



A Rare Case of Chronic Subdural Hematoma Coexisting With Metastatic Tumor

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Key words

- Acute subdural hematoma
- Chronic subdural hematoma
- Intratumoral hemorrhage
- Metastatic tumor

Abbreviations and Acronyms

CSH: Chronic subdural hematoma

CT: Computed tomography

MRI: Magnetic resonance imaging

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BACKGROUND

Although chronic subdural hematoma (CSH) is commonly observed in clinical practice, it rarely develops in association with intracranial metastasis. Most reported cases of the latter have been associated with dural metastasis, and tumor cells were almost never detected in the hematoma cavity.^{1,2} We report a rare case in which a patient with a history of gastric cancer showed sudden deterioration following CSH treatment; the CSH cavity was found to contain metastatic tumor cells, and the complete removal of the tumor and hematoma capsule was necessary.

CASE REPORT

A 68-year-old man with a history of type 3 poorly differentiated gastric adenocarcinoma, which was treated with subtotal gastrectomy and Roux-en-Y gastric bypass as well as postoperative adjuvant chemotherapy with titanium silicate-1 2 years previous, presented with dysarthria and right upper limb paralysis 2 months after suffering an apparent head trauma.

■ BACKGROUND: Incidence of chronic subdural hematoma (CSH) associated with metastases of extraneural malignancies is rare. We report a rare case of CSH wherein most of the CSH cavity was occupied with metastatic cancer cells; in addition, we review the literature.

■ CASE DESCRIPTION: A 68-year-old man with a history of gastric cancer presented to our hospital with dysarthria and shoulder paralysis; CSH was diagnosed from preoperative imaging findings. When the hematoma was removed via a small craniotomy, besides the hematoma, we observed an abnormal mass of tissue in the capsule. Pathologically, the mass was consistent with the findings of metastatic gastric cancer. Although the symptoms immediately disappeared postoperatively, a symptomatic acute subdural hematoma with midline shift was observed on postoperative day 27. Emergency craniotomy and hematoma and tumor removal were performed. Pathologic examination showed hemorrhagic necrosis in the tumor, which had not been initially observed. The postoperative course progressed without hematoma recurrence.

■ CONCLUSIONS: To the best of our knowledge, this is the first report of a CSH accompanied by tumor metastasis in most of the CSH cavity. Although rare, if a patient with cancer has CSH, the CSH should be treated considering the possibility of metastasis.

Although the patient had voluntarily discontinued treatment for the gastric cancer for a short duration, the onset of ileus 3 months previous resulted in the diagnosis of recurrent gastric cancer in the left renal hilum lymph node (pT4N3Mo, IIIC), and he began treatment with a combination chemotherapy of oxaliplatin and titanium silicate-1. The initial laboratory analysis performed in our department revealed no abnormality in the coagulation system. Computed tomography (CT) and magnetic resonance imaging (MRI) revealed a left-sided slight low-density mass and heterogeneous-intensity mass, respectively, with a slight midline shift (Figure 1A–E). The images suggested CSH with partial acute/subacute hematoma; therefore, the patient was immediately taken to the operation theater for a small craniotomy under local anesthesia, rather than a burr hole evacuation, which is typically performed for CSH.

When the dura mater was opened during surgery, a thick outer membrane of the hematoma capsule was exposed; besides

the oil-black liquid typical of CSH, the hematoma cavity contained a white and brown solid mass (Figure 2A and B). We removed the hematoma and solid mass to the maximum extent. Hematoxylin and eosin staining of the solid tissue revealed poorly differentiated adenocarcinoma, consistent with a metastasis of the gastric adenocarcinoma (Figure 2C). The boundary between the tumor and capsule was clear, and no tumor cells were observed in the outer membrane. Postoperatively, the patient's symptoms completely disappeared; however, the appearance on the imaging examination did not show a clear improvement owing to residual mass effect (Figure 1F). Because most of the tumor was not removed, we scheduled an additional surgery for total hematoma removal by craniotomy under general anesthesia; the patient refused it, however, and he was discharged on postoperative day 7.

