A Chronic Intracerebral Fluid Hematoma

MOTOHIRO NOMURA¹, KATSUYOSHI MIYASHITA¹, AKIRA TAMASE¹, TOMOYA KAMIDE¹, KENTARO MORI¹, YOSHIHISA KITAMURA¹, SHUNSUKE SEKI¹, HIROSHI SHIMA¹, KUNIO YANAGIMOTO²

¹Department of Neurosurgery, ²Department of Pathology, Yokohama Sakae Kyosai Hospital; Yokohama, Japan

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SUMMARY – Intracerebral hematoma usually resolves and a chronic fluid hematoma is rare. We describe a rare case of intracerebral fluid hematoma. This report describes a case of intracerebral fluid hematoma mimicking a brain tumor and discusses the characteristics of this condition. A 70-year-old woman had a six-month history of memory disturbance. Computed tomography scan showed a low-density lesion with a partial high-density area in the right frontal lobe. MRI revealed a lesion of the main cystic portion showing high intensity on both T1 and T2 weighted images with a low-intensity solid portion in the anteromedial side. The lesion was adjacent to the lateral ventricle. Craniotomy was carried out and the lesion was removed. Pathological examination of the solid portion revealed that the diagnosis was reactive changes due to intracerebral hemorrhage. In our case, there was a possibility that the hematoma was diluted with cerebrospinal fluid, and coagulation might have been prevented.

Introduction

Intracerebral hematoma usually resolves spontaneously¹. However, it occasionally develops a mass after a period of time¹ that may cause neurological deficits in a patient and be misdiagnosed as a brain tumor¹. Although several reports describing chronic encapsulated intracerebral hematoma (CEIH) have been published²,³,⁶,⁷, a fluid hematoma without encapsulation is rare. We describe a case of chronic fluid hematoma without encapsulation and discuss the characteristics of this rare condition.

Case Report

A 70-year-old woman had a six-month history of memory disturbances. About a month previously, her symptoms had significantly worsened and she showed abnormal behavior. She initially consulted a psychiatrist. Computed tomography (CT) scan showed a low-density lesion with a slightly high-density portion in the right frontal lobe. She was referred to our department for further examinations. She had no history of head injury or bleeding. Laboratory examinations revealed no abnormality in blood cell count and coagulation. No drug such as anti-platelet or anti-coagulation agent was administered. Repeated CT showed the same findings as the initial scan (Figure 1A). Contrast-enhanced (CE)-CT showed partial enhancement in the anteromedial portion of the lesion (Figure 1B).

MRI demonstrated a cystic lesion in the right frontal lobe. The cyst content showed high intensity on both T1 and T2 weighted images (Figure 2). The lesion was adjacent to the lateral ventricle on MRI. Cerebral angiogram showed no vascular staining of the lesion. MRI obtained four years previously showed no vascular anomalies such as cavernous angioma or arteriovenous malformation.

From the radiological findings, the lesion seemed to be a tumor and its removal was planned. Right frontal craniotomy was performed. At the operation, the surface of the brain was yellowish, implying post-hemorrhagic changes. A dilated sulcus was opened and dark red old coagulation (Figure 3A) and fluid hematoma (Figure 3B) were found. The coagulated hematoma was removed and dark red fluid hematoma was aspirated.
Figure 1  A) CT showing a low-density lesion with a partial high-density portion in the right frontal lobe. B) CE-CT showing partial enhancement in the surface portion.

Figure 2  MRI revealing a cystic lesion with a solid portion in the anteromedial side. The cyst content showed high intensity on both T1 (A) and T2 (B) weighted images.
The lesion, thought to be the solid part of the tumor, was removed and subjected to pathological examination. Pathological examination of the samples revealed reactive change with accumulation of macrophages and astrocytes (Figure 3C). Cytology of the fluid showed multiple degenerated red blood cells at a concentration of about 500 cells/μl. Chemical analysis of the fluid was as follows: protein 6 g/dl, and sugar 60 mg/dl.

Her postoperative course was uneventful. She was discharged 13 days after the operation. Follow-up CT and MRI showed no recurrence of the lesion for 46 months.

**Discussion**

Spontaneous intracerebral hematoma is usually totally absorbed, but on rare occasions, the hematoma may become encapsulated and expand with repeated internal bleeding. Chronic encapsulated intracerebral hematoma (CEIH) was first reported in 1981. CEIH is reported to be associated with vascular malformations such as cavernous angioma or arteriovenous malformation. However, CEIH with no relation to vascular anomaly was also reported. The condition showed slowly progressing symptoms, suggesting a brain tumor. CEIH is
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characterized by the presence of a fibrotic capsule, which histologically resembles the outer capsule of a chronic subdural hematoma and is thought to develop by chronic progressive growth due to repeated bleeding from the new blood vessels in the capsule.

Fujii et al. described that it required approximately one month for encapsulation of the hematoma. At first, the hematoma was divided into a fluid and a solid portion. The fluid portion enlarged by fluid escape from the intravascular fluid and edema fluid from the disrupted blood-brain barrier. A capsule was formed by a continuing inflammatory response like chronic subdural hematoma. In our case, on radiological and intraoperative findings, the lesion had a deeply located fluid portion and a surface solid portion. These findings indicated that the lesion contained clots at various stages of development or absorption. However, the lesion did not have a capsule.

With regard to the histological findings, our case indicated unorganized hematoma with hemosiderin deposition, and accumulation of macrophages and astrocytes in the periphery. The findings indicated that the lesion involved reactive changes secondary to hemorrhage.

Concerning the etiology of hematoma, the wedge-shaped lesion might be a hemorrhagic transformation of cerebral infarction, although the pathological examination showed no infarcted brain tissue. Previous MRI showed no vascular anomalies, and the location of lesion was the territory of the fronto-orbital artery. Further, the patient had no acute symptoms such as consciousness disturbance or severe headache on admission.

Although the lesion of our case was a chronic hematoma, it was different from CEIH. In our case, no direct connection between the lesion and the ventricle was observed during surgery. However, the lesion was adjacent to the lateral ventricle on MRI. Therefore, cerebrospinal fluid might flow into the hematoma resulting in dilution of the hematoma. This condition might prevent the hematoma from complete coagulation.

Conclusion
We reported a case of an intracerebral fluid hematoma mimicking a brain tumor. Histologically, the lesion was revealed to be reactive changes after hemorrhage. There was a possibility that the lesion ruptured to the lateral ventricle and was diluted with cerebrospinal fluid. In cases with radiological features of a cystic lesion close to a ventricle, fluid hematoma should be taken into consideration as a differential diagnosis.

References


