather than "congenital" lesions have arisen (Table 1). At the time of writing, there have been 19 cases reported in the literature [5–19]. These cases include AVM in both adult and pediatric populations and question our fundamental assumptions and understanding of AVM.

With the growing number of cases of AVM reported as de novo or in association with other conditions, it is necessary to reconsider our current nomenclature. We propose to use the terms "primary", "syndromic", and "secondary" to distinguish among these AVM (Fig. 3). Primary AVM occur congenitally as a result of an aberration in cerebral vasculogenesis, which is in contrast to "syndromic" AVM associated with a known and welldescribed genetic syndrome. Secondary AVM are those AVM that occur as an effect of a disparate pathophysiological process. Vascular insults, such as ischemic or traumatic events, underlie the majority of these reported cases and it may be more appropriate to consider these AVM as an abnormal vascular response to injury. Disruptions in physiologic angiogenesis in response to stressors such as trauma, stroke, and hemorrhage may lead to abnormal vessel formation that leads to the development of AVM [12.16.18.26].

In order to improve our understanding of AVM genesis and ultimately develop targeted treatment methods, it is first necessary to understand the pathways involved in the formation of AVM in both primary pathophysiological processes and secondary to vascular insults. Studies of HHT-associated AVM implicate deficiencies in the *ALK1* and *ENG1* pathways, both of which have crucial roles in tumor growth factor (TGF)-beta signaling as predisposing factors [1,27–30]. These findings are supported by genome-wide association studies of patients with AVM that illustrate single nucleotide polymorphism variations in *ALK1*. Perturbations in stromal stability influenced by matrix-metallo protein 9 expression, a possible driver of moyamoya vasculopathy and intracranial aneurysms, have also been posited as underlying mechanisms [31,32].

In addition to vascular insults from stroke, trauma, and hemorrhage, radiation therapy is known to alter the vasculature [33]. In fact, radiation therapy is often used in the treatment of AVM because of its effect on the vascular system [23]. In the present case, our patient initially presented with a near-midline lesion, which received a prescribed dose of 4680 Gy with putative clinical and radiographic evidence of *de novo* AVM formation in the setting of photon radiation therapy. Radiation therapy is known to have several long-term secondary in-field complications, including secondary malignancy, ototoxicity, endocrinologic deficits, and vasculopathy [34,35]. Radiation-induced vasculopathy includes vascular stenosis, strokes, cavernomas, capillary telangiectasias, and, in rare instances, aneurysms [34]. Radiation is known to lead to endothelial cell damage, vessel remodeling, and subsequently vascular endothelial growth factor (VEGF) and TGF-beta expression [33,36-39]. Again, VEGF and TGF-beta (signaled by ALK1 and Endoglin pathways) have been implicated as possible drivers of AVM formation, thereby implicating radiation therapy as a rare but possible factor in AVM formation [1.40]

Alternatively, the very dynamic nature of AVM may implicate an alternative explanation. Known AVM display clinical variability when followed over time. AVM recurrence, regression, and expansion are all clinically reported phenomena [41–43]. Furthermore, when compared to digital subtraction angiography, considered the gold standard, CT scanning and MR angiography have only an approximately 90% sensitivity and specificity for AVM diagnosis [44]. Thus, the possibility of a CT scan or MRI occult AVM exists. However, considering the in-field location, new symptoms, and change in imaging findings over time, a "secondary" AVM remains as the most likely phenomenon in the presented case.



Fig. 2. (A) T2-weighted and (B) T1-weighted axial MRI with contrast 3 years post-radiation demonstrating no evidence of a medial temporal lobe abnormality. (C) T2weighted and (D) T1-weighted axial MRI with contrast 12 years post-radiation displaying a medial temporal lobe abnormality. (E) Digital subtraction angiography, left internal carotid artery injection, illustrating a left medial temporal lobe nidus with a left anterior choroidal artery feeder.