On day 20 after hospital discharge, the patient was readmitted with

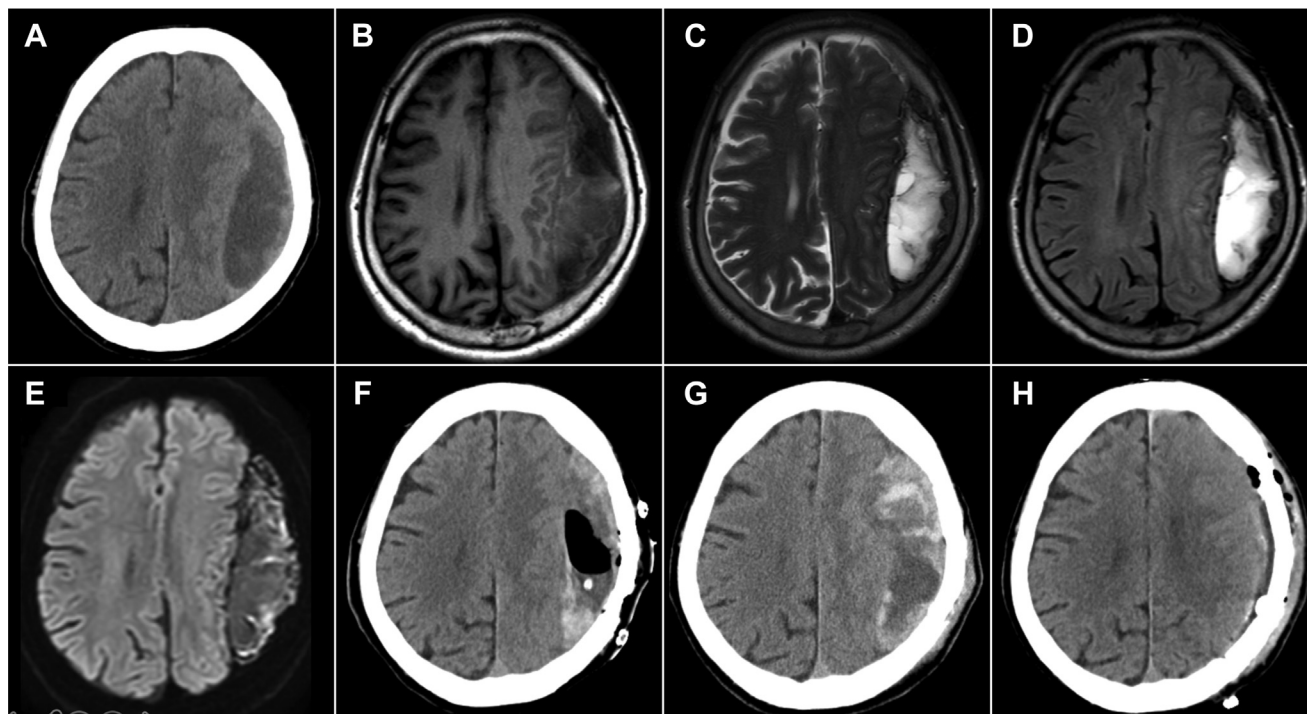


Figure 1. (A) Preoperative axial computed tomography (CT) scans of the head. The CT scans show a left-sided slight low-density mass with a slight midline shift. The mass included a small part of high-density area. (B–E) Preoperative nonenhanced magnetic resonance images of the head. The mass is mostly hypointense on diffusion-weighted images (B) and T1-weighted images (C). The mass is hyperintense on T2-weighted images (D) and fluid-attenuated inversion recovery images (E) with several septa and includes a hypointense area as indicated. (F–H) CT scans obtained a

day after the first surgery (F), at the time of recurrence (G), and after the second surgery (H). (F) CT scan showing that the hematoma and tumor were partially removed under local anesthesia, and the brain pressure improved. Tumor surrounding the excision cavity remained with slight residual mass effect. (G) CT scans showing the exacerbation of subdural hematoma in the left frontal area where the tumor remained after the first surgery; remarkable midline shift is observed. (H) CT scan obtained after the second surgery showing no residual tumor or hematoma.

