Massive Hemorrhage in Acoustic Neurinoma After Minor Head Trauma

—Case Report—

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Abstract

Massive hemorrhage within an intracranial neurinoma occurs rarely. The authors describe a 62-year-old female with intratumoral bleeding which led to the discovery of an acoustic neurinoma. She developed a gait disturbance after a minor head injury. A computed tomographic scan obtained 2 months later demonstrated multiple high-density areas in the anterior portion of the left cerebello-pontine angle. Preoperative diagnosis was acoustic neurinoma. The tumor had multiple cysts which contained a mixture of xanthochromic fluid and old, brownish hematomas, and was successfully removed. The intratumoral hemorrhage is thought to have resulted from traumatic rupture of the dilated vessels, although the trauma was slight. This is the first reported case of an acoustic neurinoma discovered through treatment for intratumoral hemorrhage occurring after a minor head injury.

Key words: acoustic neurinoma, intratumoral hemorrhage, cerebellopontine angle tumor, head trauma

Introduction

Spontaneous intratumoral hemorrhage occasionally occurs in malignant gliomas and pituitary adenomas. However, massive intratumoral hemorrhage (ITH) is extremely rare in intracranial neurinomas. Here, we describe a case of massive hemorrhage occurring after a minor head injury, and discuss the pathogenesis of ITH with a review of the literature.

Case Report

A 62-year-old female fell and struck the occipital region while descending a flight of stairs on June 4, 1988. There was no loss of consciousness and no medical treatment was sought. However, she developed a gait disturbance immediately after the accident although she could walk unassisted. She was admitted on August 1, 1988, for evaluation of the gait disturbance. She had been in good health except for 1-year history of a hearing impairment.

On admission, she was alert and had no headache or vertigo. The ocular fundi and pupillary responses

were normal. She exhibited gaze-evoked horizontal nystagmus (Bruns' nystagmus) without a rotatory component, as well as slight left facial paresis. The taste perception was normal. Air conduction exceeded bone conduction bilaterally. The hearing loss was moderate, which reached 100 dB on the left. Brainstem auditory evoked response testing showed the prolongation of wave I-V latencies (over 5.9 seconds). The other cranial nerves were intact. The tandem gait was slightly ataxic, and coordination was poor on the left. The motor and sensory responses were unremarkable. Complete laboratory blood analysis, including a hemogram, routine chemical tests, and coagulation studies, disclosed no abnormalities.

Plain skull x-rays and a skull tomogram showed enlargement of the left internal auditory meatus. A precontrast computed tomographic (CT) scan demonstrated multiple fluid-fluid levels in a low-density mass at the left cerebellopontine angle (Fig. 1 *left*). A postcontrast CT scan showed a ring-like enhancement which was approximately 3 cm in diameter, with mural nodules at the left auditory meatus (Fig. 1 *right*). There was no bleeding outside of the tumor. These findings suggested an ITH of the left cerebel-

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Fig. 1 *left*: Precontrast CT scan showing multiple fluid-fluid levels in the low-density area at the left cerebellopontine angle. *right*: Post-contrast CT scan demonstrating ring-like enhancement and mural nodules at the auditory meatus.

lopontine angle lesion. Four-vessel angiography demonstrated no vascular anomalies. A left vertebral angiogram revealed a downward shift of the choroidal point of the left inferior cerebellar artery indicating a lesion in the left cerebellopontine angle (Fig. 2).

On August 11, 1988, she underwent a suboccipital craniectomy, and a well capsulated tumor was exposed. The tumor had multiple cysts which consisted of xanthochromic and dark brown elements resembling old hematoma. Incision of the capsule revealed the fibrous, yellowish tumor wall. The tumor contents were evacuated. The cochlear nerve

Fig. 2 Left vertebral angiogram showing the downward shift of the choroidal point of the left inferior cerebellar artery. No aneurysm or arteriovenous malformation is present.

tapered off into the tumor capsule, but the facial nerve was preserved. The remnant of the tumor in the auditory meatus was removed after drilling of the pyramidal bone around the meatus.

