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Case Report

Chronic encapsulated intracerebral hematoma: Three case reports and a literature review

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Abstract

Background: Chronic encapsulated intracerebral hematoma (CEIH) is one type of intracerebral hematoma that sometimes grows progressively while forming a capsule and presenting with neurological deficits. Although many cases of CEIH have been reported, correct preoperative diagnosis is very difficult. Only around 20% of cases are diagnosed preoperatively.

Case Description: We encountered three cases of CEIH in which causes were unidentified and difficult to diagnose. All three cases were treated surgically. In the first case, a 59-year-old male was diagnosed preoperatively with metastatic brain tumor. In the second case, a 62-year-old female was diagnosed preoperatively with glioblastoma. The third case involved a 58-year-old female diagnosed preoperatively with CEIH.

Conclusion: We should keep in mind that CEIH is a differential diagnosis for intracerebral space-occupying lesions. This report describes these three cases and discusses imaging findings and characteristics of CEIH.

Key words: Chronic encapsulated intracerebral hematoma, diagnosis, neuroimaging

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INTRODUCTION

Chronic encapsulated intracerebral hematoma (CEIH) is a rare entity that was first reported in 1981. This type of hematoma expands slowly and behaves as a space-occupying lesion. Surgical resection is effective to improve the neurological symptoms and good prognosis is expected, but the lesion can be difficult to diagnose. We describe three cases of CEIH and review the associated literature, with special emphasis on the clinical and neuroimaging features relevant to diagnosis.

CASE REPORT

Case 1

A 59-year-old male presented with a 1-month history of progressive weakness in the left upper extremity. Neurological examination revealed monoparesis of the left upper limb, and muscle power assessment showed grade 3/5. Plain computed tomography (CT) showed a faint hyperdense mass in the right frontal lobe [Figure 1a]. Fluid-attenuated inversion recovery (FLAIR) imaging revealed the lesion as a low-intensity mass

accompanied by extensive perifocal edema [Figure 1b]. Gadolinium-enhanced T1-weighted magnetic resonance imaging (MRI) showed faint enhancement [Figure 1c]. The preoperative differential diagnosis included metastatic brain tumor. Left frontal craniotomy was performed, and the mass lesion was revealed to be entirely located within the brain parenchyma. The hard mass lesion with yellowish membrane was removed en bloc through a transcortical approach [Figure 2]. The mass lesion contained old clots and muddy hematoma, and histological examination confirmed the diagnosis of CEIH [Figure 3]. Postoperatively, motor paresis promptly improved. MRI showed the disappearance of CEIH and cerebral edema, and no recurrence [Figure 1d and e]. The patient currently has no neurological deficits and is back at work.

Case 2

A 62-year-old female presented with a 2-month history of speech disorder. Neurological examination on admission revealed motor aphasia. Plain CT showed a faint hyperdense mass lesion in the left frontal lobe [Figure 4a]. T2-weighted MRI revealed the lesion as an isointense region with high-intensity rims and peripheral edema [Figure 4b], and gadolinium-enhanced T1-weighted MRI showed ring enhancement [Figure 4c]. Preoperative differential diagnoses included glioblastoma, metastatic brain tumor, and brain abscess. Left frontal craniotomy was performed, and the mass lesion was reached through the cortex. The mass had a thick capsule and clear border with the surrounding brain tissue, and contained old clots and muddy hematoma. No abnormal vessels were observed in the vicinity of the lesion. Histological examination revealed the capsule contained macrophages with hemosiderin, and granulation tissue. No evidence of vascular malformation or brain tumor was present. The lesion was finally diagnosed as CEIH. The postoperative course was uneventful, and motor aphasia resolved completely. MRI showed disappearance of the mass lesion and cerebral edema, and there has been no recurrence after 2 years of follow-up [Figure 4d].

