

# **Serial Neuroimaging of Encapsulated Chronic Intracerebral Hematoma With Repeated Hemorrhage**

## **—Case Report—**

Wataro TSURUTA, Koji TSUBOI, and Tadao NOSE

*Department of Neurological Surgery, Institute of Clinical Medicine,  
University of Tsukuba, Tsukuba, Ibaraki*

### **Abstract**

A 69-year-old woman presented with headache and short memory disturbance. Computed tomography (CT) demonstrated a small cystic mass lesion in the left temporal lobe. CT and magnetic resonance imaging showed that this lesion enlarged with repeated hemorrhages, associated with progressive amnesia and headache during 3 years follow up. Surgery demonstrated a well-demarcated hard mass lesion in the medial temporal lobe through a transcortical approach after opening left sylvian fissure. The lesion was located entirely in the brain parenchyma and was removed en-bloc after cutting some capillary-like vessels on the capsule. The histological diagnosis was encapsulated old hematoma. The histological findings suggested that expansion of the lesion was due to multiple bleedings from the sinusoidal vessels in the capsule fed by small feeding arteries. The mass effect due to the expansion of the encapsulated hematoma caused progressive short-term amnesia and headache, which were completely resolved by the surgical removal.

Key words: encapsulated hematoma, intracerebral hematoma

### **Introduction**

Encapsulated chronic intracerebral hematoma (ECIH) is a very rare entity first reported in 1978<sup>2)</sup> and 1981.<sup>5)</sup> The precise etiology of this condition remains to be clarified. We recently encountered a case of ECIH, which we were able to follow for over 3 years before surgical removal. Here we describe the serial changes on neuroimaging and the histological observations of the removed specimen.

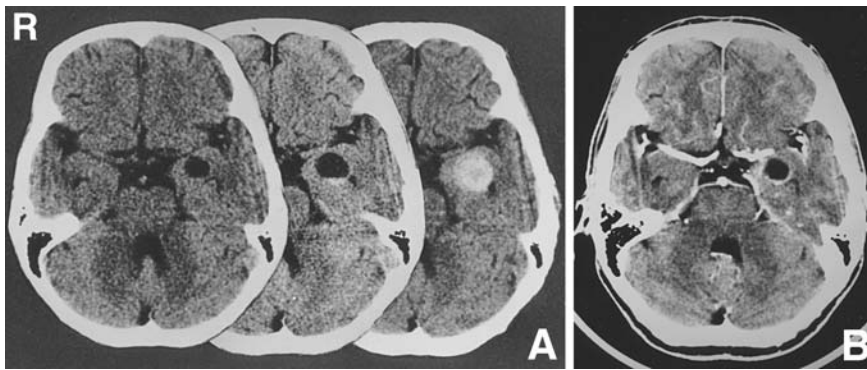
### **Case Report**

A 69-year-old woman presented with complaints of headache. Computed tomography (CT) detected a low-density cystic lesion with thin ring-like enhancement in the left temporal lobe in 1995 (Fig. 1A left, B). Neurological examination demonstrated no abnormality except for intermittent headache, which lasted approximately 30 minutes. Follow-up CT and magnetic resonance (MR) imaging revealed a small amount of hemorrhage in the cyst in 1996

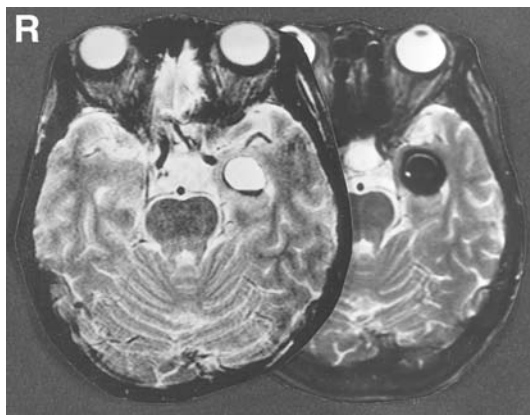
(Figs. 1A center and 2 left). Cerebral angiography demonstrated no vascular abnormality (data not shown). Her headache became severe and short memory disturbance appeared in 1998. CT and MR imaging revealed expansion of the lesion by a fresh clot (Figs. 1A right and 2 right), prompting the decision to perform surgical removal. The preoperative differential diagnosis included benign cystic tumor and cavernous hemangioma with cystic formation.

The left sylvian fissure was opened following frontotemporal craniotomy. The lesion was entirely situated in the brain parenchyma. The well-demarcated hard mass lesion was found and removed en-bloc through a medial temporal transcortical approach. No obvious abnormal vessels were observed in the vicinity of the lesion except for several capillary-like vessels in contact with the capsule. The contents of the capsule were old clots with a muddy appearance (Fig. 3A).

