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Dissecting aneurysm of intracranial vertebral artery: case report and review of literature

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Summary. A spontaneous dissecting aneurysm of the intracranial portion of the dominant right vertebral artery presented as massive subarachnoid hemorrhage, excruciating headache, and respiratory arrest in a 57-year-old white man with a history of systemic hypertension. He died on the 3rd day. Postmortem examination revealed a dissecting hemorrhage extending for 2.1 cm along the artery; rupture of the intima, media, and adventitia could be demonstrated. The intramural accumulation of blood in the proximal segments appeared to be related to retrograde dissection within a media weakened by cystic degeneration. Accumulation of pools of mucoid ground substance was also demonstrated in other intracranial and extracranial arteries. Hemodynamic stresses due to arterial hypertension and physical exertion may have played a contributory role in the etiopathogenesis of this uncommon form of cerebrovascular accident. A comprehensive literature review permits a comparison of supratentorial and infratentorial dissecting aneurysms; vertebral and basilar artery dissections are presented in tabular form.

Key words: Dissecting aneurysm – Vertebral artery dissection – Cystic medial degeneration – Subarachnoid hemorrhage – Intramural hematoma

Zusammenfassung. Ein spontan dissezierendes Aneurysma des intrakraniellen Abschnittes der dominanten rechten A. vertebralis mit massiver Subarachniodalblutung bei einem 57jährigen Mann mit anamnestisch bekannter Hypertonie führte zu starken Kopfschmerzen, Atemstillstand und schließlich am 3. Tag zum Tode. Die Autopsie ergab eine Blutung aus einer 2,1 cm langen Dissekation mit einem Riß durch Intima, Media und Adventitia. Die Blutansammlung im proximalen Abschnitt spricht für eine retrograde Dissekation innerhalb der durch eine zystische Degeneration geschwächten Media. Eine Häufung poolförmiger mucoider Grundsubstanz wurde auch in anderen intra- und extrakraniellen Arterien nachgewiesen. Hämodynamischer Streß infolge Hypertonie und körperliche Belastung konnten eine mitbestimmende Rolle in

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der Ätiopathogenese der ungewöhnlichen Form eines cerebralen Insultes spielen. Ein umfassender Literaturüberblick gestattet eine Vergleich supra- und subtentorieller dissezierender Aneurysmen; diese werden tabellarisch einmal im Bereich der Vertebralarterien, zum anderen im Bereich der A. basilaris dargestellt.

Introduction

Dissecting aneurysms of intracranial arteries are being encountered with an accelerating tempo in neurologic and neurosurgical practice, by neuroradiologic studies, and in surgical and autopsy material. Histopathologically documented intracranial arterial dissections now number 67 in the European, North American, and Japanese literature. An analysis by dates discloses that eight were reported during the first five decades of this century; during the next two decades 30 cases were reported, and since 1970, there have been 29 cases. Case reports with literature surveys still predominate, but occasional larger series have been published [1, 3, 4, 12, 20], including cases with only a presumptive and/or neuroradiologic diagnosis. Several cases have been republished [3 and 25; 4 and 10; 12 and 21; 12 and 17; 28 and 35]; attempting to assemble a reasonably comprehensive review of all published cases is thus fraught with the potential problem of double-counting.

The purpose in presenting an additional case and in reviewing the literature is to compare the clinicopathologic features between vertebral and basilar artery dissections and, to a lesser extent, the relative frequency and age incidence of supratentorial versus infratentorial aneurysms.