consciousness disorder. His Glasgow Coma Scale score was 12 of 15 (E3V4M5), and he had moderate right hemiparesis at the time of readmission. An emergency CT performed revealed an acute subdural

hematoma with midline shift in the left frontal area, where the tumor had not been removed during CSH removal (Figure 1G). The patient required emergency surgery for mass reduction.

Because the inner membrane of the hematoma did not adhere to the arachnoid mater and brain surface, we removed the tumor and hematoma capsule en bloc (Figure 3A and B). The

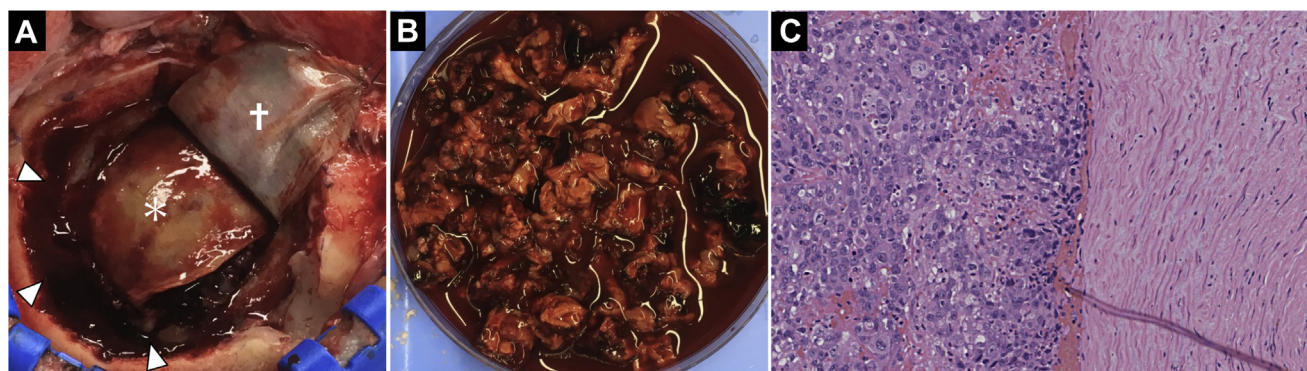


Figure 2. (A and B) Intraoperative findings during the first surgery. (A) The dura mater (dagger), the outer membrane of the hematoma capsule (asterisk), and the hematoma that drained spontaneously from the cavity (white arrowheads). (B) Solid tissues in the hematoma cavity. (C) Pathologic

findings of the hematoma and capsule after the first surgery. Hematoxylin and eosin staining reveals the border between the metastatic tumor and the outer membrane of the hematoma capsule. No tumor cells are found in the membrane (original magnification, $\times 200$).

