Chronic Encapsulated Intracerebral Hematoma Associated with Angiographically Occult Arteriovenous Malformation

—Case Report—

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Abstract

A 34-year-old female presented with a rare chronic encapsulated intracerebral hematoma associated with an angiographically occult arteriovenous malformation. A neovascularized fibrous capsule containing various stages of intracerebral hematoma formation was removed. These unusual entities mimic brain tumors or abscesses because of gradual growth and slowly progressive neurological deficits. Repeated bleeding or exudation from the capillaries of the capsule may allow expansion of the chronic encapsulated intracerebral hematoma.

Key words: chronic encapsulated intracerebral hematoma, arteriovenous malformation, fibrous capsule, ring enhancement

Introduction

Chronic encapsulated intracerebral hematomas may demonstrate fibrous capsules at surgery.^{1-7,9,11-16,18-20,23)} However, such cases associated with arteriovenous malformation are very rare.^{5,16)} We report a 34-year-old female with chronic encapsulated intracerebral hematoma associated with an angiographically occult arteriovenous malformation, and discuss the etiology and mechanisms of encapsulation.

Case Report

A 34-year-old female had suffered from occasional migraine for 10 years, and had developed increasing severity of headache with nausea and vomiting since June, 1990. She was referred to our hospital on July 20, 1990, with a presumptive diagnosis of brain tumor based on computed tomography (CT) performed at a local hospital. She denied any history of

head injury, epilepsy, hypertension, or coagulopathy.

Physical and neurological examination on admission revealed no abnormalities. All laboratory examinations were within normal ranges. Precontrast CT scans demonstrated a well-demarcated isodense mass in the left temporal lobe surrounded by a hyperdense zone. Postcontrast CT scans showed distinct ring enhancement, but no enhancement of the mass (Fig. 1). Magnetic resonance (MR) imaging detected heterogeneous signals in the round mass. The T₁-weighted image showed central iso- and peripheral high-intensity signals with thin rim enhancement using gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA). The T₂-weighted image showed the lesion as a marked high-intensity area separated from the surrounding edema by a low-intensity rim (Fig. 2). Cerebral angiograms revealed only mass effect.

On August 6, 1990, a left frontotemporal

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Fig. 1 *left*: Precontrast CT scan, showing a round isodense mass surrounded by a thin hyperdense zone in the left temporal lobe. *right*: Postcontrast CT scan, showing a non-enhanced isodense mass with distinct ring enhancement.

craniotomy was performed under a diagnosis of chronic intracerebral hematoma. A well-defined, tough capsule was found about 15 mm from the temporal cortex. Incision of the capsule revealed the liquid and solid components of the hematoma. The fibrous capsule was about 2 mm thick. A small red vein was noted in the medial pericapsular region, and a tiny vascular malformation was removed with a small amount of adjacent brain tissue.

Histological examination of the surgical samples revealed that the capsule consisted of abundant collagenous tissue on the outside and fibroblastic granulation tissue with rich neovascularity on the inside. Hemosiderin-laden macrophages were also present in the granulation tissue layer. The vascular

Fig. 3 *left*: Photomicrograph of the capsule, consisting of abundant collagenous tissue on the outside and fibroblastic granulation tissue, hemosiderin-laden macrophages, and rich neovascularity on the inside. *right*: Photomicrograph of the surgical specimen of the arteriovenous malformation. HE stain, $\times 30$.

anomaly was confirmed as an arteriovenous malformation (Fig. 3).

The postoperative course was satisfactory and she was discharged without neurological deficit on August 24, 1990.

Discussion

Hirsh *et al.*⁵⁾ first reported chronic encapsulated intracerebral hematoma in 1981, but two previous descriptions of this condition were reported.^{6,23)}

Fig. 2 *left*: T_1 -weighted MR image, showing a mixed high-intensity mass with a low-intensity boundary of the medial surface. *center*: T_1 -weighted MR image with Gd-DTPA, showing rim enhancement with no enhancement of the mass. *right*: T_2 -weighted MR image, showing the markedly high-intensity content of the mass, emphasizing the peripheral low-intensity rim and perifocal edema.