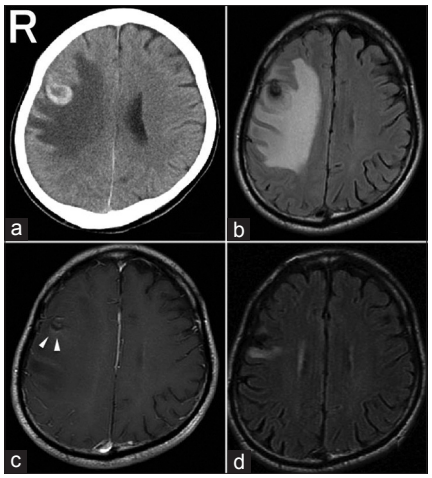

Figure 1: Case 1 Plain computed tomography (CT) shows a hyperdense mass lesion in the left frontal lobe (a). Fluid-attenuated inversion recovery (FLAIR) reveals the lesion as a mixture of hypo- and hyperintensity mass with extensive brain edema (b). Gadolinium-enhanced T1-weighted magnetic resonance imaging (MRI) shows mild enhancements (arrow heads) (c). Follow-up FLAIR imaging performed 1 year after surgery shows improvement of perifocal edema and no recurrence of chronic encapsulated intracerebral hematoma (CEIH) (d)

Case 3

A 58-year-old female presented with a 3-week history of progressive confusion, disorientation, apathy, and left hemiparesis (muscle power assessment showed grade 4/5). CT demonstrated a faint hyperdense mass in the right frontal lobe [Figure 5a]. FLAIR revealed the lesion as mixed-intense and hyperintense mass with

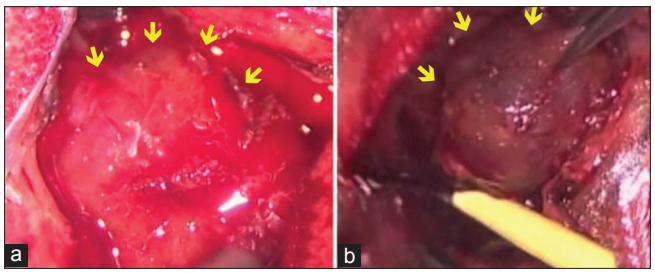

Figure 2: Intraoperative view Left frontal craniotomy was performed and a well-defined mass lesion was found located entirely within the brain parenchyma (arrows) (a). The hard mass lesion with yellowish membrane was removed en bloc (arrows) (b)

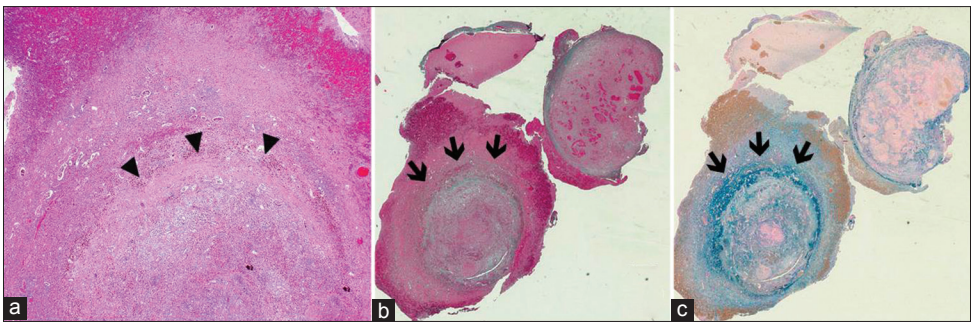

Figure 3: Photomicrographs of surgical specimen Hematoxylin and eosin stain shows hemosiderin (arrowheads) and granulation tissues (a). Masson trichrome stein shows collagen fibers within the capsule wall (black arrows) (b). Berlin blue stain shows hemosiderin deposition in the capsule wall (black arrows) (c)

perifocal edema [Figure 5b]. T2*-weighted gradient echo imaging revealed the lesion as a high-intensity mass with low-intensity rims suggestive of hemosiderin deposition [Figure 5c]. Gadolinium-enhanced T1-weighted MRI showed ring enhancement [Figure 5d]. FLAIR revealed the lesion as mixed-intense and hyperintense mass, with the lateral part of the lesion becoming hyperintense 2 weeks after initial MRI [Figure 5e]. Preoperative differential diagnoses included CEIH. Right frontal craniotomy was performed, and a thin yellowish capsule was identified. After separation from the surrounding structures, the mass was completely removed. Contents of the capsule comprised clots in various stages of hematoma formation. Histological examination showed no abnormal vessel malformation and confirmed the diagnosis of CEIH. The postoperative course was uneventful and symptoms gradually improved. Follow-up MRI showed no recurrence of the lesion at the 1-year follow-up [Figure 5f, g].