Author	Age at baseline imaging, years	Age at diagnosis	Sex	Reason for prior imaging	Reason for diagnostic imaging	Location of AVM	Initial modality	Diagnostic modality
Schmit et al. 1996 [45]	2	11	М	Moyamoya	Seizure	Left parietal	MRI/ Angiography	MRI/Angiography
Friedman et al. 2000 [5]	61	68	М	dAVF	Follow-up	Right cerebellum	MRI/ Angiography	MRI/Angiography
Bulsara et al. 2002 [6]	26	32	F	Demyelinating disease	ICH	Right posterior temporal	MRI/ Angiography	MRI/Angiography
Akimoto et al. 2003 [7]	10	27	F	IVH, 2 other AVM	IVH	Corpus callosum and cingulate	Angiography	Angiography
Gonzalez et al. 2005 [8]	3	7	F	Trauma	Follow-up	Right posterior temporal	MRI	MRI Angiography
O'Shaughnessy et al. 2005 [9]	3	6	F	Moyamoya	Follow-up	Right Sylvian	MRI	MRI Angiography
Stevens et al. 2009 [11]	6	9	F	Developmental delay	Follow-up	Left-temporal-occipital	MRI	MRI/Angiography
Jeffree et al. 2009 [10]	17 days	5	Μ	Right temporal hemorrhage	Follow-up	Right insular cortex	Angiography	CT/Angiography
	10	15	Μ	Right temporal hemorrhage	Follow-up	Right temporal-parietal	Angiography	Angiography
	8	18	Μ	Left parietal hemorrhage	Hemorrhage	Left parietal-occipital	Angiography	Angiography
Mahajan et al. 2010 [12]	16	30	F	Bell's palsy	Seizure	Left frontal-parietal	MRI	MRI/Angiography
Alvarez et al. 2012 [13]	6	8	Μ	Cerebral cavernous malformation	Follow-up	VOG/straight sinus	MRI/MRA	MRI/Angiography
Mathon et al. 2013 [14]	5	9	Μ	Medulloblastoma	Follow-up	Right peri-sylvian	MRI	MRI/Angiography
Kibourne et al. 2014 [16]	12	19	Μ	Congenital hydrocephalus	Hemorrhage	Pontine	MRI	Angiography
Morales-Valero et al. 2014 [17]	31	35	Μ	TIA	TIA	Let parietal-occipital	MRI	MRI/Angiography
	42	56	Μ	Hepatic encephalopathy	Hemorrhage	Left temporal	Angiography	Angiography
Fujimura et al. 2014 [15]	10	14	F	Moyamoya	Follow-up revascularization	Right occipital	MRI/ Angiography	MRI/Angiography
Yeo et al. 2015 [19]	7	16	F	Seizure	Seizure	Left temporal	MRI	Angiography
	1	7	Μ	Seizure	Seizure	Left cerebellar	MRI	Angiography

 Table 1

 Previous case reports of "secondary" arteriovenous malformations reported in the literature

AVM = arteriovenous malformation, dAVF = dural arteriovenous fistula, F = female, ICH = intracerebral hemorrhage, IVH = intraventricular hemorrhage, M = male, MRA = magnetic resonance angiography, TIA = transient ischemic attack, VOG = vein of Galen.



Fig. 3. Schematic of proposed nomenclature based on the etiology of AVM. AVM = arteriovenous malformation, HHT = hereditary hemorrhagic telangiectasia.

#### 4. Conclusion

AVM are considered congenital lesions, yet a small body of evidence supports the possibility of post-natal AVM formation due to tissue damage or vascular insult. This patient demonstrates an AVM secondary to photon radiation therapy and adds to the growing body of clinical evidence supporting the plausibility of a subset of AVM as secondary rather than primary lesions.

#### **Conflicts of interest/disclosures**

The authors declare that they have no financial or other conflicts of interest in relation to this research and its publication.

