Cerebral and Subdural Abscess With Spatio-temporal Multiplicity 12 Years After Initial Craniotomy for Acute Subdural Hematoma

—Case Report—

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Abstract

A 34-year-old man presented with a case of subdural empyema and cerebral abscess that developed 12 years after initial neurosurgical intervention for a traffic accident in 1998. Under a diagnosis of acute subdural hematoma and cerebral contusion, several neurosurgical procedures were performed at another hospital, including hematoma removal by craniotomy, external decompression, duraplasty, and cranioplasty. The patient experienced an epileptic seizure, and was referred to our hospital in March 2010. Magnetic resonance imaging revealed a cerebral abscess extending to the subdural space just under the previous surgical field. Surgical intervention was refused and antimicrobial treatment was initiated, but proved ineffective. Surgical removal of artificial dura and cranium with subdural empyema, and resection of a cerebral abscess were performed on May 12, 2010. No organism was recovered from the surgical samples. Meropenem and vancomycin were selected as perioperative antimicrobial agents. No recurrence of infection has been observed. Postneurosurgical subdural empyema and cerebral abscess are recently emerging problems. Infections of neurosurgical sites containing implanted materials occur in 6% of cases, usually within several months of the surgery. Subdural empyema and cerebral abscess developing 12 years after neurosurgical interventions are extremely rare. The long-term clinical course suggests less pathogenic organisms as a cause of infection, and further investigations to develop appropriate antimicrobial selection and adequate duration of antimicrobial administration for these cases are needed.

Key words: subdural empyema, cerebral abscess, surgical site infection, post-neurosurgery, complication

Introduction

Subdural empyema and cerebral abscess are rapidly progressive conditions, and the rapid rate of progression results in mortality rates of up to 20%. Severe sequelae are reported in about 20% of survivors. Delays in hospitalization, focal neurological deficits on admission, impaired host immunity, uncontrolled diabetes, and Glasgow Coma Scale score less than 12 are reported to be associated with death and permanent neurological deficits.9–11,16) Bacterial cerebral abscess is most commonly the result of contiguous spread of infection from the otopharynx, middle ear, and paranasal sinuses.3) Head injury is another source of cerebral abscess. The prevalence of cerebral abscess after penetrating head injury or neurosurgical procedures ranges from 2% to 14%.2,5,11,16) Streptococcus species are the most common cause of pyogenic cerebral abscess because of extension from the nasopharynx and otopharynx. Anaerobic bacteria are reported to be another major cause of cerebral abscess, often as part of a polymicrobial infection.3,8) Postneurosurgical subdural empyema and cerebral abscess are recently emerging problems. Surgical site infection occurs in several percent of neurosurgical patients, and repeated surgery is needed for neurosurgical site infections after procedures with implanted materials including ventriculoperitoneal shunt, artificial dura, and artificial bone, which may lead to a poor prognosis for these patients.1,13–15) Neurosurgical site infections are sometimes fatal, and clinicians must take great care to prevent and treat these postoperative infectious complications. We present a case of subdural empyema and cerebral abscess that developed 12 years after initial neurosurgical interventions.
Case Report

A 34-year-old man experienced a traffic accident in 1998 at age 22 years. Under a diagnosis of acute subdural hematoma and cerebral contusion, hematoma removal by craniotomy was performed at another hospital. External decompression and duraplasty with artificial dura (Lyodura; B. Braun Melsungen AG, Melsungen, Germany) were conducted for postoperative intracerebral hemorrhage on the next day. The patient was discharged after cranioplasty with an autologous bone and a resin plate, with mild intellectual disturbance. The patient experienced an epileptic seizure, and was referred to another hospital in July 2007. Computed tomography (CT) revealed a low density lesion in the left parietal lobe (Fig. 1A), and the patient was treated with only antiepileptic medication.

The patient experienced another epileptic seizure on March 10, 2010, and was treated at another hospital for a minor palpebra injury. Five days after the second epileptic episode, the patient was referred to our hospital for repeated epileptic seizure. CT revealed a massive low density area in the left frontal lobe, and the lesion in the subdural space had grown compared to 3 years earlier (Fig. 1B). Magnetic resonance (MR) imaging demonstrated a heterogeneously enhanced lesion approximately 2 cm in diameter just under the previous surgical field, which extended continuously into the subdural space and was enhanced with gadolinium (Fig. 2 upper row). Body temperature was 38.5°C, and moderate mental retardation caused by previous head injury was observed. Aerobic and anaerobic blood culture, and cerebrospinal fluid examination did not reveal any causative organism, so meropenem and vancomycin were selected for treatment considering possible causative organisms such as anaerobic, Gram-positive, and Gram-negative species. Surgical intervention was refused, and the patient was discharged after a 2-week antimicrobial treatment with meropenem and vancomycin.