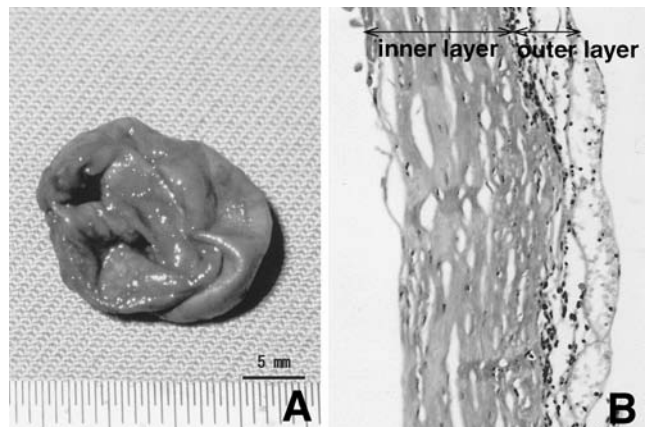
Histological examination revealed that this capsule consisted of outer and inner layers. Lymphocytic infiltration and sinusoidal vessels were observed in the outer layer, whereas the inner layer contained degenerative sinusoidal vessels and hemosiderin-laden macrophages in proliferating fibroblasts



**Fig. 1** A: Computed tomography scans at initial examination (left) revealing a low-density cystic mass lesion in the left temporal lobe, one year later (center) showing the cystic lesion had enlarged and contained a fluid level due to a small hemorrhage, and 2 years later (right) demonstrating expansion of the cyst by fresh hemorrhage. B: Computed tomography scan with contrast medium at initial examination revealing thin ring-like enhancement of the lesion.



**Fig. 2** T<sub>2</sub>-weighted magnetic resonance images taken one year after initial admission (left) demonstrating an old hematoma containing a small amount of fresh hemorrhage in the left temporal lobe, and 2 years later (right) showing the lesion is filled with low-intensity fresh clot.



**Fig. 3** A: Photograph showing the hematoma encapsulated by hard connective tissue, which contained old clots with a muddied appearance. B: Photomicrograph of the capsule demonstrating that the capsule consists of outer and inner layers. Lymphocytic infiltration and sinusoidal vessels are present in the outer layer, whereas the inner layer contains degenerative sinusoidal vessels and hemosiderin-laden macrophages in proliferating fibroblasts. HE stain,  $\times 200$ .

(Fig. 3B).

Her postoperative course was uneventful, and her headaches and memory disturbance were completely resolved. There has been no recurrence after 4 years of follow up.

## Discussion

Small spontaneous intracerebral hematomas are usually totally absorbed, but on rare occasions the hematoma may become encapsulated and expand over a long period with repeated internal bleeding, as seen in this case. CT and MR imaging clearly demonstrated the repeated bleeding and expansion

of the lesion during 3 years. Thirty cases of ECIH with histological evidence have been reported (Table 1).<sup>1,3-8,11-17,19</sup> The mean age was 41.8 years old and the most frequent symptoms were headache and seizure. The male to female ratio is two to one, and the most frequent locations were the frontal, temporal, and parietal lobes. Most reported cases of ECIH were followed up for less than several months before surgery, and only two cases including ours demonstrated expansion of the lesion with

**Table 1 Clinical characteristics of the patients**

Case No.	Author (Year)	Age/ Sex	Symptoms	Location	Cause	Outcome
1	Hirsh et al. (1981) <sup>5)</sup>	64/M	PND	occipital	vascular malformation	MD
2		72/M	PND	occipital	vascular malformation	GR
3	Takahashi et al. (1983) <sup>17)</sup>	55/F	seizures, headache	temporoparietal	?	GR
4	Aoki and Mizuguchi (1984) <sup>1)</sup>	0.6/M	no head control	cerebellum	?	GR
5	Lin et al. (1984) <sup>6)</sup>	56/M	trauma	frontoparietal	?	SD
6		44/F	seizures, headache	temporoparietal	?	GR
7		21/M	headache	intraventricular	?	GR
8	Terada et al. (1985) <sup>19)</sup>	1/F	seizures, increased ICP	temporal	?	GR
9	Masuzawa et al. (1985) <sup>7)</sup>	43/M	seizures, PND	parietal	cavernoma	MD
10		8/M	headache	cerebellum	vascular malformation	GR
11	Pozzati et al. (1986) <sup>12)</sup>	66/F	increased ICP	parieto-occipital	?	D
12		11/M	seizures	frontoparietal	vascular malformation	GR
13		36/M	seizures	temporal	?	GR
14		31/F	seizures	frontal	?	GR
15		62/F	frontal syndrome	frontal	?	GR
16		65/M	headache	frontal	vascular malformation	GR
17		44/M	seizures, PND	parieto-occipital	?	GR
18		57/M	PND	frontoparietal	?	D
19	Chan and Tse (1987) <sup>3)</sup>	20/M	headache	temporoparietal	vascular malformation	GR
20	Steiger et al. (1987) <sup>15)</sup>	21/M	seizures	parietal	cavernoma	?
21		45/M	PND, increased ICP	frontoparietal	cavernoma	?
22	Fiumara et al. (1989) <sup>4)</sup>	33/M	seizures, PND	parietal	?	MD
23		64/M	PND	occipital	?	MD
24	Monma et al. (1990) <sup>8)</sup>	29/F	seizures	parietal	cavernoma	MD
25		32/M	increased ICP	frontal	cavernoma	GR
26	Roda et al. (1993) <sup>13)</sup>	21/F	headache	frontal	cavernoma	GR
27	Okuno et al. (1993) <sup>11)</sup>	71/F	headache	frontal	cavernoma	?
28	Suyama et al. (1996) <sup>16)</sup>	55/M	headache, sensory disturbance	temporal	?	GR
29	Sakamoto et al. (1997) <sup>14)</sup>	57/M	seizures, headache	frontal	AVM	GR
30	Present case	69/F	headache, memory disturbance	temporal	?	GR