Case report

Clinical summary

A 57-year-old white man with a known history of moderate hypertension (treated with diuretics for 6 years) and type IV hyperlipidemia developed a sudden, severe headache behind the right eye and pain in the neck while playing tennis; there had been no trauma nor did he lose consciousness, but he did complain of blurred vision. Two days later he complained of an excruciating headache, which was followed by gasping respiration and loss of consciousness. On admission to the Emergency Department, the patient had regained consciousness and was fully oriented, but reported numbness of the right leg and weakness of the left arm. Respiratory arrest was treated promptly with endotracheal intubation. Thereupon he regained consciousness slowly and moved all limbs equally, and even removed the endotracheal tube. He developed another severe nuchal headache, with respiratory arrest and pupillary dilatation and nonreactivity. There was no response to either the doll's eye maneuver or to ice-water caloric stimulation. On reintubation he did not regain spontaneous breathing but his lungs became congested and blood pressure rose to 230/170 mm Hg. Treatment consisted of intravenous Lasix, mannitol, dexamethasone, and Apresoline, whereupon blood pressure fell to 160/100 mm Hg. A right parietal twist-drill ventriculostomy initially released clear CSF, but the fluid quickly became bloody and clotted. The left extremities were flaccid and unresponsive to pain, pupils were nonreactive, the neck was moderately rigid, and there were subhyaloid hemorrhages in the left fundus; the temperature was 38.3° C. Hypertension was treated with Apresoline. The pulse and respiratory rates and blood pressure fell to unobtainable levels and he was pronounced dead on the 3rd day after onset of symptoms.

Clotting parameters were within normal limits. The clinical impression was subarachnoid hemorrhage due to a ruptured berry aneurysm, though angiography could not be performed because of the poor clinical state of the patient.

Postmortem examination

Left cardiac ventricular hypertrophy and benign arteriolar nephrosclerosis were indicative of long-standing systemic hypertension, and a moderate degree of atherosclerosis affected most of the larger systemic and cerebral arteries. The viscera were markedly congested and the lungs edematous.

The edematous brain weighed 1650 g and particularly the basal subarachnoid spaces were filled with fresh and clotted blood. Both cerebellar tonsils showed pressure grooves. The vertebrobasilar arteries and circle of Willis demonstrated focal, moderate atherosclerosis; the right vertebral artery was larger than the left. No berry aneurysm was identified, but an oblique, 0.3 cm long adventitial tear in the anterior wall and in the distal portion of the right vertebral artery was evident, 0.7 cm from its termination. Two bulging hematomas in the proximal intradural and middle segments of the artery were also seen in its anterior wall. Coronal sections of the cerebral hemispheres revealed the ventricular system to be filled with blood, a massive disruption of the fimbriae and hippocampal commissure providing the portal of entry.

Microscopically, the cerebral cortex, basal ganglia, diencephalon, cerebellum, and brainstem demonstrated multifocal, bland, ischemic necrosis as seen in nonperfused brains. The subpial layers of the medulla oblongata (especially the anterior surface of the pyramids), pons, and midbrain and the ependymal lining were irregularly disrupted by extension of hemorrhages from the subarachnoid space and ventricles.

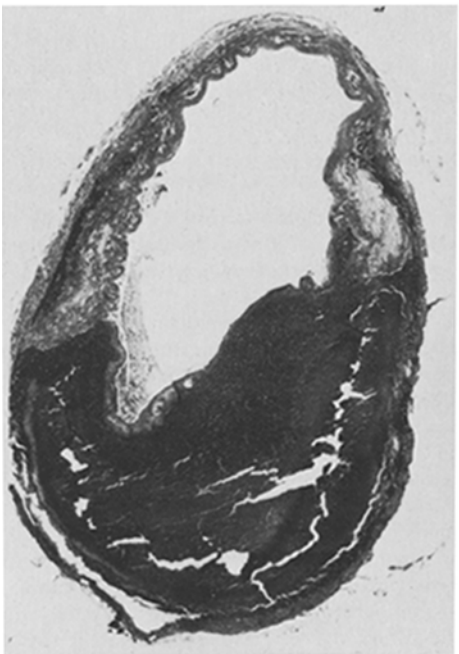
The 2.4-cm-long intracranial segment of the right vertebral artery was sequentially cut into eight blocks, each measuring 3 mm in length; semiserial sections were obtained and stained with hematoxylin and eosin, Masson's trichrome, PTAH, elastica-van Gieson, Hale's colloidal iron, PAS, and Gram. The left vertebral artery and right internal carotid/middle cerebral artery were similarly stained.

The proximal four segments of the right vertebral artery demonstrated an intact intima and elastic lamina, but its media exhibited a sharp disruption in continuity for up to one half of the circumference; the crescentic gap was filled with fresh blood (Fig. 1). At a level 1.3 cm from the dural penetration by the artery, an irregular intimal tear was apparent (Fig. 2), with thrombus filling the dissection and part of the lumen. In the more distal segments, the adventitia became progressively more attenuated and more heavily deposited with fibrin and infiltrated by polymorphonuclear leukocytes (Fig. 3). Medial smooth muscle cells were commonly separated by irregular pools of mucoid ground substance, particularly near the edges of medial disruption, and delicate elastic fibrils were frayed and short. Only rare acute inflammatory cells occupied the tunica media. Microorganisms could not be identified with the special stains. The left vertebral, internal carotid, middle cerebral, aorta, and common iliac arteries also exhibited multifocal cystic medial degeneration.