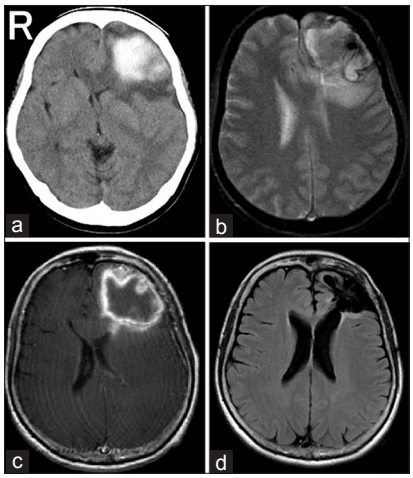

Figure 4: Case 2 Plain CT shows a hyperdense mass lesion in the left frontal lobe (a). T2-weighted MRI reveals the lesion as an isointense mass with low-intensity rims (b) and gadolinium-enhanced T1-weighted MRI shows ring enhancement (c). Follow-up FLAIR imaging and CT performed 1 year after surgery reveal disappearance of perifocal edema and no recurrence of CEIH (d)

DISCUSSION

CEIH is a special type of intracerebral hematoma, first described by Hirsh in 1981[12] and characterized by the presence of a fibrotic capsule that histologically resembles the outer capsule of chronic subdural hematoma. CEIH sometimes grows progressively while forming the capsule and in such cases may present with neurological deficits. The mechanism of growth is thought to involve repeated bleeding from the new blood vessels in the capsule. To date, 54 other cases have been reported in the literature.[1-44] Table 1 depicts background characteristics and details of the patients. CEIH is a disease affecting all ages; the youngest reported patient was 2 months old and the oldest was 80 years old. The mean age is 44 years. Men may be affected more often than women, with 37 male patients and 20 female patients reported. Compared with intracerebral hematoma, which shows a sudden onset, CEIH often begins with progressive neurological deficits due to mass effects. However, one-third of patients present with sudden onset in the form of seizures. Preoperative diagnosis is very difficult, and only around 20% of cases are diagnosed preoperatively. The most common preoperative diagnostic error is brain tumor (glioblastoma, metastatic brain tumor), due to the perifocal edema observed on imaging and gradually