#### References

- Leblanc GG, Golanov E, Awad IA, et al. Biology of vascular malformations of the brain. Stroke 2009;40:e694–702.
- [2] Lasjaunias P. A revised concept of the congenital nature of cerebral arteriovenous malformations. Interv Neuroradiol 1997;3:275–81.
- [3] Putman CM, Chaloupka JC, Fulbright RK, et al. Exceptional multiplicity of cerebral arteriovenous malformations associated with hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu syndrome). AJNR Am J Neuroradiol 1996;17:1733–42.
- [4] Bhattacharya JJ, Luo CB, Suh DC, et al. Wyburn-Mason or Bonnet-Dechaume-Blanc as Cerebrofacial Arteriovenous Metameric Syndromes (CAMS). A new concept and a new classification. Interv Neuroradiol 2001;7:5–17.
- [5] Friedman JA, Pollock BE, Nichols DA. Development of a cerebral arteriovenous malformation documented in an adult by serial angiography. Case report. J Neurosurg 2000;93:1058–61.
- [6] Bulsara KR, Alexander MJ, Villavicencio AT, et al. De novo cerebral arteriovenous malformation: case report. Neurosurgery 2002;50:1137–40 [discussion 1140–1].
- [7] Akimoto H, Komatsu K, Kubota Y. Symptomatic de novo arteriovenous malformation appearing 17 years after the resection of two other arteriovenous malformations in childhood: case report. Neurosurgery 2003;52:228–31 [discussion 231–2].
- [8] Gonzalez LF, Bristol RE, Porter RW, et al. De novo presentation of an arteriovenous malformation Case report and review of the literature. J Neurosurg 2005;102:726–9.
- [9] O'Shaughnessy BA, DiPatri Jr AJ, Parkinson RJ, et al. Development of a de novo cerebral arteriovenous malformation in a child with sickle cell disease and moyamoya arteriopathy. Case report. J Neurosurg 2005;102:238–43.
- [10] Jeffree RL, Stoodley MA. Postnatal development of arteriovenous malformations. Pediatr Neurosurg 2009;45:296–304.
- [11] Stevens J, Leach JL, Abruzzo T, et al. De novo cerebral arteriovenous malformation: case report and literature review. AJNR Am J Neuroradiol 2009;30:111–2.
- [12] Mahajan A, Manchandia TC, Gould G, et al. De novo arteriovenous malformations: case report and review of the literature. Neurosurg Rev 2010;33:115–9.

