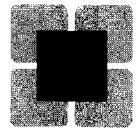

Omentum graft for intractable subdural empyema

GENE H. BARNETT, MD AND HARRY K. MOON, MD

■ A subdural empyema developed in a young man after craniotomy for evacuation of a hematoma in a sylvian fissure arachnoid cyst and the subdural space. Despite prolonged systemic and subdural antibiotic administration and a debridement of the subdural space, infection persisted, as evidenced by persistent fever, an elevated white blood cell count, and an extremely low cerebrospinal fluid glucose level. The infection was cured after a second debridement operation where microsurgically revascularized free omentum was used to obliterate the cyst and to cover the cerebral hemisphere in the craniotomy defect. The use of vascularized free omentum may prove useful in cases of refractory cranial wound infection and cerebrospinal fluid fistulas.

□ INDEX TERMS: CASE REPORTS; OMENTUM; SUBDURAL EMPYEMA □ CLEVE CLIN J MED 1989; 56:311–315

INTRACRANIAL subdural empyemas (SDEs), which account for 15% to 20% of intracranial abscesses, most commonly result from parameningeal infections or trauma.[1] SDE may also arise as a complication of craniotomy.[2] Antibiotic therapy and early diagnosis by computed tomography (CT)[3] have substantially improved the prognosis of SDE,[4] but successful therapy often requires surgical debridement in conjunction with pharmacotherapy.[5–7] Despite aggressive surgical and antibiotic treatment, SDE frequently results in permanent neurologic deficits or death.[8]

We report a case of subdural empyema that arose as a complication of craniotomy, in which conventional therapy failed to resolve the infection. Transposing microsurgically revascularized omentum to the empyema cavity obliterated the dead space of the cyst, stopped a persistent cerebrospinal fluid (CSF) leak, and provided a barrier between the skin and the surface of the brain. These factors led to the rapid cure of the infection.

From the Departments of Neurological Surgery (G.H.B.) and Plastic and Reconstructive Surgery (H.K.M.), The Cleveland Clinic Foundation. Submitted Jan 1988; accepted March 1988.

Address reprint requests to G.H.B., Department of Neurosurgery, The Cleveland Clinic Foundation, One Clinic Center, 9500 Euclid Avenue, Cleveland, Ohio 44195.

CASE REPORT

A 16-year-old boy was in good health until six months prior to hospitalization, when a generalized tonic clonic seizure disorder developed. A CT scan of his head at the time showed a large arachnoid cyst occupying the left sylvian fissure and a small left temporal lobe. His seizures were controlled with phenobarbital and he did well until one month prior to hospital admission when he had a severe headache associated with nausea and vomiting. His headache resolved after three days; however, it abruptly recurred two weeks later and persisted despite use of one gram of acetaminophen every three hours. Magnetic resonance (MR) imaging showed regions of high signal on T1- and T2-weighted images consistent with hematoma in the subdural space and cyst cavity.

A left frontotemporoparietal craniotomy was performed for evacuation of the hematoma and marsupialization of the arachnoid cyst. At the time of surgery, a bridging vein across the cyst was found to be a source of