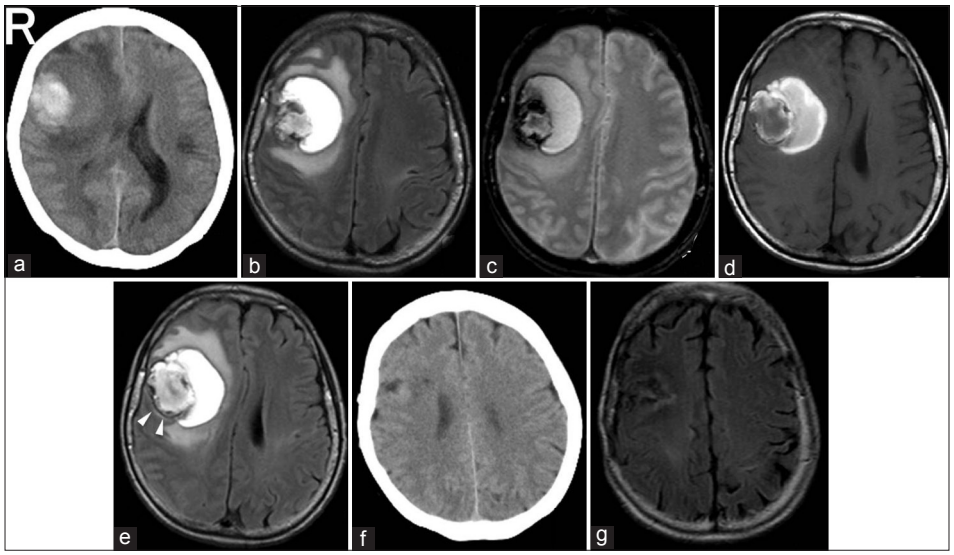

Figure 5: Case 3CT shows a faint hyper dense mass lesion in the right frontal lobe (a). FLAIR reveals the lesion as a mixed hypo- and hyperintensity mass with brain edema (b). T2*-weighted gradient echo imaging reveals the lesion as high intensity with low-intensity rims suggestive of hemosiderin deposition (c). Gadolinium-enhanced T1-weighted MRI shows ring enhancement (d). FLAIR imaging 2 weeks after initial MRI reveals that the lateral region of the mass has become hyperintense (arrowhead) (e). Follow-up CT and FLAIR MRI performed 6 months after surgery show no recurrence of CEIH (f,g)

Table 1: Patient characteristics and symptoms

Variable	*n*=57 (%)
Male	37 (64.0)
Age (mean)	44 years
(range)	0.2-80 years
Onset type	
Sudden onset	19 (33.3)
Gradual onset	37 (64.9)
Pre-operative diagnosis	*n*=28
Brain tumor	17 (53.6)
CEIH	6 (21.4)
Vascular malformation	3 (12.5)
Old hematoma	2 (7.0)
Craniotomy	55 (96.4)
Outcome	
Good	42 (73.6)
PND	8 (14.0)
Deceased	2 (3.5)
No detail	5

CEIH: Chronic encapsulated intracerebral hematoma, PND: Persistent neurological deficit

progressive neurological deficit. Our Cases 1 and 2 were diagnosed as brain tumor for the same reason.

We encountered three cases of CEIH with unidentified causes in which diagnosis was difficult. Because of chronological changes in the hematoma, CEIH shows various CT and MRI features. Cases 1 and 2 were therefore not initially diagnosed as CEIH. Case 3 was diagnosed as CEIH thanks to our experiences with Cases 1 and 2 and the use of phase imaging at multiple time points.

The hematoma in CEIH is commonly associated with vascular lesions such as arteriovenous malformation (AVM), cavernoma, micro-aneurysm, and venous angioma. In recent years, CEIH has also been found to be associated with stereotactic radiosurgery for AVM.[37-39]

Even if no vascular lesion is clearly identified, clinical features and characteristic findings on neuroimaging are useful for preoperative diagnosis of CEIH. First, the patient is likely to present with gradually progressive neurological deficit. Second, CT shows a faint high-density area. Third, CT and MRI demonstrate changes in the lesion over time. This last point helps to confirm the diagnosis, whereas misdiagnosis may result if imaging is performed only once. Fourth, CT and MRI show disproportionately severe perifocal edema. Fifth, T2-weighted and T2* imaging reveal a low-intensity rim. Close attention is necessary, because metastatic brain tumor sometimes shows similar features. Furthermore, many cases of CEIH show an avascular area on cerebral angiography. Eleven of the 33 reported cases in which cerebral angiography was performed showed an avascular area.

CEIH can be surgically removed with excellent outcomes. All but two cases of CEIH reported to date have been resected surgically, achieving good results. We recommend early surgical intervention and total removal of CEIH. Because of the tough membrane, separating the hematoma from normal brain tissue is very easy compared with cavernous angioma. We suggest total removal of the capsule to prevent recurrence of CEIH.

Although CEIH is a condition that is hard to diagnose preoperatively, good outcomes are provided by appropriate surgical treatment. CEIH should therefore be kept in mind as a differential diagnosis for intracerebral space-occupying lesions.

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Commentary

This is a well documented review of three cases of chronic encapsulated intracerebral hematoma, a very rare entity, particularly in the English literature. Clinical and radiological presentations and differential diagnoses are well discussed. Pathophysiology would suggest that the surrounding edema might indicate inflammation from some unidentified associated pathogen, but this is speculation. Even if true, it awaits further cases.

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