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Case Report

Organized Chronic Subdural Hematoma (OCSDH) Mimicking Meningioma

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Organized chronic subdural hematoma (OCSDH) is a relatively rare condition that forms over a longer period of time compared to chronic subdural hematoma and is sometimes difficult to diagnose with preoperative imaging. We resected an intracranial lesion in a 37-year-old Japanese man; the lesion had been increasing in size for >17 years. The preoperative diagnosis based on imaging findings was meningioma; however, pathological findings revealed OCSDH. Clinicians should be aware that OCSDH mimics other tumors and consider surgical strategies for this disease.

Key words: meningioma, organized chronic subdural hematoma

Chronic subdural hematoma (CSDH) is one of the most common neurological disorders, but organized chronic subdural hematoma (OCSDH) is a relatively rare condition, accounting for only 0.5-2% of CSDH cases [1]. A CSDH usually forms over a time course of at least 3 weeks after a head injury [2]. An OCSDH is thought to form over a longer time course, but the underlying mechanism remains unclear. Most cases of OCSDH are diagnosed with the use of preoperative computed tomography (CT) or magnetic resonance imaging (MRI) and are considered for excision if the patient is symptomatic [1,3-7]. The removal of an OCSDH has traditionally been performed through a large craniotomy in order to expose the entire OCSDH, but due to the nature of the disease, most patients with OCSDH are elderly, and the application of a small craniotomy with preoperative embolization of the middle meningeal artery was recently described [7].

Meningiomas are the most common brain tumor, accounting for >30% of primary tumors of the central nervous system [8,9]. Meningiomas are generally considered benign tumors and are often treated by resection. Most of them are diagnosed based on head CT or MRI findings, and it has been reported that meningiomas can mimic other malignant tumors or metastatic brain tumors [10,11]. Conversely, cases have been reported in which patients suspected of having a primary malignant tumor of the central nervous system, metastatic brain tumor, or OCSDH have been diagnosed with meningioma [12-16]. The diagnosis of meningioma made preoperatively thus sometimes turns out to be a different diagnosis postoperatively. We report the case of a patient with an OCSDH that was difficult to differentiate from meningioma due to atypical preoperative imaging findings.

Case Presentation

A 37-year-old Japanese male was referred to our hospital due to an intracranial lesion. At the age of 12 he had undergone a craniotomy and received bilateral

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ventriculo-peritoneal (VP) shunts for non-communicating hydrocephalus. At the age of 35 he underwent surgery for two neoplastic lesions in the lumbar region and was diagnosed with a schwannoma. In the same year, a tumor noted in the lung field was removed, and it was also confirmed to be a schwannoma. The patient's was referred to us for a mass lesion with calcification in the right frontal and left parietal areas. Shunt tractography demonstrated that the patient's VP shunts were occluded. He was diagnosed with neurofibromatosis type 1 (NF1) due to the presence of multiple neurofibromas and café au lait spots. No abnormalities in coagulation were observed. There was no family history of malignancy or NF1.

Plain CT of the patient's head showed a slowly enlarging mass with a thick capsule and internal calcification, plus bony destruction of the skull adjacent to the mass (Fig. 1A-D). Plain MRI revealed a well-defined mass with internal calcification and heterogeneous signal areas. Contrast-enhanced MRI showed no obvious contrast effect, although there was an accompanying structure that appeared to be a dural tail sign (Fig. 1E-M). Both lesions had already been observed on head CT when the patient was 19 years old (Fig. 1N, O), but that prior imaging information was unavailable.

The increasing size of the lesion over the prior 17 years and the imaging findings suggested the possibility