Discussion

Of the 67 histopathologically documented cases of intracranial dissecting aneurysms reviewed in this paper, 40 (60%) involved the internal carotid system, while 27 (40%) affected the vertebrobasilar arteries (including extensions into the posterior cerebral arteries).

The distribution of this relatively rare vasculopathy between basically supratentorial and infratentorial lesions thus differs substantially from that of the more prevalent vascular catastrophes; viz., vertebrobasilar aneurysms represent 3.6 to 9.6% of all intracranial berry aneurysms [9, 24]; subtentorial arteriovenous malformations represent less than 8% [19], and hypertensive hemorrhages approximately 20% [2], of the total number of such cases. The cases grouped as



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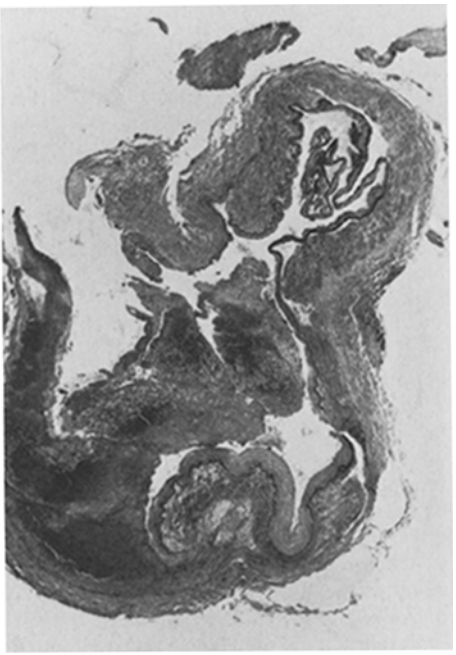

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Fig. 1. Proximal segment of right vertebral artery showing intact intima but a dissecting hematoma in the medial plane. The ruptured media has retracted and is clumped at the edges of the hematoma (PTAH; × 25)

Fig. 2. Distal segment of the artery at the site of external rupture illustrates the dissection, early thrombus in the lumen and portion of separated intima and internal elastic lamina as a coil within the lumen *(right upper field)* (Elastica-van Gieson; × 25)

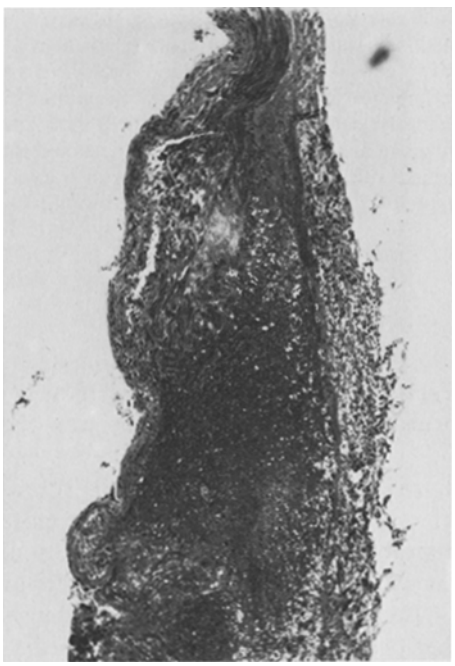

Fig. 3. View of the arterial wall depicts intact tunics in uppermost field; retracted media and pools of mucoid ground substance are evident in the next quarter; intramural dissecting hematoma and adventitial acute inflammation are seen in the lower three-quarters of the field; the snapped and slightly curled intima and internal elastic membrane are seen in the left lower corner (PTAH; × 63)