Table 1 Summary of 32 cases of chronic encapsulated intracerebral hematomas

Author (Year)	Age	Sex	Side	Location	Signs and symptoms	Duration	Etiology	Outcome*
Zimmerman <i>et al.</i> (1977) ²³⁾	25	M	—	frontal	seizure	—	head trauma	—
Kawakami <i>et al.</i> (1978) ⁶⁾	31	F	middle	cerebellum	headache, dizziness, cerebellar signs	8 mos	unknown	GR
Hirsh <i>et al.</i> (1981) ⁵⁾	64	M	rt	parieto-occipital	lt arm weakness, lt homonymous hemianopsia	2 mos	AVM	MD
Takahashi <i>et al.</i> (1983) ¹⁹⁾	72	M	rt	parieto-occipital	lt hemiparesis	—	AVM	MD
	55	F	rt	parietotemporal	seizure	17 yrs	unknown	MD
Aoki and Mizuguchi (1984) ¹⁾	7 mos	M	middle	cerebellum	disturbance in holding head steady	—	unknown	SD
Lin <i>et al.</i> (1984) ⁹⁾	56	M	lt	frontoparietal	rt hemiparesis, aphasia	8 mos	head trauma	MD
	44	F	rt	parietotemporal	seizure, lt agnosia of body	12 mos	unknown	GR
	21	M	lt	frontotemporal	headache, blurred vision, lt abducens palsy	2 mos	unknown	GR
Terada <i>et al.</i> (1985) ²⁰⁾	1	F	lt	temporal	seizure, vomiting, rt facial palsy	2 wks	unknown	GR
Masuzawa <i>et al.</i> (1985) ¹²⁾	43	M	rt	parietal	seizure, lt hemiparesis	—	cavernoma	MD
	8	M	rt	cerebellum	headache, vomiting, loss of consciousness	3 mos	unknown	GR
Aoki and Mizuguchi (1986) ²⁾	24	M	rt	cerebellum	headache, cerebellar signs	1 mo	unknown	GR
Pozzati <i>et al.</i> (1986) ¹⁶⁾	66	F	—	parieto-occipital	headache, papilledema	—	unknown	died
	11	M	rt	frontoparietal	seizure	—	unknown	GR
	36	M	rt	temporal	seizure	—	unknown	GR
	31	F	—	frontal	seizure	—	unknown	GR
	62	F	lt	frontal	frontal lobe syndrome, papilledema	—	unknown	GR
	65	M	—	frontal	headache	—	AVM	GR
	44	M	—	parieto-occipital	seizure, lt hemiparesis	—	unknown	GR
	57	M	—	frontoparietal	lt hemiparesis	—	unknown	died
Steiger <i>et al.</i> (1987) ¹⁸⁾	45	M	—	frontal	hemiparesis, papilledema	—	cavernoma	—
Chan and Tse (1987) ³⁾	20	M	lt	parietotemporal	headache, diplopia	3 mos	UCVM	GR
Matsumoto <i>et al.</i> (1988) ¹³⁾	60	F	lt	occipital	headache, rt homonymous hemianopsia	2 mos	metastatic adenocarcinoma	GR
Fiumara <i>et al.</i> (1989) ⁴⁾	33	M	lt	parietal	seizure, dysphasia, dyslexia, dysgraphia	4 mos	unknown	MD
	64	M	rt	frontal	frontal lobe syndrome	3 mos	unknown	MD
Monma <i>et al.</i> (1990) ¹⁴⁾	29	F	rt	parietal	seizure	21 yrs	cavernoma	MD
	32	M	rt	frontal	headache	1 wk	cavernoma	GR
Kumabe <i>et al.</i> (1990) ⁷⁾	29	M	rt	basal ganglia	headache, lt lower quadrantoanopsia	2 mos	venous angioma	GR
Murakami <i>et al.</i> (1990) ¹⁵⁾	14	M	rt	frontal	headache, nausea, vomiting	2 wks	cavernoma	GR
Mabuchi <i>et al.</i> (1991) ¹¹⁾	43	F	lt	parietal	headache, nausea, rt hemiparesis	19 mos	cavernoma	GR
Present case	34	F	lt	temporal	headache, nausea, vomiting	10 yrs	AVM	GR

*Glasgow Outcome Scale; GR: good recovery, MD: moderate disability, SD: severe disability. AVM: arteriovenous malformation, UCVM: unclassified vascular malformation.

Chronic encapsulated hematomas occur extracranially and in many other locations of the body.¹⁷⁾

Table 1 lists the 31 reported cases of chronic encapsulated intracerebral hematomas with surgically

proven fibrous capsules.^{1-7,9,11-16,18-20,23)} Two cases strongly resembling this entity were excluded because no surgical confirmation of the fibrous capsules was reported.^{21,22)} The neurological signs and symptoms

are not specific as with space-occupying lesions, but slow progression or increased frequency occurred in most patients.

The etiology of the hemorrhage was detected in 15 cases: 12 were caused by vascular malformations including six cavernomas,^{11,12,14,15,18} four arteriovenous malformations,^{5,16} one venous malformation,⁷ and one unclassified vascular malformation.³ The other etiologies were two head injuries^{9,23} and a metastatic adenocarcinoma.¹³ Our case is unique in that the vascular malformation was a true arteriovenous malformation histologically. Previous possible cases^{5,16} did not provide sufficiently detailed information, and many cases are unknown. The incidence of this lesion associated with arteriovenous malformation cannot be estimated and it is unclear why encapsulated hematoma in this setting is rare.

Chronic encapsulated intracerebral hematomas do not develop from hypertensive intracerebral hemorrhages. Postcontrast CT demonstrates resolving intracerebral hematomas with a ring of enhancement. Laster *et al.*⁸ reported that early ring formation was related to blood-brain-barrier breakdown and later enhancement to vascular granulation tissue based on histological examination. The final stage was the healed phase of hematoma resorption, during which no ring enhancement occurred. Therefore, another cause of the capsule formation should be considered.

The tough, fibrous capsule of chronic encapsulated intracerebral hematoma is similar to the outer membrane of chronic subdural hematoma,² with abundant fibrous connective tissue with rich neovascularity. Hirsh *et al.*⁵ suggested that the capsule originated from fibroblasts related to the abnormal vessels of an occult vascular malformation. Lobato *et al.*¹⁰ found that over half of 262 angiographically occult intracranial vascular malformations had caused macroscopically visible intraparenchymal hematomas (either recent or old) and/or intraventricular or subarachnoid hemorrhage. Most cases had repeated ictal episodes for variable periods before the causative lesion was identified and removed. Therefore, repeated bleeding may encourage capsule formation with the rich neovascularity caused by the fibroblasts from the vascular malformation. A common hypothesis is that repeated bleeding or exudation from the capillaries of the capsule allows expansion of the chronic encapsulated intracerebral hematoma.

The 31 reviewed cases all had fibrous capsules, and most hematomas were totally removed. The postoperative courses of most patients were satisfactory. Two deaths were reported but details are unclear.¹⁶ The necessity for total removal of the

hematoma with the capsule is still uncertain. Therefore, further investigation of the optimum treatment is necessary.

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