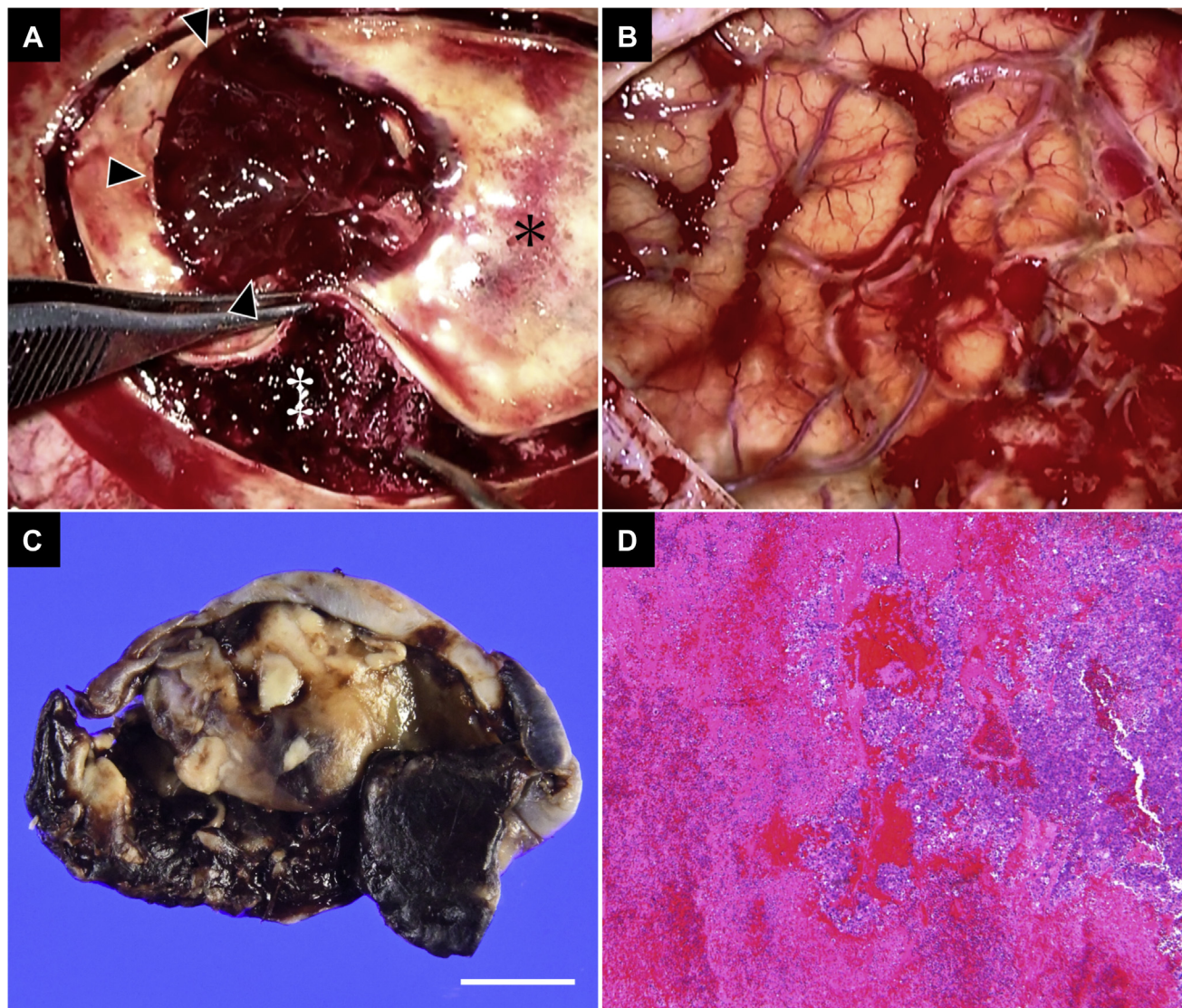


Figure 3. (A and B) Intraoperative findings at the second surgery. (A) The outer membrane of the hematoma capsule (*asterisk*), solid tumor mass mixed with hematoma (*double dagger*), and defect of the outer membrane of the hematoma capsule following the first surgery (*black arrowheads*) are shown. The outer membrane of the hematoma capsule was thickened, and most of the hematoma cavity was occupied by tumor mass mixed with hematoma. (B) The tumor and inner membrane of the hematoma capsule

were completely removed, and the arachnoid membrane was preserved on the brain surface. (C) Macroscopic examination of the hematoma and the inner membrane. The membrane was thickened, and tumor components had adhered. Scale bar indicates 10 mm. (D) Pathologic findings of the hematoma and capsule after the second surgery. Hematoxylin and eosin staining of the hematoma contents revealed hemorrhagic necrosis in the tumor (original magnification, $\times 100$).

dura mater did not adhere to the outer membrane of the hematoma capsule, and no suspicious findings of dural metastasis were observed; therefore, the dura mater was preserved. Postoperative CT revealed no characteristics of residual hematoma or tumor recurrence (Figure 1H). Histopathologic examination showed hemorrhagic necrosis in the tumor, which had not been confirmed during the first operation (Figure 3C and

D). Chemotherapy was resumed after the patient was discharged from our hospital, and no recurrence of the hematoma has been detected since (>2 months).