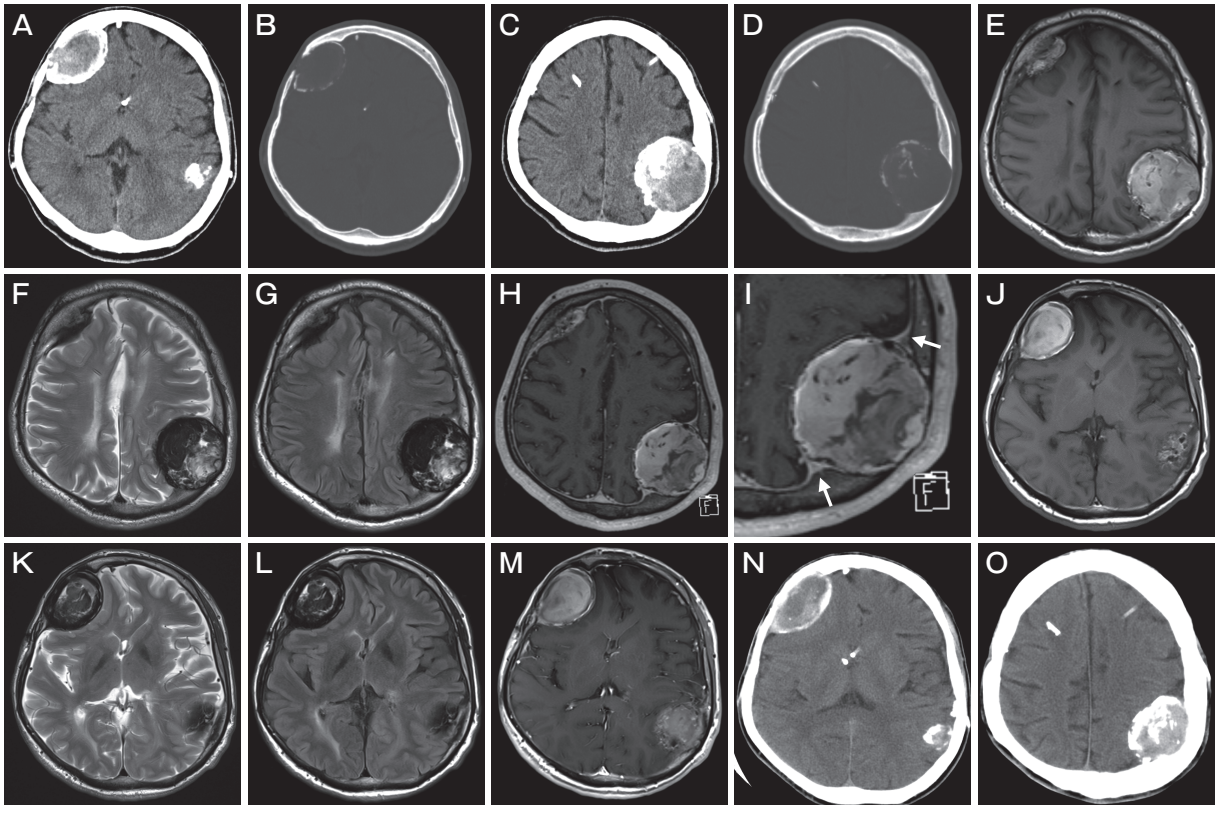

Fig. 1 Radiographic findings of the patient's lesions. **A**, Right frontal lesion at age 37 with a thick calcified capsule; **B**, Right frontal lesion (age 37) showing bone destruction; **C**, Left parietal lesion (age 37) with a thick calcified capsule and internal calcification; **D**, Left parietal lesion (age 37) showing bone destruction; **E**, T1-weighted image (T1WI) of the left parietal lesion showing relatively high signal uniformity within the tumor; **F**, T2-weighted imaging (T2WI) of the left parietal lesion presenting mixed low and high signal within the tumor; **G**, Fluid-attenuated inversion recovery (FLAIR) of the left parietal lesion presenting mixed low and high signal within the tumor; **H**, No obvious enhancement was observed in the left parietal lesion by contrast-enhanced T1WI; **I**, Contrast-enhanced T1WI of the left parietal lesion presenting as suspicious for dural tail sign (*arrows*); **J**, T1WI of the right frontal lesion presenting with a relatively uniform high-signal tumor; **K**, T2WI of the right frontal lesion presenting with a mixed low- and high-signal tumor; **L**, FLAIR of the right frontal lesion presenting with a mixed iso- to high-signal tumor; **M**, No obvious enhancement on contrast-enhanced T1WI; **N**, Right frontal lesion (age 19); **O**, Left parietal lesion (age 19).

of a meningioma. Although the patient was asymptomatic at his presentation to our department, we suspected that he would be symptomatic in the near future as the mass had increased in size. We thus decided to perform a craniotomy to address the left parietal lesion. Digital subtraction angiography was conducted prior to the surgery, and it detected no vessels feeding the tumor (Fig. 2A-J). Tumor embolization was therefore not performed.

The patient was under general anesthesia for the surgery. The lesion had destroyed bone, and the dura was brown in color and bulging toward the surface. The lesion was filled with a dark brown clay-like substance and surrounded by thick calcified hard tissue. Several pieces of tissue from different areas were subjected to a frozen-section examination, which revealed no tumor cells. Because the surface of the lesion was strongly adherent to the thickened arachnoid membrane, we removed as much lesion as possible without forcibly removing some areas of calcification. The skull was closed with custom-made artificial bone. The final surgical procedure was a craniectomy, hematoma removal, and cranioplasty using artificial bone (Fig. 3A-C).