- [13] Alvarez H, Perry V, Solle M, et al. De novo cerebral arteriovenous malformation in a child with previous cavernous malformation and developmental venous anomaly. J Neurosurg Pediatr 2012;9:327–30.
- [14] Mathon B, Blauwblomme T, Bolle S, et al. De novo arteriovenous malformation after brain radiotherapy for medulloblastoma in a child. Neurology 2013;81:398–9.
- [15] Fujimura M, Kimura N, Ezura M, et al. Development of a de novo arteriovenous malformation after bilateral revascularization surgery in a child with moyamoya disease. J Neurosurg Pediatr 2014;13:647–9.
- [16] Kilbourn KJ, Spiegel G, Killory BD, et al. Case report of a de novo brainstem arteriovenous malformation in an 18-year-old male and review of the literature. Neurosurg Rev 2014;37:685–91.
- [17] Morales-Valero SF, Bortolotti C, Sturiale CL, et al. Are parenchymal AVMs congenital lesions? Neurosurg Focus 2014;37:E2.
- [18] Neil JA, Li D, Stiefel MF, et al. Symptomatic de novo arteriovenous malformation in an adult: case report and review of the literature. Surg Neurol Int 2014;5:148.
- [19] Yeo JJ, Low SY, Seow WT, et al. Pediatric de novo cerebral AVM: report of two cases and review of literature. Childs Nerv Syst 2015;31:609–14.
- [20] Pollock BE, Flickinger JC. Modification of the radiosurgery-based arteriovenous malformation grading system. Neurosurgery 2008;63:239–43 [discussion 243].
- [21] Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. J Neurosurg 1986;65:476-83.
- [22] Lawton MT, Kim H, McCulloch CE, et al. A supplementary grading scale for selecting patients with brain arteriovenous malformations for surgery. Neurosurgery 2010;66:702–13 [discussion 713].
- [23] Loeffler J, Agarwalla P. Treatment of an arteriovenous malformation in eloquent areas: proton beam. In: Al-Mefty O, editor. Controversies in neurosurgery II. Germany: Thieme; 2013. p. 310–4.
- [24] Mullan S, Mojtahedi S, Johnson DL, et al. Embryological basis of some aspects of cerebral vascular fistulas and malformations. J Neurosurg 1996;85:1–8.
- [25] Stapf C, Mast H, Sciacca RR, et al. The New York Islands AVM Study: design, study progress, and initial results. Stroke 2003;34:e29–33.
- [26] Yeo JJ, Low SY, Seow WT, et al. Pediatric de novo cerebral AVM: report of two cases and review of literature. Childs Nerv Syst 2015;31:609–14.
- [27] Wrana JL. Regulation of Smad activity. Cell 2000;100:189-92.
- [28] Johnson DW, Berg JN, Baldwin MA, et al. Mutations in the activin receptor-like kinase 1 gene in hereditary haemorrhagic telangiectasia type 2. Nat Genet 1996;13:189–95.
- [29] McAllister KA, Grogg KM, Johnson DW, et al. Endoglin, a TGF-beta binding protein of endothelial cells, is the gene for hereditary haemorrhagic telangiectasia type 1. Nat Genet 1994;8:345–51.
- [30] Chen W, Sun Z, Han Z, et al. De novo cerebrovascular malformation in the adult mouse after endothelial Alk1 deletion and angiogenic stimulation. Stroke 2014;45:900–2.
- [31] Todor DR, Lewis I, Bruno G, et al. Identification of a serum gelatinase associated with the occurrence of cerebral aneurysms as pro-matrix metalloproteinase-2. Stroke 1998;29:1580–3.
- [32] Sturiale CL, Puca A, Sebastiani P, et al. Single nucleotide polymorphisms associated with sporadic brain arteriovenous malformations: where do we stand? Brain 2013;136:665–81.
- [33] van der Kogel AJ. Radiation-induced damage in the central nervous system: an interpretation of target cell responses. Br J Cancer Suppl 1986;7:207–17.
- [34] Murphy ES, Xie H, Merchant TE, et al. Review of cranial radiotherapy-induced vasculopathy. | Neurooncol 2015;122:421–9.
- [35] Kirsch DG, Tarbell NJ. Conformal radiation therapy for childhood CNS tumors. Oncologist 2004;9:442–50.

- [36] Brown WR, Blair RM, Moody DM, et al. Capillary loss precedes the cognitive impairment induced by fractionated whole-brain irradiation: a potential rat model of vascular dementia. J Neurol Sci 2007;257:67–71.
- [37] Reinhold HS, Calvo W, Hopewell JW, et al. Development of blood vessel-related radiation damage in the fimbria of the central nervous system. Inte J Radiat Oncol Biol Phys 1990;18:37–42.
- [38] Rubin P, Gash DM, Hansen JT, et al. Disruption of the blood-brain barrier as the primary effect of CNS irradiation. Radiother Oncol 1994;31:51–60.
- [39] Soares MP, Seldon MP, Gregoire IP, et al. Heme oxygenase-1 modulates the expression of adhesion molecules associated with endothelial cell activation. J Immunol 2004;172:3553–63.
- [40] Sandalcioglu IE, Wende D, Eggert A, et al. Vascular endothelial growth factor plasma levels are significantly elevated in patients with cerebral arteriovenous malformations. Cerebrovasc Dis 2006;21:154–8.