vertebral (Table 1) and basilar (Table 2) artery dissections demonstrate a decided difference in mean age. The cases of Hassin [14, 15] are not considered relevant, because slight disruption and separation of the internal elastic lamina of several large intracranial arteries was an agonal event of legal or accidental electrocution. The 11 cases with vertebral artery dissection ranged in age from 30 to 57 years, with a mean of 39.8 years, whereas the 14 with basilar artery dissection tended to affect a younger age group, ranging from 15 to 69 years, with a mean of 31.7 years. The age difference becomes further accentuated, if one subdivides those five dissections confined to the vertebral artery (mean age of 45.2 years) from those five dissections of the vertebral artery extending into the basilar artery (mean age of 36.1 years). Thus, there appears to be a trend for basilar dissections to occur in a relatively younger age group than for vertebral artery dissections. Dissection of arteries in the supratentorial compartment affected a still younger age group, with a mean age of 17.5 years.

Analysis of the data also points to a preferential affliction of males, with a prevalence of 78% among those with basilar artery dissection, 60% in those with dissecting aneurysms of the vertebral artery, and 58% in the carotid arterial system. Among dissections affecting the vertebral arteries, there is a ratio of 8 : 3 in favor of the right side. Whether this reflects any relationship to the discrepancy in size between the two vessels in a similar proportion of the general population is a moot point. In the carotid system, the right side was affected in 20 cases, the left in 15, and both sides in 2 patients. In 8 of the 14 with dissections of the basilar artery, the posterior cerebral arteries (and occasionally other smaller branches) were also involved.

There was no difference in the severity of symptoms and prognosis between vertebral and basilar artery dissections. Both presented in various combinations and sequences of excruciating headache, vomiting, vertigo, seizures, hemiplegia or quadriplegia, coma, and cranial nerve dysfunctions, usually with sudden and a dramatic onset. In both categories, survival was less than 2 weeks in approximately 50%, death generally resulting from extensive cerebral infarction, massive edema, and elevated intracranial pressure. However, in two of the patients in whom the vertebral artery was affected, surgery was attempted; unfortunately, they died 3 and 4 days postoperatively [8, 38]. Nevertheless, for two other patients in whom no histopathologic confirmation is available, operative inspection and clipping of the vertebral artery affected by intramural dissection has resulted in survival with no or only insignificant deficit [30, 34]. Less commonly, but exemplified in this case report, massive subarachnoid hemorrhage caused death.

The etiology in both locations includes a spectrum of predisposing or associated factors, including syphilis, trauma, cystic medial degeneration, atherosclerosis, and hypertension. Fibromuscular dysplasia was present in one case of vertebral artery dissection; a congenital defect of the internal elastic lamina was deemed a relevant, contributory factor in four cases in which the basilar artery was dissected. Several patients had complained for years of migraine or recurrent headache while others were known to have suffered from hypertension. For several cases in each group, no cause could be assigned.

The acute inflammatory cell infiltrate in the adventitia in the case reported is interpreted as the tissue response to the medial disruption and dissection, similar to

Table 1. Histopathologically documented dissections of intracranial vertebral artery

Case no.	Sex/ age (years)	Signs and symptoms	Duration of illness	Angiography	Extent and extension of dissection	Histopathology	Etiology	Remarks	Reference
1	M, 47	Dysphagia, dyspnea, ataxia, lower cranial nerve paresis	11 days	—	R BA	—	—	—	[29]
2	F, 35	Headache, ↑ ICP	40 days	—	R, 18 mm	—	Syphilis	Trephine; post-op. meningitis	[32]
3	M, 45	Vertigo, neck pain, dysphagia	6 weeks to 5 years	—	L	Between int. elastica and media	Atherosclerosis	Suspected brainstem metastasis; RVA hypoplastic	[8]
4	—	—	—	—	R, 8 mm	—	—	Subarachnoid hemorrhage	[6]
5	F, 30	Headache, emesis, decerebration, coma	7 days	—	L BA	Between int. elastica and media	? Atherosclerosis	Ventricular drainage; brainstem infarct	[28]
6	M, 32	Headache, emesis, dysarthria, quadriplegia, coma	6 days	N carotid, with opacification of basilar and cerebellar arteries; RVA severely narrowed	R BA	Medial dissection	? Trauma, ? congenital	Positive serology; brainstem	[11]
7	M, 32	Left hemiparesis; severe nuchal headache; resp. arrest	56 days	BA occlusion, RVA narrowed	R BA R PCA	Outer medial dissection	Subacute arteritis	↑ BP; violent neck rotation	[22]
8	F, 44	Headache, photophobia	29 days	RVA aneurysm, segmental narrowing	R	Between media and adventitia; acute and chronic inflammation	—	VA ligated, died of abdominal hemorrhage	[38]