The pathological findings of the excised lesion were mainly coagulum with strong internal necrosis and degeneration, with no findings of tumor cells (Fig. 3D, E). Based on these findings, the final diagnosis of the resected lesion was OCSDH. We suspected that the lesion in the patient's right frontal lobe was also likely to be OCSDH, and we decided to continue an imaging follow-up with head CT. Follow-up with plain CT was continued for 1 year after the surgery, and no recur-

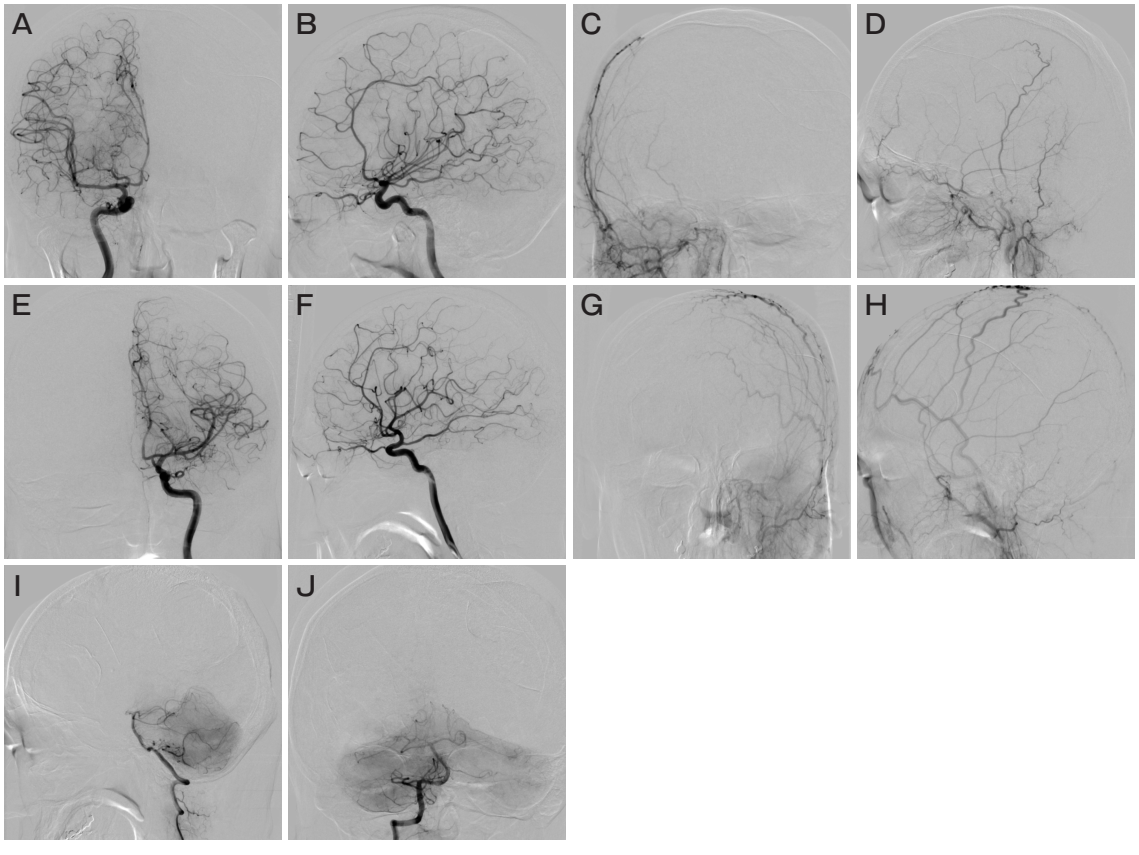

Fig. 2 Preoperative digital subtraction angiography findings. **A**, Frontal view, right internal carotid artery (ICA) angiography showing no feeding artery; **B**, Lateral view, right ICA angiography showing no feeding artery; **C**, Frontal view, right external carotid artery (ECA) angiography showing no feeding artery; **D**, Lateral view, right ECA angiography showing no feeding artery; **E**, Frontal view, left ICA angiography showing no feeding artery; **F**, Lateral view, left ICA angiography showing no feeding artery; **G**, Frontal view, left ECA angiography showing no feeding artery; **H**, Lateral view, left ECA angiography showing no feeding artery; **I**, Frontal view, right vertebral artery (VA) angiography showing no feeding artery; **J**, Lateral view, right VA angiography showing no feeding artery.

