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Case Report

# Bilateral Chronic Subdural Hematoma Contaminated with Klebsiella Pneumoniae : An Unusual Case

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This article presents the case of a bilateral chronic subdural hematoma which was contaminated with Klebsiella pneumoniae and resulted in a life-threatening central nervous system infection. After repeated of bilateral burr-hole drainage, the patient became hyperpyrexic and drowsy. Suppuration within the subdural space was suspected and then the patient underwent bilateral fronto-temporo-parietal craniotomies, and pus was evacuated. Its cultures revealed Klebsiella pneumoniae. Intravenous meropenem was given for 6 weeks. He recovered completely. Microorganisms like Klebsiella pneumoniae may directly infect the subdural space with iatrogenic contamination.

**KEY WORDS :** Bilateral chronic subdural hematoma · Burr-hole drainage · Contamination · Klebsiella pneumonia · Subdural empyema.

## INTRODUCTION

Subdural empyema (SDE) which refers to suppuration along the intracranial subdural spaces is a common condition accounting for 13-23% of all intracranial infections[20]. Although the advents of new generation antibiotics and early diagnosis with ever-developing diagnostic radiology have helped greatly in decreasing the rate of suppuration, it still bears high morbidity and mortality (7-30%). Yet, SDE developing as suppuration of chronic subdural hematoma material (CSDH) is a rare condition[1,3,4]. We present a case of a bilateral CSDH which was contaminated with Klebsiella pneumoniae and resulted in a life-threatening central nervous system infection.

## CASE REPORT

The patient was a 48-year-old right-handed man who suffered from intense headaches for 15 days prior to admission. There was a history of a minor head injury while loading off his car trunk approximately 8 weeks prior to development of headaches. A small scalp laceration was treated with local antibiotic ointments and dressing. He has consumed moderate ethyl alcohol and has taken acetyl salicylic acid for prophylaxis of hyperlipidemia for over a year. When cranial magnetic resonance (MR) scans showed bilateral fronto-temporo-parietal (FTP) CSDH, bilateral burr-hole drainage was done elsewhere as the first line treatment (Fig. 1). At first surgery, brownish liquefied hematoma content was evacuated from each side of the brain and the subdural space was irrigated and a temporary closed drainage system was placed. Fifteen days after the initial drainage the patient became nauseated with recurring headaches. With cranial computed tomography (CT) scans showing re-accumulation of FTP chronic subdural hematoma bilaterally with persisting frontal air, he was again taken for bilateral burr-hole hematoma drainage and a closed subdural drainage system insertion. The patient was tous two days after repeated burr-hole surgery. The drainage system was removed the same day and he became hyperpyrexic and drowsy. Laboratory report showed elevated white blood cell (WBC) counts and C-reactive protein levels as well as increased liver enzymes. Despite the lack of neck stiffness and other meningeal irritation signs, a lumbar tap with a fine needle was considered necessary. The cerebro-spinal fluid (CSF) protein was slightly elevated but no organism or inflammatory cell was detected under microscopy. CT of the chest, ultrasound (US) of the abdomen and venous Doppler-US of the lower limbs revealed no infectious focus. Since all the culture results of blood, urine, feces and the CSF were negative, empiric sulbactam-sefaperazone IV was

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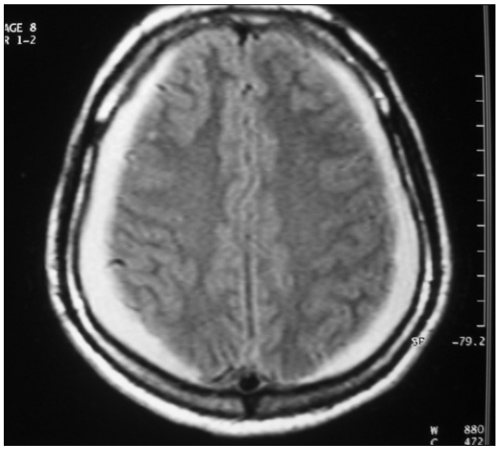

**Fig. 1.** T1-weighted magnetic resonance scan before the first operation.

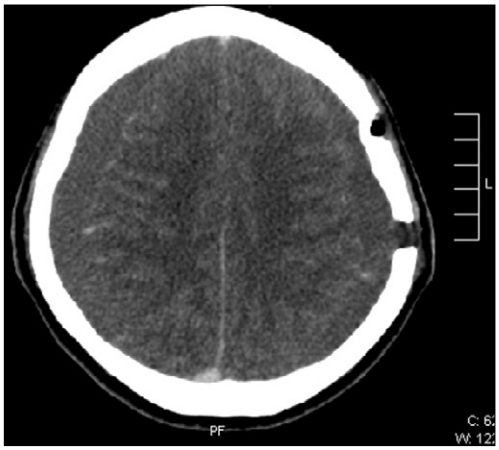

**Fig. 2.** Cranial computed tomography scan with contrast reveals bilateral subdural empyema which was isodense with brain parenchyma.