Histological examination showed a neurinoma composed predominantly of cells with uniform, rod-shaped nuclei (Antoni A-type) (Fig. 3A). In some areas, the tumor contained loosely packed Antoni B-type cells (Fig. 3B). There were several hypervascular regions in which numerous clusters of thin-walled, dilated vessels were seen (Fig. 3C). In some portions, hemosiderin-laden macrophages indicative of a prior hemorrhage were observed, as well as the areas of fresh hemorrhage (Fig. 3D).

Postoperative course was uneventful and she resumed normal life 2 weeks after surgery. The left cerebellar signs had disappeared, although only slight left facial weakness and hearing loss persisted.

Discussion

Hemorrhage within intracranial neurinomas accounts for approximately 8% of all brain ITH.¹⁵⁾ However, massive hemorrhage within intracranial neurinomas is rare.¹⁸⁾ Massive ITH or subarachnoid hemorrhage (SAH) has been reported in only 18 cases of acoustic neurinoma^{1-9,11-14,16,19-21)} and two of trigeminal neurinoma^{10,17)} (Table 1). In all reported cases including ours, the neurinomas were large (mean diameter > 3 cm). Although a large tumor appears to be an important risk factor for major bleeding, hemorrhage is also associated with intrinsic tumor vascularity and extrinsic vessel erosion, compression, and aneurysm formation.⁶⁾ In 14 of the 21 reported cases, histological examinations found dilated vessels, hypervascularity, or hematoma. Extrinsic vessel erosion was not the cause of hemorrhage in any case.

Occasionally, neurinomas display staining on cerebral angiograms. However, no tumor staining was observed in the reported cases. A contact aneurysm was identified in one case.⁹⁾ CT scans were obtained in 18 cases. Fifteen acoustic neurinomas and one trigeminal neurinoma appeared as high-density areas. Only one case each of acoustic and trigeminal neurinomas had normal findings. Fluid-fluid levels were observed in only two cases (Case 16 and ours). The type of bleeding was SAH in three cases, ITH in eight, and both SAH and ITH in ten.

Two patients were treated conservatively, 18 underwent elective surgery, and one had emergency surgery. One surgically treated patient (Case 5) and two conservatively treated patients (Cases 1 and

Fig. 3 Photomicrographs of tumor specimens. A: Typical Antoni A-type features are evident. B: Typical Antoni B-type features. C: Numerous thin-walled, dilated vessels are seen. D: Hemosiderin-laden macrophages surround the dilated vessels and the fresh hemorrhage are shown. HE stain, $\times 100$ (A,C,D) and $\times 200$ (B).

4) died (14%). The remaining 17 except for Case 9 had good outcome, with only cranial nerve deficits generally, and were able to return to normal life.

Hypervascularity on histological study is a common feature of neurinomas. Goetting and Swanson⁶ reported that approximately 60% of tumors larger than 2 cm exhibited markedly increased vascularity, in sharp contrast to the 20–25% in smaller tumors.⁶ The vessels are often dilated, ectatic, and thin-walled, frequently being lined by a single layer of endothelium.¹⁴ We previously experienced eight acoustic neurinomas, and histological examination demonstrated four containing hemosiderin-laden macrophages and four with numerous dilated vessels.

Although microscopic bleeding within acoustic neurinomas is a frequent phenomenon, we have encountered only one case of massive hemorrhage into an acoustic neurinoma. Gleeson *et al.*⁵ postulated a sequential mechanism causing massive ITH of malignant brain tumors: vascular endothelial proliferation with subsequent obstruction of the vessel lumen; distal vessel necrosis; vessel distention, dis-

tortion, and displacement as a result of tumor growth; and vessel erosion by the tumor. However, the mechanism causing massive bleeding in benign tumors is not yet well understood.

Shimabukuro *et al.*¹⁷ described an ITH case of a trigeminal neurinoma that might have been caused by a minor head injury. Goetting and Swanson⁶ suggested that an abrupt, large increase in arteriovenous pressure creates a transient gradient between the vascular and extravascular intracranial compartments which can lead to the rupture of fragile tumor vessels. In our case, since the signs and symptoms developed shortly after a minor head trauma, it is possible that the trauma participated in the induction of the hemorrhage. The numerous dilated vessels and microcysts may have been distorted by the force of the impact, causing their thin walls to rupture.