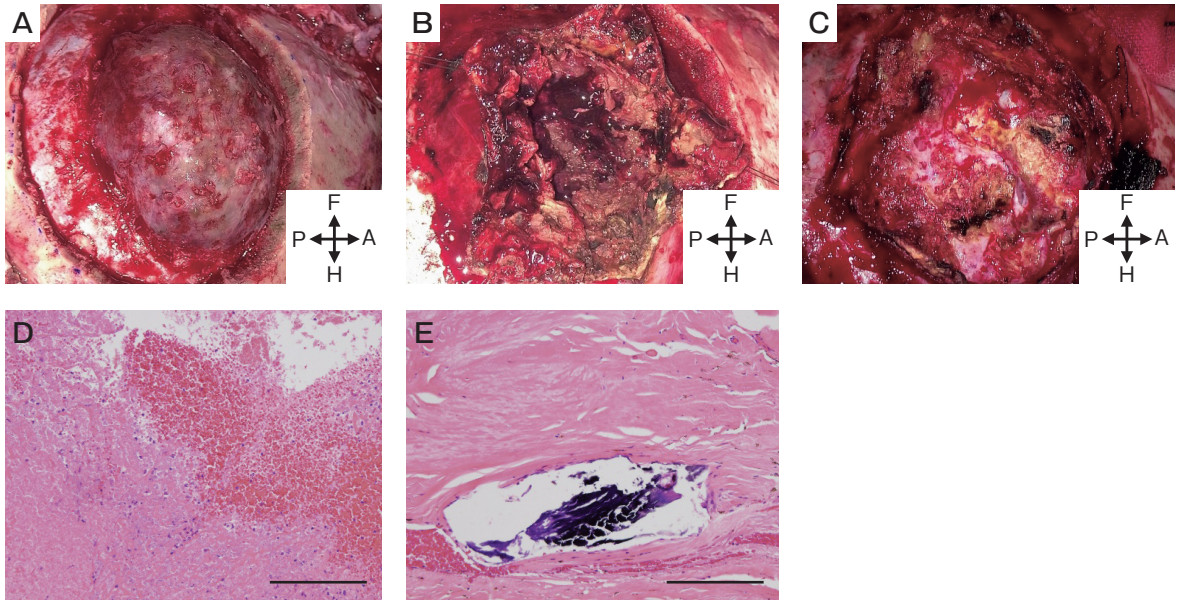

Fig. 3 Intraoperative and pathological findings of the left parietal lesion. **A**, Skull was removed, and the appearance of the lesion was brown and generally bulging; **B**, The lesion was filled with partially clay-like clots; **C**, The operative field after removal of the hematoma and capsule. Pathological findings showing blood clots with necrotic tissue (**D**) and the capsule showing fibrous tissue with calcification (**E**). Black bar: 200 μm.

rence was observed in the extraction cavity in the left parietal area; in addition, the size of the right frontal lobe lesion did not increase (Fig. 4A-D).

Ethics approval. All of the patient's procedures were conducted in accord with our hospital's ethical standards and with the 1964 Helsinki Declaration and its later amendments. This report was approved by our institutional review board (IRB# 1911-023). The patient's written consent for the publication of this report and his images was obtained.

Discussion

CSDH develops after head trauma and is a relatively common disease encountered in daily neurosurgery practice. OCSDH is rarely reported, and compared to CSDH it takes a longer time to form. Although in the elderly a CSDH commonly occurs after head trauma, cases in children have been reported, especially in patients with a VP shunt, trauma, or blood disorders [17, 18]. In our patient's case, preoperative shunt tractography confirmed that the shunt tubes were occluded, but the lateral ventricles were slit-like. These findings suggested that a tunnel had formed around the shunt tube or another corridor that allowed an adequate drainage of spinal fluid. Although it is not clear whether the patient had a CSDH prior to his OCSDH, the OCSDH was noted when he was 19 years old. It is possible that the VP shunts placed during the patient's childhood caused excessive spinal fluid drainage and may have caused the CSDH, which might have led to the OCSDH.

Most of the reported cases of OCSDH were diagnosed by preoperative imaging modalities such as head CT and MRI. Imaging findings of OCSDH generally include a thickened and calcified capsule on CT, a high signal on T2-weighted imaging (T2WI), and a low-to-high signal on T1-weighted imaging (T1WI) with a heterogeneous internal signal and surrounding low intensity reflecting capsular calcification on MRI [5, 19]. Unlike our patient's case, there are reports of patients who underwent surgery with a preoperative diagnosis of OCSDH that turned out to be a meningioma. The preoperative diagnosis in those cases was based on a history of head contusion and the findings of plain CT that mimic OCSDH [20, 21].

