Supra- and Infratentorial Subdural Empyema Secondary to Septicemia in a Patient With Liver Abscess
—Case Report—

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Abstract
An 81-year-old man presented with subdural empyema in the left parietotemporal convexity 2 months after treatment under diagnoses of liver abscess and septicemia. Systemic investigation found no evidence of otorhinological or other focal infection except for liver abscess. Emergency drainage of pus was performed via a single burr hole and additional intravenous antibiotics were administered. Six weeks later, magnetic resonance imaging revealed subdural empyema in the right cerebellopontine angle in addition to recurrence of pus in the left parietotemporal subdural space. Ischemic changes were also shown in the right cerebellar hemisphere and brainstem. Although subdural empyema secondary to septicemia is rare, the possibility of this type of intracranial infection must be kept in mind, especially in compromised patients with septicemia.

Key words: infarction, liver abscess, septicemia, subdural empyema

Introduction
Subdural empyema is a rare and highly lethal central nervous system infection, and usually arises as a complication of paranasal sinusitis, otitis media, or head trauma. Only two of 699 cases of intracranial subdural empyema originated from sepsis. We report a case of supra- and infratentorial subdural empyema which caused extensive ischemic change to the cerebellum and brainstem in a patient with liver abscess and septicemia.

Case Report
An 81-year-old man was transferred to our hospital from a nearby hospital to which he had been admitted 2 months earlier under diagnoses of cholelithiasis and liver abscess. The diagnosis of liver abscess was based on endoscopic retrograde cholangiopancreatography and computed tomography (CT) (Fig. 1). The patient’s past history was unremarkable. Klebsiella pneumoniae was isolated from his blood. He was treated with intravenous cefazolin sodium and amikacin sulfate for one month. CT revealed gradual regression of the liver abscess, although low-grade fever persisted. Three days prior to admission to our hospital, he presented with headache and stiff neck. CT revealed subdural fluid collection with marginal enhancement in the left parietotemporal convexity (Fig. 2A).

On admission, neurological examination found only stiff neck. He was in a state of shock. Emergency drainage of the abscess was performed via a
Fig. 2 A: Preoperative computed tomography (CT) scan with contrast medium on admission demonstrating extensive subdural collection of pus in the left parietotemporal subdural space. B: Postoperative CT scan with contrast medium revealing disappearance of subdural empyema. The arrow indicates the drainage tube.

Fig. 3 A, B: T1-weighted magnetic resonance (MR) images with gadolinium demonstrating subdural empyema in the cerebellopontine angle and in the left parietotemporal subdural space. Extensive thickening of dura mater adjacent to the subdural empyema is also visible. C: T2-weighted MR image showing hyperintense areas in the right part of midbrain and right cerebellar hemisphere, suggesting ischemic changes.

Discussion

Intracranial subdural empyema is associated with significant morbidity and mortality. Early surgical drainage, simultaneous eradication of the primary source of sepsis, and intravenous administration of high doses of appropriate antibiotic agents have all been recommended, although nonsurgical treatment of subdural empyema has also been reported. The optimum surgical management of subdural empyema is widely thought to be a large craniotomy with aggressive removal of subdural pus rather than the burr hole approach, although the burr hole should not be disregarded. In our case, we decided on the burr hole approach with catheter drainage because of the advanced age of the patient and state of shock on admission. Postoperative MR imaging showed recurrence of

single burr hole. The dura mater was opened and bloody purulent fluid was evacuated. After irrigation of the subdural space with saline containing flomoxef sodium (1 g), a drainage tube was placed in the subdural space for 4 days to eliminate the remaining pus. CT with contrast medium on the first postoperative day showed disappearance of the subdural empyema (Fig. 2B). Culture of the pus was negative, although methicillin-resistant Staphylococcus aureus was isolated from the blood culture taken on the first postoperative day. The patient was treated with intravenous vancomycin hydrochloride for 1 week followed by intravenous cefotaxime sodium. Two weeks after the operation, the blood culture was negative.

Six weeks after the operation, he presented with right facial paresis and disturbance of consciousness. T1-weighted magnetic resonance (MR) imaging showed recurrence of pus in the left parietotemporal convexity and newly-formed subdural empyema in the right cerebellopontine angle (Fig. 3A, B). The dura adjacent to both lesions was enhanced by contrast medium. T2-weighted MR imaging disclosed hyperintense areas in the right cerebellar hemisphere and right pons, indicative of ischemic changes (Fig. 3C). He was treated with intravenous edaravone for 14 days. His right facial paresis improved. Two months after admission to our hospital, he was transferred to a chronic care hospital, although CT showed that subdural empyema persisted in the left parietotemporal convexity and the right cerebellopontine angle.
the subdural empyema in the left parietotemporal convexity. Earlier detection of the recurrence of the subdural empyema and subsequent surgical removal of pus could have prevented the additional dissemination of empyema into the posterior fossa.

In this case, the pus taken from the subdural empyema was sterile. However, we concluded that the subdural empyema was secondary to the liver abscess and septicemia, because there was no remarkable past history and no evidence of otorhinologic or other focal infection except for the liver abscess. Several cases of subdural empyema have been caused by hematogenous dissemination. Preexisting chronic subdural empyema may contribute to the development of the subdural empyema by hematogenous dissemination. In our case, no preexisting chronic subdural empyema was found by CT.

The subdural empyema spread into the contralateral cerebellopontine angle despite complete evacuation of the pus and intravenous administration of antibiotics. Infratentorial empyema is an uncommon and lethal form of intracranial suppuration that usually occurs secondary to neglected otogenic infection. Two previous cases of posterior fossa subdural empyema following supratentorial subdural empyema as a complication of acute frontal sinusitis suggested that the pus could seep from the established supratentorial empyema into the infratentorial compartment aided by gravity and changes in the position of the head. In our case, the cerebellopontine angle empyema was found on the opposite side to the supratentorial subdural empyema. Therefore, bacteria which entered the blood stream from the left supratentorial lesion may have been implanted in the right cerebellopontine angle. However, we could not exclude the possibility of the direct extension of pus from the supratentorial to the infratentorial compartments.

In our case, the infarction was localized in the territories of the right posterior and anterior inferior cerebellar arteries, superior cerebellar artery, and perforating vessels branching from the basilar artery, all of which seemed to be affected by the nearby pus in the cerebellopontine angle. Cerebral infarction is known to occur secondary to subdural empyema. Indeed, reduced regional cerebral blood flow is a frequent finding in adult patients with bacterial infection. The angiographic findings of intracranial infection include arterial narrowing and occlusion caused by vasospasm, retrograde flow, slowing of circulation, venous dilation, massive shifting of the vessels, and extravasation of contrast medium. Such angiographic changes are also influenced by vasculitis with involvement of the intima. Septic cerebral embolism may also cause cerebral infarction. The death of patients with cerebellopontine angle extension of pus may have been caused by thrombophlebitis of vital brainstem perforating vessels with subsequent infarction. In our case, the extensive ischemic change in the posterior fossa was likely to be a vascular complication of the subdural empyema in the cerebellopontine angle.

Supra- and infratentorial subdural empyema caused infarction in the cerebellum and brainstem of the present patient. This case suggests that we should be aware of the possibility of extensive bacteremic seeding into the subdural spaces secondary to liver abscesses in elderly patients.

References

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