Anterior Communicating Artery Aneurysm Following Radiation Therapy for Optic Glioma: Report of a Case and Review of the Literature

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ABSTRACT

A 42-year-old female presented with subarachnoid hemorrhage (SAH), presumably from a radiation-induced anterior communicating artery aneurysm. Six years earlier, she had undergone radiation treatment for an optic glioma that was diagnosed based on imaging criteria. The aneurysm was successfully clipped, and the optic glioma was biopsied to verify the diagnosis histologically. Radiation-induced cerebral aneurysms often manifest with a fatal SAH. These aneurysms typically develop in the field of radiation and are diagnosed a mean of 8.52 years after radiation. Rarely, the aneurysm sac thromboses spontaneously. Clipping or coiling of the aneurysm can be an effective treatment.

KEYWORDS: Intracranial aneurysm, subarachnoid hemorrhage, optic glioma, radiation therapy

The destructive effects of irradiation on blood vessels are well known.^{1,2} Thrombo-occlusive changes frequently occur in cerebral vessels after cranial irradiation.¹⁻³ However, radiation-induced intracranial aneurysms are rare; including our case, only 21 have been reported.⁴⁻¹⁷ In these cases, the aneurysms occurred a mean of 8.52 years

after treatment, and most manifested with a fatal subarachnoid hemorrhage (SAH).¹⁵ We report a patient with a previous history of cranial radiotherapy who developed SAH from an anterior communicating artery (ACoA) aneurysm and was treated successfully with surgical clipping of the aneurysm.

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CASE REPORT

In 1993, an optic glioma was diagnosed, based on imaging without tissue biopsy, in a 42-year-old female after her visual acuity decreased. She was treated with radiation therapy only. Her last magnetic resonance imaging (MRI) study of the brain had been performed in 1997. She was blind in her left eye and had decreased vision in her right eye. In February 1999, the patient briefly lost consciousness and then developed severe headaches, nausea, vomiting, and lethargy. The next day she sought treatment in the emergency room where a head computed tomography showed SAH and an intraparenchymal hematoma with intraventricular hemorrhage. Her initial Glasgow Coma Scale score was 15. The remainder of her cranial nerves was intact, and she could move all extremities symmetrically. Her neck was supple.

Four-vessel angiography (Fig. 1A) showed an ACoA aneurysm. MRI disclosed the residual optic glioma (Fig. 1B) that had been treated 6 years earlier. A right frontal external ventricular drainage catheter was placed to treat hydrocephalus. Two days after admission, the patient underwent surgical

clipping of the aneurysm. A piece of the aneurysm wall and of the optic glioma were sent for pathology. The aneurysm's wall (Fig. 2A) was characterized by poorly cellular, irregular fibrous thickening of the media and intima. There was no residual smooth muscle or internal elastic lamina. These features are compatible with, but do not specifically suggest, radiation-induced vascular injury. Microscopic examination confirmed the presumptive diagnosis of pilocytic astrocytoma. The residual neoplasm showed microvascular fibrosis (Fig. 2B) beyond that which is occasionally found in pilocytic astrocytomas. The pale, hyaline, fibrous mural thickening and loss of endothelial cells are characteristic if not pathognomonic of radiation-induced changes.

The patient recovered fully and was discharged home on the 29th postoperative day.

DISCUSSION

Radiotherapy affects both small and large vessels within the field of treatment. Radiation-induced

Figure 1 (A) Angiogram shows the anterior communicating aneurysm. (B) Sagittal T1-weighted magnetic resonance image shows the residual optic nerve glioma 6 years after radiotherapy.

Figure 2 (A) The vessel wall is irregularly thickened by poorly cellular fibrous tissue. There is no residual smooth muscle or elastic lamina and no transition from "normal" vessel to aneurysm [hematoxylin and eosin (H&E), $\times 400$; *, lumen of the aneurysm]. (B) The pilocytic astrocytoma is typical, composed of well-differentiated bipolar astrocytes with minimal atypia. Microvessels (arrows) show pale hyaline fibrosis. Note the paucity of endothelial cells (H&E, $\times 400$).

vascular changes develop as early as 4 months or as late as 23 years after radiotherapy.¹⁸ In 1959, Thomas and Forbus¹⁹ published the first report of radiation injury to blood vessels.