Miki *et al.* reported the usefulness of MRI for identifying OCSDH, noting that postoperative MRI showed a high signal on T1WI, a low signal on T2WI, and an iso-signal on fluid-attenuated inversion recovery

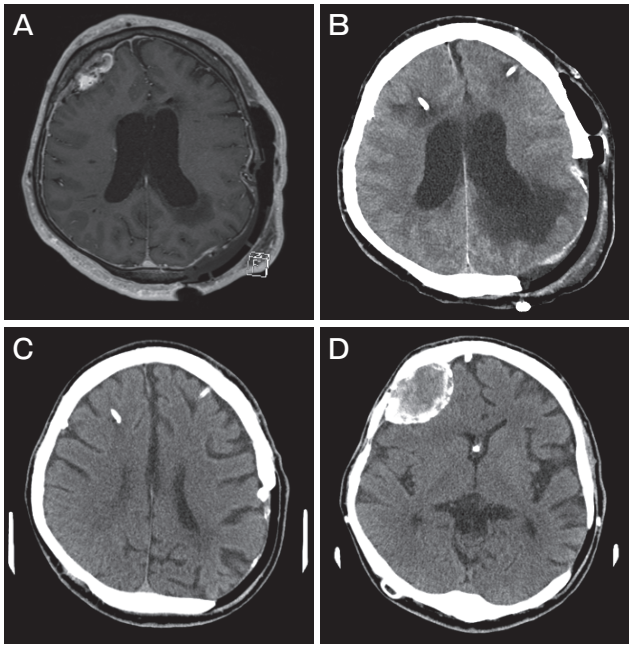

Fig. 4 Postoperative and follow-up images. **A**, Postoperative Gd-T1WI showing the left parietal extraction cavity at the time of the patient's first image evaluation after extraction; **B**, Postoperative CT showing the left parietal extraction cavity; **C**, CT showing no recurrence of the left parietal lesion 1 year postsurgery; **D**, CT showing no enlargement of the right frontal lesion 1 year postsurgery.

(FLAIR) findings, consistent with meningioma. Wu *et al.* also described the usefulness of MRI in a patient who exhibited a dural tail sign suggestive of meningioma. Our patient had two intracranial lesions, both of which had increased in size over a number of years. MRI showed mixed images of an iso- to high signal on T1WI and low-to-high intensity on T2WI, consistent with OCSDH. Nevertheless, imaging also showed findings suggestive of meningioma, such as the thick-tailed sign and bone destruction in the skull adjacent to the hematoma, making it difficult to confirm the preoperative diagnosis based on preoperative imaging findings. In this case, the surgeon was prepared for a meningioma and was able to successfully complete the resection. Conversely, if a meningioma had been expected to be an OCSDH, the surgical procedure may have been difficult due to intraoperative bleeding and other factors. Therefore, even when an OCSDH is suspected, surgeons should keep in mind the possibility of a meningioma when preparing for surgery.

OCSDH is generally followed up in asymptomatic cases, but surgical excision is performed in symptomatic cases [1, 2]. Conventionally, a craniotomy is performed to remove the entire dura mater, including the hematoma. The rationale for recommending a large craniotomy for dural resection is that the epidural layer can cause hematoma expansion due to repeated bleeding, and resection of the intima can promote expansion of the brain parenchyma into the subdural space [5]. In contrast, a technique has been proposed to remove the hematoma and dura mater as feasibly as possible through a small craniotomy. The advantage of a small craniotomy is that it is less invasive for OCSDH patients, many of whom are elderly and have systemic complications.

An investigation of the postoperative results of large and small craniotomies found no significant difference between the patients who had undergone a large craniotomy and those who had undergone a small craniotomy [6]. A small craniotomy combined with middle meningeal artery embolization was also recently reported to reduce the postoperative recurrence rate [7]. In our patient's case, a large craniotomy was performed to surround the mass based on the suspicion that it was a meningioma, and the OCSDH was completely removed, including the capsule and dura mater, with no postoperative recurrence.

Conclusion

Our patient's OCSDH mimicked meningioma in preoperative imaging. When clinicians are selecting a treatment strategy for OCSDH, it is necessary to consider several treatment strategies such as a small or large craniotomy or observation, taking into account the patient's age and general condition. In addition, if other neoplastic lesions such as meningiomas are suspected, a large craniotomy may be advisable.

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