9	F, 43	Sudden coma, L hemiplegia	14 days	N carotid; occluded LVA	L BA, 40 mm	Between int. elastica and media	Intimal fibrodysplasia	Car accident 2 months earlier; ↑ BP due to chr. glomerunephritis	[23]
10	M, 33	Headache, emesis, hemiplegia, coma	6 days	N carotid; bil. VA fusiform aneurysms	R BA	Between media and adventitia	?Atherosclerosis, ?congenital	Hypertension, SAH	[33]
11	M, 57	Headache, resp. arrest × 3, coma	3 days	—	R, 21 mm	Rupture of media; focal acute inflammation	Cystic medial degen.	↑ BP; twist drill ventriculostomy; massive SAH	This report

— no information or not performed

Table 2. Histopathologically documented dissections of the basilar artery

Case no.	Sex/ age (years)	Signs and symptoms	Duration of illness	Angiography	Extent and extension of dissection	Histopathology	Etiology	Remarks	Reference
1	M, 24	Heat stroke, coma	6 days	—	PCA	—	Syphilis	—	[31]
2	M, 42	Hemiplegia	21 days	—	—	Medial dissection	Cystic medial degeneration	Positive luetic serology; brainstem infarct	[18]
3	M, 32	Headache, confusion semicoma	4 days	—	—	Between intima and media	Cystic medial degeneration	Brainstem infarct	[36]
4	F, 33	Miosis, coma	7 days	—	Bil. PCA, sup. cerebellar Aa.	Between intima and media	Congenital	Brainstem infarct	[37]
5	M, 30	Headache, emesis, seizures, coma	Hours	—	Bil. PCA, and sup. cerebellar Aa.	Between intima and media	Atherosclerosis	15 years of headaches	[7]
6	F, 22	Headache, tetraplegia, coma	7 weeks	N carotid	Bil. PCA	Between intima and media	Congenital	Post-partum catastrophe; diencephalic infarct	[26]
7	M, 21	Headache; emesis, tetraplegia, coma	14 days	N carotid	—	Between intima and media	Congenital	Brainstem infarct	[25]
8	M, 15	Semicoma	8 weeks	N carotid	—	Medial dissection	—	Slight mental retardation; brainstem infarct	[16]
9	M, 29	Oculomotor palsy, coma, tetraplegia	5 months	BA stenosis; later total occlusion	Bil. PCA	Between intima and media	? Congenital, ? homocystinuria	Thalamic and mesencephalic infarcts	[5]

10	M, 22	Headache, emesis, tetraplegia, coma	2 months	N carotid, bil.; BA occlusion	L PCA	Between intima and media	—	Lightening of coma	[4]
11	M, 40	Coma, miosis, tachypnea	3 days	—	—	Subintimal	Hemodynamic	23 years headache, hypertension	[20]
12	F, 35	Seizures	2 days	N carotid	—	Between intima and media	Cystic medial degeneration	Antiovulants; migraine	[27]
13	M, 69	Dementia, paraparesis, deafness	2 years	Irregular BA	(Bil. VA)	Medial dissection	Atherosclerosis	Died 3 days postop. aneurysmal mass effect and hydrocephalus	[1]
14	M, 30	Migraine, scotoma, semicoma	21 days	N carotid, filling of PCA	1.5 cm; sup. cerebellar Aa.	Medial dissection	—	15 years migraine	[1]

— no information given

the changes described by others [11, 38]. The relationship of the type IV hyperlipidemia to the cystic medial degeneration is unknowm. What role physical exertion and hypertension played in the intramural dissection and rupture of the arterial wall is also a moot point.

Antemortem diagnosis depends on a high index of suspicion and appropriate neuroradiologic investigations. Diagnostic angiographic features have been the "string sign", "rosette sign", "pearl reaction", and luminal narrowing [11, 12, 17, 38]. Giedke et al. [13] maintain that the only angiographically diagnostic sign is the demonstration of a "double lumen". Unfortunately, the rapid clinical deterioration of the patient precluded performance of this study.

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