The effects of radiation on the carotid arteries of rats include injury to the elastic membrane, intimal thickening, fibrosis, and plaque formation. Degeneration of the endothelium, vacuolization, and thickening of the intima associated with changes in the elastic fibers have been described in the radiated arteries of humans. Histopathological changes are predominantly thrombo-occlusive, and vascular occlusion is the most common form of presentation.¹⁻³ For example, carotid artery stenosis, occlusion, or both were detected in 62 symptomatic patients who had undergone previous cervical or cranial irradiation.²

Bole et al²⁰ first reported the formation of an aneurysm after radiation on the cervical segment of the internal carotid artery (ICA). The formation of aneurysms on various other arteries exposed to previous radiation therapy has been suggested.^{8,21-23} Intracranial aneurysm formation after radiotherapy is rare; only 21 patients have been reported (Table 1).^{4-17,24} With the inclusion of our case, these patients include 12 males and 9 females (mean age, 39.85 years; range, 11 to 65). The interval between radiotherapy and the diagnosis of intra-

cranial aneurysm has varied from 7 months to 21 years (mean, 8.52 years). In our patient, the aneurysm was diagnosed 6 years after radiotherapy. In five cases, multiple intracranial aneurysms were detected, most of which were fusiform.^{4,8,11,12,14}

In general, radiation-induced intracranial aneurysms localize to the region of the radiated primary tumor. In one patient with a left parietal arteriovenous malformation, the aneurysm was on the distal right middle cerebral artery.⁶ In another patient with a medulloblastoma, the aneurysm was on the distal right anterior cerebral artery.⁹ In our patient, the location of the primary tumor and aneurysm was similar. However, this finding could also be coincidental because no vascular examination obtained prior to the radiation treatment is available to prove the absence of the aneurysm. Furthermore, the histological changes observed in the aneurysmal wall are not pathognomonic for radiation.

Eight patients died from SAH before surgical intervention could begin. One patient who had a giant saccular ICA aneurysm died from rebleeding 2 weeks after embolization. In seven patients, including ours, the aneurysms were clipped. In two other cases, an ICA ligation was performed with an extracranial-intracranial bypass. One aneurysm was successfully stented; another

Table 1 Review of 21 Reported Cases of Radiation-Induced Cerebral Aneurysms^a

Reference	Sex/Age (years)	Pathology	Aneurysm Location	Latency to SAH (years)
Azzarelli et al (1984) ⁴	F/17	Suprasellar germinoma	Right ICA basilar, right ACA, right vertebral artery	5
Gomori et al (1987) ⁷	M/44	Nasopharyngeal squamous cell CA	Basilar	3
Nishi et al (1987) ¹²	M/57	Pituitary adenoma	Carotid bifurcation ICA (3 fusiform)	9
Benson and Sung (1989) ⁵	M/21	Medulloblastoma	Right PCA	19
	F/31	Medulloblastoma	Right PCA	17
	M/14	Medulloblastoma	Right PCA	9
Scodary et al (1990) ¹³	M/47	Left temporoparietal astrocytoma	Left pericallosal artery	15
Thun and Lanfermann (1991) ¹⁷	M/28	Pituitary adenoma	Right infraclinoid ICA	7
	M/53	Pituitary adenoma	Left supraclinoid ICA	11
Moriyama et al (1992) ¹¹	F/51	Pituitary adenoma	Right MCA saccular, 3 right MCA, and two PCA fusiform	1
Casey et al (1993) ⁶	M/44	Left parietal AVM	Right distal MCA	21
	F/65	Left temporoparietal astrocytoma	Left giant MCA	3.5
John et al (1993) ¹⁰	M/55	Nasopharyngeal CA	Right ICA	5
Holodny et al (1996) ⁸	F/62	Metastasis	Basilar and right ACA and two right MCA	0.6
Jensen and Wagner (1997) ⁹	M/11	Medulloblastoma	Right distal ACA	0.8
Cheng et al (2001) ²⁴	M/54	Nasopharyngeal CA	Right petrous ICA	7
	M/35	Nasopharyngeal CA	Right petrous ICA	2
McConachie and Jacobson (1994) ¹⁴	F/51	Pituitary adenoma	Bilateral cavernous ICA	17
Maruyama et al (2000) ¹⁵	F/15	Optic glioma and SAH	Right ICA bifurcation	15
Pereira et al (2002) ¹⁶	F/19	Craniopharyngioma	ICA bifurcation	5
Present report	F/42	Optic glioma	ACoA	6

^aICA, internal carotid artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; MCA, middle cerebral artery; AVM, arteriovenous malformation; CA, carcinoma; SAH, subarachnoid hemorrhage; and ACoA, anterior communicating artery.

lesion was embolized with detachable coils. A single aneurysm thrombosed spontaneously without intervention.

patients with SAH who have undergone prior cranial radiotherapy. Successful outcomes have been described for clipping or endovascular management of such aneurysms.

CONCLUSION

In summary, radiation-induced aneurysms appear to form relatively late. Because of the diffuse changes in the cerebral vessels, fatal hemorrhage is common. Angiography should be performed in symptomatic

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