http://dx.doi.org/10.1016/j.jocn.2015.11.021

- [41] Abdulrauf SI, Malik GM, Awad IA. Spontaneous angiographic obliteration of cerebral arteriovenous malformations. Neurosurgery 1999;44:280–7 [discussion 287–8].
- [42] Codd PJ, Mitha AP, Ogilvy CS. A recurrent cerebral arteriovenous malformation in an adult. J Neurosurg 2008;109:486–91.
- [43] Hamada J, Yonekawa Y. Spontaneous disappearance of a cerebral arteriovenous malformation: case report. Neurosurgery 1994;34:171–3.
- [44] Gross BA, Frerichs KU, Du R. Sensitivity of CT angiography, T2-weighted MRI, and magnetic resonance angiography in detecting cerebral arteriovenous malformations and associated aneurysms. J Clin Neurosci 2012;19:1093–5.
- [45] Schmit BP, Burrows PE, Kuban K, et al. Acquired cerebral arteriovenous malformation in a child with moyamoya disease. Case report. J Neurosurg 1996;84:677–80.

## Spinal aneurysmal bone cyst presenting as acute paraparesis during pregnancy



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#### ABSTRACT

Aneurysmal bone cysts (ABC) are benign but locally aggressive osseous lesions characterized by bloodfilled cystic cavities that account for 1–2% of all bone tumors. While pain remains the most common presenting symptom of spinal ABC, extensive anterior column involvement can result in vertebral fractures, spinal instability, and neurological deficits from compression of neural elements. An exceedingly rare case of thoracic ABC presenting as acute paraparesis in a 21-year-old pregnant woman, that was successfully treated with urgent surgical decompression and stabilization, is reported, with discussion of management strategies and review of the pertinent literature.

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#### 1. Introduction

Aneurysmal bone cysts (ABC) are benign but locally aggressive osseous lesions characterized by blood-filled cystic cavities that account for 1–2% of all bone tumors [1,2]. When ABC occur in the spine, they typically involve the posterior elements of the vertebrae, resulting in local bone destruction that often leads to pain. Extensive bony destruction can, however, result in vertebral fractures, spinal instability, and neurological deficits from compression of neural elements [3]. We report an exceedingly rare case of thoracic ABC in a 21-year-old pregnant woman that was successfully treated with urgent surgical decompression and stabilization, with discussion of management strategies and review of pertinent literature.

#### 2. Case report

A 21-year-old woman who was 15 weeks pregnant presented with progressive sudden onset paraparesis along with decreased sensation in the bilateral lower extremities for 3 days and urinary incontinence. Physical examination was significant for paraparesis (1/5 left leg, 3/5 right leg), decreased sensation in the bilateral lower extremities, hyperreflexia and ankle clonus. MRI of the thoracic and lumbar spine revealed a large lytic lesion involving predominantly the T1 and T2 vertebrae with severe spinal cord compression (Fig. 1). The lesion demonstrated intrinsic "fluid-fluid" levels characteristic of ABC. Urgent spinal cord decompression and stabilization were deemed necessary to prevent further neurological deterioration and to optimize clinical outcome. Potential risk of radiation from intraoperative imaging to the fetus was discussed extensively with the patient and her family before the procedure. During positioning, the patient's abdomen and pelvis were wrapped with lead aprons to minimize radiation exposure to the fetus from intraoperative imaging. Following placement of the patient in the prone position and standard posterior exposure, posterior segmental instrumentation was performed at the C5-T5 levels. Given the tumor involvement of the left T1 pedicle and the entire T2 vertebrae, a left T1 pediculectomy and T2 spondylectomy were performed followed by expandable cage placement for anterior column reconstruction (Fig. 2, 3). The patient had dramatic improvement in her bilateral lower extremities strength on postoperative day 1 with near full strength on physical examination. Final histopathology confirmed the diagnosis of ABC. At the 6 week follow-up visit, the patient was doing well with essentially normal neurological examination. Her pregnancy progressed without any complications.

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