started. Brain CT obtained postoperatively demonstrated persistence of bilateral fronto-temporo-parietal effusion with same density as the brain parenchyma (Fig. 2). Considering progressive drowsiness such as lethargy with no verbal or eye response to painful stimuli and the fact that no source for the febrile condition was identified outside the central nervous system, suppuration within the subdural space was suspected. He then underwent bilateral FTP craniotomies. At surgery, yellow-brown pus gushed out from preexisting burr-holes. Suppurated hematoma membranes were removed totally. The piamater was covered with white-yellow pertinacious material especially along the cerebral vessels and the brain appeared hyperemic bilaterally. The patient awoke from the surgery with a close-to-normal speech and a Glasgow Coma Scale (GCS) score of 14. Fractionated heparin was used to prevent septic cerebral venous and/or dural venous sinus thrombosis. The cultures of the subdural pus revealed growth of Klebsiella pneumoniae which was sensitive to all antibiotics but not to ampicillin. After discontinuation of sulbactam-sefoperazone, 1 gm of meropenem was initiated intravenously three times a day.

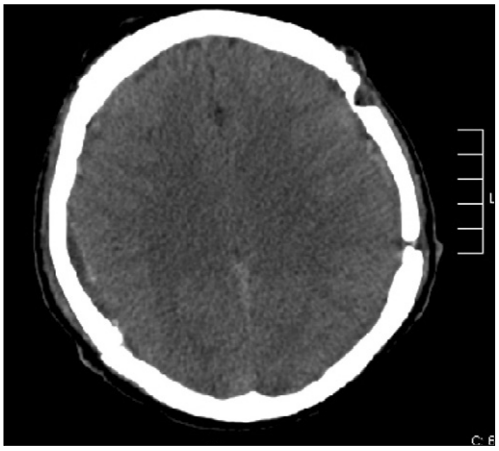

**Fig. 3.** Cranial computed tomography scan before the patient's discharging from the hospital.

Three days after craniotomy, his consciousness level decreased once again and became right hemiparetic and dysphasic. Cranial CT scans showed re-accumulation under the right bone flap and he then underwent re-exploration of the right craniotomy site. Epidural clot adherent to the bone was removed and subdural exploration revealed 2-3 mm fresh clot formation over the cerebral cortex. After right craniotomy revision, consciousness improved a little but dysphasia progressed to aphasia. After two postoperative seizures, the patient had to be intubated and mechanically ventilated under intracranial pressure monitoring. Three days later, 6 days after the initial craniotomy, he underwent re-exploration of the left craniotomy site. Again the epidural clot adherent to the bone was removed and fresh and soft clot over the left cerebral cortex was removed. After fifth surgery, consciousness level and power recovered rather quickly but speech improved slowly (Fig. 3). Cranial MR scans obtained at 15th day demonstrated left temporal edema and MR-angiography demonstrated that all dural venous sinuses were patent.

The clotting cascade parameters of protein C and S activities, antithrombin III, factor V-Leiden mutation, opsonization factors (complement C3, C4) and serum immunoglobulin (Ig A, D, G, M) levels were in normal range.

The histopathological examination of the subdural membranes revealed infiltration with lymphocytes, polymorph nuclear leukocytes, foreign body-giant cells and macrophages with hemosiderine. Bone curetted from the inner aspect of the previous burr-holes revealed degenerated bone tissue with new bone formation but not osteomyelitis.

A month later he was discharged home with GCS score of 15 and Karnofsky performance score of 90. IV meropenem chemotherapy was extended to 6 weeks which was

given during three out-patient visits a day. He recovered completely.

## DISCUSSION

SDE is a condition with high morbidity and mortality rates (7-30%). Most common etiological factors are untreated meningitis in children and rhino-otologic infections in adults. In all age groups, SDE may develop after open or penetrating head injury, intracranial surgery, chronic systemic infections mostly from intra-abdominal abscess, open wounds, lung infections, genitourinary tract infections, and immunodeficiency of primary or secondary with diabetes mellitus, tumors or associated chemotherapy[4,15,19,20]. Although our patient had a history of minor head injury two months prior to onset of his headaches, he did not have other predisposing factors for SDE. Nevertheless he had two predisposing factors for development of chronic subdural hematoma, namely chronic use of ethyl alcohol and acetyl salicylic acid.

The previous series underlined the fact that at first admission of SDE patients, the most common symptoms are fever (77-96%), headache (32-78%), vomiting (8-23%), frontal skin and periorbital edema (31%), unconsciousness (50-62%), meningeal irritation (60-74%), focal neurological deficits (35-48%) and the seizures (29-39%)[13,15]. In our patient, the clinical picture 2 weeks after the initial burr-hole drainage surgery included a febrile attack, vomiting, frontal skin edema and periorbital edema and drowsiness but there was no meningeal irritation or seizure before craniotomy.