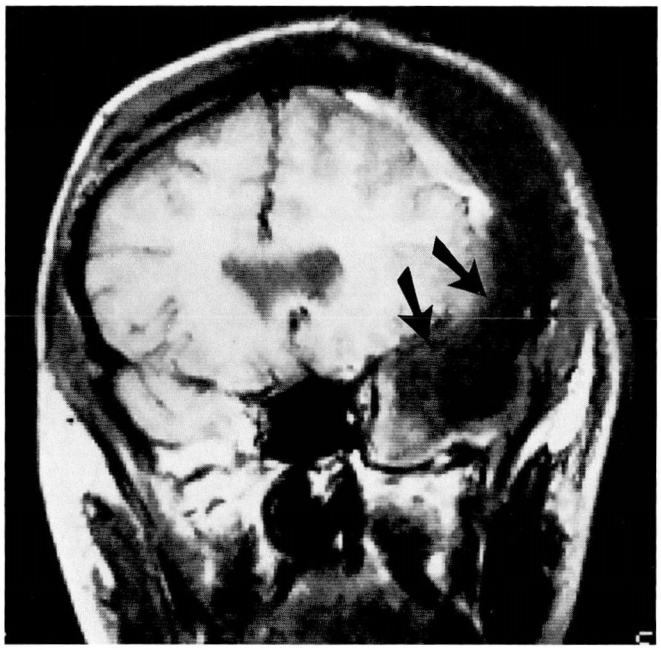

FIGURE 1. Coronal MR image (1.5 Tesla, TR=600, TE=32 ms) obtained before omental transposition, showing wound empyema (arrows) involving the sylvian arachnoid cyst.

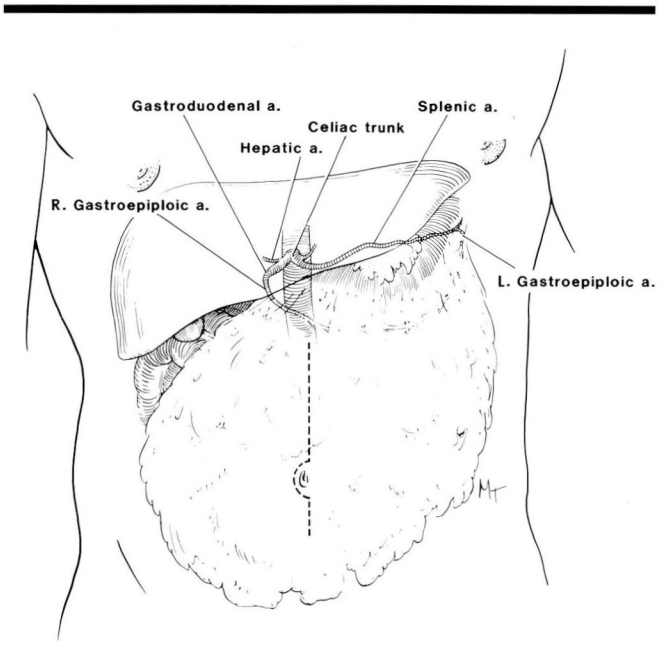

FIGURE 2. Abdominal incision (dashed line) and vascular supply of greater omentum.

bleeding and this was coagulated. His immediate postoperative course was unremarkable; on the second postoperative day, he complained of severe headache and his wound was noted to be bulging. A CT scan demonstrated a subgaleal hematoma; however, the intracranial compartment appeared benign. His headache worsened and he became hemiparetic and mute. He was taken to the operating room where hematomas were found in the subgaleal, extradural, and subdural spaces. The stump of the superficial temporal artery was bleeding and was presumed to be the source of the hematomas, which had dissected through these compartments. The clots were evacuated and hemostasis obtained.

Upon awakening the patient recovered limb and speech function. Twelve hours after surgery, he again became hemiparetic and mute. His CT scan at that time showed increased midline shift due to enlargement of the cyst cavity, but no recurrence of the hematoma. A catheter was placed in the subdural space and his hemiparesis immediately improved; however, he remained mute. CSF obtained at the time of catheter placement had many gram-positive cocci and gram-negative bacilli (which were later identified as *Streptococcus* and *Escherichia coli*) and glucose was undetectable. Vancomycin, one gram every eight hours, cefotaxime (Claforan), two grams every four hours, and tobramycin, 100 mg every eight hours, were given systemically. Four milligrams of gentamicin and 100 milligrams of vancomycin were given in the subdural space daily. Despite four days of this treatment, CSF glucose remained undetectable and there were 8,000 white blood cells/mm^3 in his CSF.

He was taken back to surgery, where his bone flap was removed and the intracranial cavity debrided. Large amounts of pus were encountered in the subgaleal and extradural space. The dural flap appeared grossly infected and both this and the free bone flap were removed. The cyst and subdural hematoma bed were filled with soft, yellow, cheesy material which, on culture, grew the same bacteria as his CSF had done earlier. A catheter was placed directly in the arachnoid cyst for CSF drainage and instillation of antibiotics. A lyophilized dural graft was used between the brain and the skin flap as a barrier to CSF leakage. Necrotic regions of his scalp flap and temporalis muscle were debrided as well.