Antimicrobial agents were replaced with oral garenoxacin and linezolid, which were revealed as ineffective by CT on April 27, 2010. CT showed progressive massive cerebral edema on April 27, 2010, and serial MR imaging demonstrated persistent ring-enhanced lesion in the left frontal lobe (Figs. 1C and 2 lower row). The patient was again referred to our hospital because of repeated epileptic seizure, and surgical intervention was conducted on May 12, 2010. The time course from the initial neurosurgical procedure to surgical intervention for the cerebral abscess is presented in Fig. 3. Using the previous skin incision, the hypertrophic autologous bone and resin plate were exposed. The cranium-fixation plate was covered by hypertrophic autologous bone. Piece by piece removal of the bone flap was performed using a craniotome and Luer rongeur. After removing the cranial bone flap, yellowish artificial dura (Lyodura) was exposed, which was covered by granulation tissue and milky-brown pus (Fig. 4). Compressing the artificial dura caused massive exudation of pus from beneath the dura. The dura was partially firmly adhered to the brain at the site of cerebral edema, and was surrounded by massive pus on the cerebral cortex. Careful dissection was performed to prevent injury to the cortical surface. An intracerebral abscess containing much pus...
was also removed using a minimum corticotomy (Fig. 4). The site was irrigated with saline containing gentamycin, and the wound was closed temporarily using artificial dura (Gore-Tex®; W. L. Gore and Associates, Newark, Delaware, USA). No organism was recovered from the surgical samples, and meropenem and vancomycin were selected as perioperative antimicrobial agents, which were replaced with garenoxacin and erythromycin after discharge to target anaerobic species.

The specimen obtained at surgical intervention was 2.0 × 1.0 × 1.0 cm in size (Fig. 5 left). Histological examination found the nodular lesion corresponded to the abscess lesion, surrounded by fibrous tissue and brain tissue with reactive astrocytes, attached to the dural tissue (Fig. 5 center, right). During the clinical course, no systemic disease such as diabetes mellitus or other immunosuppressive disease was confirmed. After the surgical intervention, the edematous lesion was resolved on CT (Fig. 1D). Four months after resection of the cerebral abscess, cranioplasty was performed. Gore-Tex artificial dura was replaced with bioabsorbable artificial dura, and artificial bone made of hydroxyapatite was applied to the cranial defect.

Discussion

Generally, neurosurgical sites containing implanted materials are infected in 6% of cases, usually within several months after surgery.1,7,13–15) Subdural empyema and cerebral abscess developing as long as 12 years after neurosurgical interventions are extremely rare. A series of 31 cases of postneurosurgical cerebral abscess demonstrated an interval from neurosurgical procedures to detection of cerebral abscess between 8 days and 35 days (mean 20 days).16) In the present case, progressive cerebral edema on CT, resistance against antimicrobial treatment demonstrated on serial MR imaging, and refractory epileptic seizures were the major reasons to select surgical intervention. The previous scalp injury was on the palpebra, and we could not confirm any causative event of infection other than the surgical interventions performed 12 years ago.
ago. Moreover, intraoperative findings demonstrated that artificial dura was suspended in pus, and the pus extended into the cerebral abscess. CT revealed a refractory cerebral abscess during a 3-year period, and the operative findings demonstrated that the infection occurred just at the site of surgical intervention, so we considered that the infection was related to the previous surgical intervention.

Vancomycin with third-generation cephalosporins (cefepime or ceftazidime) or meropenem are recommended as empirical treatments for ventriculitis or meningitis caused by neurosurgical procedures. Third- or fourth-generation cephalosporins, metronidazole, and vancomycin are a typical regimen for cerebral abscess, to cover Gram-positive cocci in 19.4% of cases, and mixed infections in 29.0% of cases. Causative organisms were not identified in 19.4%. However, no recommendation of empirical treatment was made for cerebral abscess after neurosurgical procedures.

The present case did not meet the criteria of surgical site infection proposed by the Centers for Disease Control and Prevention (CDC). The CDC criteria of surgical site infection are infections which occur within one year after surgical intervention with implanted materials. In this case, surgical site infection was diagnosed 12 years after the initial surgery. Moreover, it was not clear that the surgical intervention was the cause of this intracranial infection. In this case, the causative organisms were not identified, so vancomycin with meropenem were selected for antimicrobial treatment using the referring guidelines for postneurosurgical meningitis, which were ineffective without removal of the foreign body and cerebral abscess.

Removal of foreign bodies and the abscess followed by appropriate antimicrobial administration is essential in covering the site of surgical intervention, so we considered that the infection occurred just at the site of surgical intervention, and further careful follow up will be needed.

References


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