The most causative microorganism in SDE is Streptococcus milleri which stays in oral flora normally (17%). Following pathogens are B. hemolytic streptococcus, Staphylococcus aureus and its other spp., Haemophilus influenzae, Proteus mirabilus. Very rarely seen pathogens are Escherichia coli, Pseudomonas aeuroginosa, Enterococcus faecalis and Klebsiella pneumoniae which often appeared as single case or small series in the medical literature. However, in a significant number of patients (20-30%), causative agent for SDE may not be isolated at all[3,4,6,7,9,10,13,15,19,21].

From a small number of SDE cases diagnosed to have Klebsiella species as the causative agent, two facts emerged. The agent often reached the subdural space via hematogenous dissemination of from other foci such as liver abscess or pulmonary abscess. The passage and colonization was facilitated by immune-compromised condition of diabetes mellitus, cancer therapy etc.[4,7,10]. Although our patient had a long history of ethyl alcohol consumption, his liver function parameters were all normal before the infection appeared clinically. Moreover, chest CT and abdominal US in search of a source failed to detect any abscess formation. So, all these leave only one possibility : iatrogenic contamination. Although closed drainage systems both for ventricular and subdural drainage have long been safely used without any contamination per se it is almost impossible to disprove a contamination from scalp exit wound.

Studies have shown that the early diagnosis, urgent surgery with long-term intravenous administration of appropriate antimicrobial drugs have decreased morbidity and mortality of SDE from 30% down to 7%[2,10,15]. In this context, surgery plays the key role although there are several authors who dared to suggest the use of empirical antibiotics as the sole treatment[11,14]. For those undergoing surgery, there is little controversy about the choice of surgical approach. Craniotomy that enables total removal of infected subdural membranes is definitely the procedure of choice. It is superior to burr-hole drainage although burr-hole drainage is superior to blind antimicrobial treatment. Craniotomy reduces the re-operation risk because of large operative field however the bone flap can be lost because of bone infections[2,15]. We suggest the use of temporalis muscle pedicled craniotomy flaps to avoid or decrease the likelihood of postoperative osteomyelitis or better heal a preexisting osteomyelitis. In our patient, histopathology of the bone surrounding the burr holes revealed new bone formation with chronic inflammatory infiltration caused by foreign body-giant cell and macrophage suggest that despite the lack of macroscopic infection, the bone was also contaminated. This small detail in fact had a very serious impact on the extent of postoperative treatment. Because of histopathological documentation of bony inflammation, IV antibiotics were continued for 6 weeks instead of the initial well-being state obtained at 3 weeks postoperatively.

In patients with subdural empyema, the most important reasonable factor of the morbidity and mortality is cerebral vascular occlusions such as dural venous sinus thrombosis and/or arterial vasculitis. Septic thrombosis of the dural venous sinuses most frequently accompanies bacterial meningitis, brain abscess, subdural empyema or air sinus infections. The most causative microorganisms include Streptococcus pneumoniae and other streptococci, Staphylococcus aureus, thrombosis and Klebsiella species[18]. These thromboses could occur as a result of spread from primary of focus infection or from hematological dissemination such as septic thromboemboli. Also, spasm or inflammation of the arterial wall could be appeared by arterial invasion of infection[8,12,17]. Septic thrombosis and/or vasculitis of the cerebral vessels and dural venous sinuses are still life threatening conditions; and they should be carefully managed by using

antimicrobial drugs combined with anticoagulants. Although these occlusive phenomena could be successfully managed with anticoagulants such as heparin and low molecular weight heparin (LMWH) in non-operated patients, there has been still some complexity about using the LMWH before the neurosurgical procedures. Many investigations showed that there is no difference in the postoperative incidence of intracranial hemorrhage between the administration of LMWH in patients and placebo groups. These investigations also suggested that this prophylaxis protocol should be administered after 24 hour from all initial and subsequent craniotomies. However, it is well known that preoperative usage of anticoagulants could increase the risk of the intra-cerebral hemorrhage related to the surgery[5,16]. Our patient's frontal skin edema and periorbital edema made us to suspect from the dural venous sinus thrombosis especially cavernous sinus thrombosis; and to avoid from severe complications of this (such as hemiparesis, blindness, debility, etc.) we initiated fractionated heparin. Although this prophylaxis may have increased risk of the surgical re-bleeding, and caused the re-accumulation of subdural hematoma resulted in two times of re-exploration, we have still thought that it could reduce the morbidity and mortality risks originating from dural venous sinus thrombosis and/or arterial lumen occlusions.

## CONCLUSION

As microorganisms like Klebsiella pneumoniae may directly infect the subdural space with iatrogenic contamination, early diagnosis and urgent surgery with long-term intravenous administration of appropriate antimicrobial drugs could decrease the morbidity and mortality of SDE.

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