Intrathecal and systemic antibiotics were continued; however, CSF glucose levels remained very low (<20 mg/dL). He remained febrile and had a persistent CSF and systemic glucose leukocytosis. Attempts to clamp his drainage catheter resulted in leakage of large volumes of cloudy CSF from the wound. MR imaging showed a large subdural empyema with extradural and subgaleal extension (*Figure 1*).

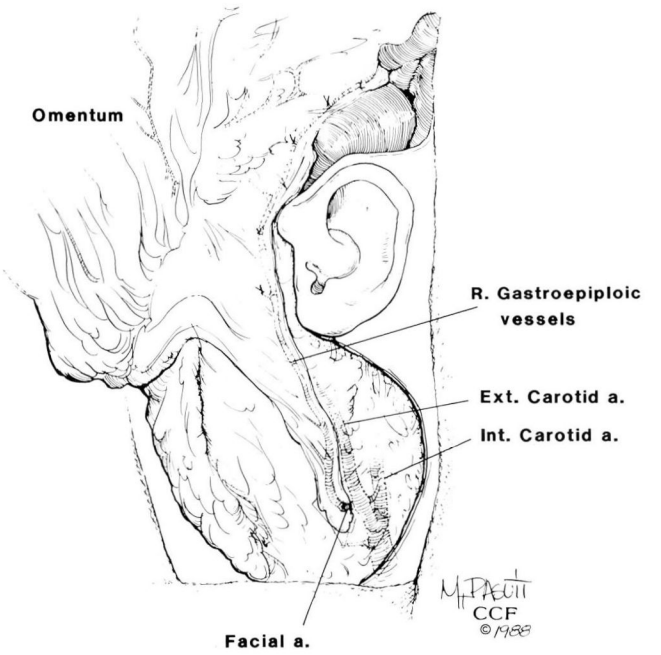

FIGURE 3. Free omentum with vascular pedicle microsurgically anastomosed to the left facial artery and vein.

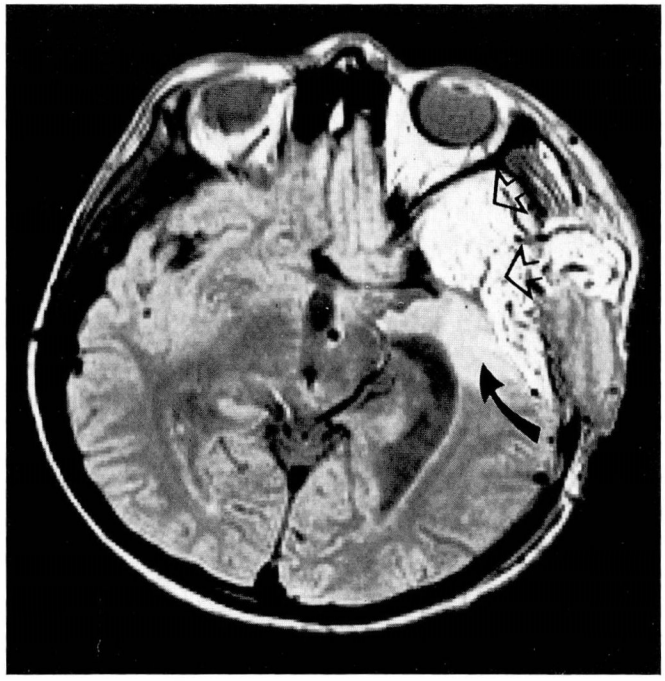

FIGURE 4. Transverse MR image of the head (1.5 Tesla, TR=2010, TE=32 ms) after subdural omental transposition. Note obliteration of dead space (open arrows). Areas of white matter with increased signal (solid arrow) are thought to represent zones of venous infarction due to the patient's empyema.