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Table 1 Summary of reported cases of massive hemorrhage within neurinomas

Case No.	Author (Year)	Age, Sex	Initial symptoms	Location (cranial nerve)	Size (cm)	CT finding	Type of bleeding		Treatment	Vascularity	Outcome	Remarks
							ITH	SAH				
1	McCoy <i>et al.</i> (1974) ¹³⁾	64, F	unconsciousness, lt hemiparesis	VIII	2 × 3 × 3.5	NP	+	+	conservative	dilated vessels	died	
2	Fine <i>et al.</i> (1977) ³⁾	21, F	hearing loss, headache	VIII	3 × 4	NP	+	+	elective surgery	none	good	bloody CSF
3	Gleeson <i>et al.</i> (1978) ⁵⁾	54, F	headache, hearing loss	VIII	4	normal	—	+	elective surgery	none	good	
4	Kasantikul and Netsky (1979) ⁸⁾	64, M	NR	VIII	NR	NP	+	+	conservative	cavernous hemangioma	died	
5	Baba <i>et al.</i> (1980) ¹⁾	71, F	gait disturbance, headache, lt facial palsy, mental deficits	VIII	NR	ITH	+	—	elective surgery	dilated vessels	died	hydrocephalus, VP shunt
6	Kubota <i>et al.</i> (1981) ¹⁰⁾	43, M	trigeminal neuralgia, headache	V	3	normal	+	+	elective surgery	dilated vessels	good	
7	Shephard and Cheeks (1981) ¹⁶⁾	37, M	unconsciousness	VIII	large	SAH	—	+	elective surgery	NR	good	
8		66, M	hearing loss, ataxia	VIII	3	SAH	+	+	elective surgery	NR	good	
9	Castillo <i>et al.</i> (1982) ²⁾	61, M	hearing loss, headache	VIII	3	ITH	+	+	elective surgery	dilated vessels	NR	
10	Shimabukuro <i>et al.</i> (1983) ¹⁷⁾	33, M	double vision, headache	V	5.8 × 3.5	ITH	+	—	elective surgery	dilated vessels	good	head injury
11	Yonemitsu <i>et al.</i> (1983) ²¹⁾	49, M	hearing loss, headache	VIII	5 × 5 × 4	ITH	+	+	elective surgery	dilated vessels	good	
12	Ihara <i>et al.</i> (1984) ⁷⁾	54, M	hearing loss, headache	VIII	NR	ITH	+	+	emergency surgery	dilated vessels	good	
13	Mironov <i>et al.</i> (1986) ¹⁴⁾	61, M	headache	VIII	2	SAH	+	+	elective surgery	dilated vessels	good	
14	Goetting and Swanson (1987) ⁶⁾	19, M	facial palsy, headache	VIII	3.2	ITH	—	+	elective surgery	dilated vessels	good	head injury
15	Kodama <i>et al.</i> (1987) ⁹⁾	66, F	hearing loss, headache	VIII	3	SAH	+	+	elective surgery	contact aneurysm	good	
16	Tsuchiya <i>et al.</i> (1988) ¹⁹⁾	37, F	hearing loss, facial palsy	VIII	3	ITH	+	—	elective surgery	NR	good	
17	Kurata <i>et al.</i> (1989) ¹¹⁾	56, M	hearing loss, facial palsy	VIII	3 × 4 × 5	ITH	+	—	elective surgery	dilated vessels	good	
18	Yamamoto <i>et al.</i> (1989) ²⁰⁾	60, M	headache, hearing loss	VIII	3	ITH	+	—	elective surgery	dilated vessels	good	
19	Lee and Wang (1989) ¹²⁾	65, F	headache, hearing loss	VIII	4 × 4 × 4	ITH	+	—	elective surgery	dilated vessels	good	
20	Fuse <i>et al.</i> (1989) ⁴⁾	39, M	headache, hearing loss	VIII	3	ITH	+	—	elective surgery	dilated vessels	good	
21	Present case	62, F	hearing loss, ataxia	VIII	3	ITH	+	—	elective surgery	dilated vessels	good	head injury

ITH: intratumoral hemorrhage, SAH: subarachnoid hemorrhage, CSF: cerebrospinal fluid, VP: ventriculoperitoneal, NP: not performed, NR: not reported.

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