AVM: arteriovenous malformation, D: died, GR: good recovery, ICP: intracranial pressure, MD: moderately disabled, PND: progressive neurological deficit, SD: severely disabled.

evidence of repeated hemorrhage on CT or MR imaging.<sup>14)</sup>

Histological examinations demonstrated sinusoidal vessels in the capsule in most cases, and the two layer pattern in the capsule in nine cases.<sup>1,3,7,11,12,14,16)</sup> Among these nine cases, sinusoidal vessels were located in the outer layer in five cases including ours,<sup>3,11,14,16)</sup> and in the inner layer in the other four cases.<sup>1,7,12)</sup> Thus, the clinicopathological characteristics of genuine ECIH may be summarized as the presence of a distinct fibrous capsule that may consist of two layers, sinusoidal vessels in the capsule, and expansion of the hematoma with repeated internal bleeding from the capsule.

In our case, initial CT demonstrated a small low-density cystic lesion, which indicated that encapsulation of the hematoma was already completed. At least two hemorrhagic events occurred during the 3 years of follow up. The cause of the initial hemorrhage in our case was not clear. Irregular fibroblastic proliferation has been observed in a thalamic hematoma secondary to a vascular malformation,<sup>10)</sup>

and occult intracerebral vascular malformation may be responsible for spontaneous intracerebral hematoma with a chronic clinical course.<sup>5)</sup> We speculate that the mechanism of the formation of ECIH occurs as follows. First, the initial small hematoma was encapsulated by fibrin membrane, followed by the proliferation of reactive fibroblasts that may have originated from an occult vascular lesion. Then, sinusoidal vessels fed by small arteries developed on the outside of this thin capsule. Leakage of blood, especially the plasma fraction, from this sinusoidal layer through the inner rough fibrous tissue resulted in repeated hemorrhage in the cavity with further stimulation of fibroblast proliferation and enlargement of the lesion. In addition, the formation of membrane might be induced by growth factors.<sup>9)</sup>

This mechanism is similar to that of the outer membrane of chronic subdural hematoma (CSDH). The outer membrane of CSDH consists of granulation tissue formed of fibroblasts and capillaries,<sup>2)</sup> with many capillary-like vessels in the outer layer of the capsule, and a few feeding arterioles exist

between the dura mater and the capsule.<sup>18)</sup> These observations are very similar to ours in ECIH except that CSDH is formed between the dura mater and pia mater whereas ECIH is located entirely within the brain parenchyma. The most important factor in the development of ECIH, as with CSDH,<sup>20)</sup> is the sinusoidal vessels of the capsule, which are fragile. Contact between the hematoma and pia mater may be correlated with capsule formation, based on the observation that superficial white matter lesions are the most frequent.<sup>4)</sup> However, there was no contact between the capsule and the pia mater in our case.

ECIH is a rarely reported clinical entity; but cases may remain unreported, or followed up as possible low-grade tumors or unknown cystic lesions because of the failure to obtain diagnostic tissue. ECIH can be surgically removed with excellent outcome. Histological observation of the capsule suggests that expansion of the hematoma is caused by repeated hemorrhage from sinusoidal vessels in the capsule fed by small arteries.

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Address reprint requests to: K. Tsuboi, M.D., D.M.Sc., Department of Neurological Surgery, Institute of Clinical Medicine, University of Tsukuba, 1-1-1 Tennohdai, Tsukuba, Ibaraki 305-8575, Japan.  
e-mail: tsuboi@md.tsukuba.ac.jp