He was returned to surgery and the craniotomy reopened. The dural graft was removed and the intracranial contents again debrided. A laparotomy was performed and a free omental flap with a vascular pedicle was obtained (*Figure 2*). His craniotomy incision was extended caudally and the facial artery and vein were exposed. An anastomosis was then created microsurgically between the pedicle of the omental graft and these vessels (*Figure 3*). The omentum was laid in the cyst cavity and over the cerebral hemisphere. It was tacked in place using 3/0 chromic suture. The skin was then closed using a single layer of vertical mattress sutures. A ventricular catheter was placed in the right frontal horn of the lateral ventricle and the subdural catheter removed.

He was awakened and taken to the Neurosurgical Intensive Care Unit where his antibiotic therapy was continued and his CSF drained at atmospheric pressure. Resolution of his fever, reduction in his leukocytosis, and return of CSF glucose to >50 mg/dL occurred within three days. His ventricular catheter was drained at pressures of 5 mmHg and then 10 mmHg. On the sixth postoperative day, the catheter was clamped and subsequently removed. MR imaging at this time showed obliteration of the subdural and cyst space by the omental flap and areas of increased signal in the white matter of the inferior frontal gyrus and the temporal pole that were believed to be areas of venous infarction (*Figure 4*). He has remained free of fever and has required no further CSF drainage.

DISCUSSION

Before the availability of antibiotics, SDE was associated with a mortality of nearly 90%. Aggressive antibiotic therapy alone has been advocated by some authors as sufficient therapy for SDE[5–7]; however, it is generally held that early surgical drainage and debridement used in conjunction with antibiotic therapy is the best treatment.[1,9] Although combined therapy may result in mortality rates as low as 5% or 10%,[4,9] Arseni et al[8] have found mortality remaining as high as 25%. The cause of death in these patients often is brain infarction from cerebrovenous thrombophlebitis and inability to control the infection.

Omental transpositions have proven to be quite effective in the management of mediastinal sepsis after

median sternotomy[10–12] and infection of hip replacements.[13] Although omentum can scavenge bacteria within the peritoneal cavity, it has been speculated that much of the infection-controlling property of omental graft in these settings is due to its obliteration of dead space, generous blood supply, and ability to adhere to and granulate surfaces to which it is applied.[12] Regardless, the infection bed must be debrided and free of gross purulence as grafts may not survive or "take" when placed into a bed of gross purulence.

The use of omental transpositions in neurosurgical practice is not without precedent. Levander and Asard,[14,15] Wennerstrand and Levander,[16] and Andersson et al[17] have reported on the abilities of the greater omentum to absorb CSF, speculating upon its use in the treatment of hydrocephalus. The use of omental grafts to repair large defects of the skull was first described by Browning et al[18] in 1979 and since then, omental transpositions have been used in cranial repairs for electrical trauma,[19,20] mechanical trauma,[21] thermal burns,[22] and tumor surgery.[23,24] The use of omental grafts to provide a collateral blood supply to regions of cerebral ischemia has been studied extensively in animals,[25–29] successfully used to treat Moya Moya disease,[30] and used as a treatment for cerebral ischemia in humans.[31–33] To our knowledge, ours is the first report of use of omentum in the treatment of subdural empyema and CSF leakage that was refractory to antibiotic and conventional surgical techniques.

It may be argued that the use of a lyophilized dura graft may have perpetuated the infection in our patient. The decision to use this was based on the need for a CSF barrier as the required skin debridement would not allow a watertight skin closure. We believe that the omental transposition was successful in resolving the infection because of its highly vascular nature, obliteration of dead space, and arrest of CSF leakage.

The use of a revascularized free omental flap may pose some problems for our patient in the future. First, a cosmetic cranioplasty to repair his bony defect may be made more difficult by the presence of omentum and its vascularized pedicle in the craniotomy defect. Also, a substantial weight gain could, theoretically, produce enlargement of the omental graft that could act as a mass lesion. Regardless, use of a revascularized free omental graft proved lifesaving in this case and should be considered in other cases of intractable subdural empyemas or infections of cystic intracranial cavities.

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