DISCUSSION

The mechanisms underlying the pathogenesis of CSH associated with tumor is typically considered to be the following:

(1) hemorrhagic effusion from the metastatic tissue, (2) dural vessel obstruction by neoplastic cells, and (3) an angiodysplastic reaction of the dura to tumor infiltration.³ Additionally, some investigators have reported the direct hematogenous metastasis of malignant tumors to the CSH capsule as rare cause of CSH.^{4,5}

In our patient, the intraoperative findings, postoperative radiologic findings,

and histologic findings indicated neither dural nor hematoma membrane metastasis. From the point of the metastatic route to CSH, the dura mater and CSH outer membrane received ample blood flow from the middle meningeal artery,⁶ and tumor cells may have been transported via this artery. However, no evidence of metastatic tumor cells on the dura mater and outer membrane was observed in the present patient. Although the possibility of metastasis via a meningeal artery could not be ruled out, these findings in our patient may have resulted from a different metastatic route, such as brain parenchymal vessels. In addition, most of the hematoma cavity was occupied by the tumor mass, and the mass effect of the tumor might be greater than that of the hematoma. With regard to the unclear metastatic route and existing tumor in the hematoma cavity, no other case of CSH coexisting with metastatic tumor, as observed in the present case, has previously been reported.

It was difficult to diagnose the presence of the tumor within the hematoma cavity using preoperative imaging. In ordinary CSHs, CT often shows hypodensity; however, if bleeding has recently occurred, a part of the area may show hyperdensity.⁷ MRI is reportedly superior to CT in determining the size and content of hematomas, particularly the presence of the septa in CSH.⁸ MRI can depict the stages of hematoma: in the early stage, T1- and T2-weighted images show low signaling that corresponds to fresh hemorrhage; in the middle stage, T1-weighted images show a high-intensity signal, with reduced blood vessel rupture; and in the late stage, T2-weighted images show high-intensity signals, regardless of T1-weighted images showing high or low intensity signal.⁹ These various images obtained during different phases of CSH may have led to preoperative misdiagnosis of a simple CSH in our patient. Retrospectively, there were areas

of mixed signal intensity within the hematoma, and several septum-like structures were observed, reflecting the neoplastic components within the hematoma.

In the first surgery, we performed a small craniotomy, which we occasionally perform for CSH when it is expected that burr hole evacuation would result in inadequate removal; however, on the postoperative images, the residual mass effect showed no improvement because of the remaining tumor, although the symptoms disappeared. During the postoperative course, acute subdural hematoma developed as a result of intratumoral hemorrhage in the left frontal area, where the tumor could not be removed during the first surgery. In cases where an acute subdural hematoma is caused by intratumoral hematoma, in addition to hematoma removal, it is necessary to remove the tumor to the maximum extent, considering the risk of rebleeding. During the second surgery, the entire tumor, including the hematoma capsule membrane, was successfully resected. Complete removal of the subdural metastasis and hematoma may improve the prognosis and quality of life of the patient.

CONCLUSIONS

To the best of our knowledge, this is the first report of a CSH where most of the CSH cavity was occupied by metastatic tumor cells. If a patient with cancer has CSH, the CSH should be managed considering the possibility of metastasis; if metastasis is detected, prompt removal of the entire CSH should be performed owing to the risk of hemorrhage within the tumor or tumor recurrence.

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