Repair of middle fossa cerebrospinal fluid leaks using a novel combination of materials: technical note

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Object. Methods for repairing middle fossa CSF (MFCSF) leaks have varied and yielded mixed results. The objective of this study was to evaluate the safety and durability of the authors’ repair technique using a novel combination of 3 synthetic materials.

Methods. The authors performed a retrospective case review of patients treated for CSF leaks between January 2009 and September 2011. Eight patients were found to have undergone middle fossa craniotomies for CSF leaks. Inclusion criteria for the study included age greater than 18 years, neuroimaging-documented temporal bone defect, and symptoms consistent with CSF leaks or gross CSF otorrhea. Seven patients, 3 men and 4 women, met the inclusion criteria, and their charts were reviewed. Hydroxyapatite cement, collagen-based dural substitute matrix, and polyethylene glycol hydrogel sealant were used in all patients for the repair.

Results. In all patients the MFCSF leaks were successfully repaired. Initial presenting symptoms included CSF otorrhea in 4 patients (57.1%), hearing loss in 3 (42.9%), and CSF rhinorrhea in 1 (14.3%). The mean follow-up duration was 12 months (range 5–33 months). In 1 patient an epidural hematoma developed at the operative site on postoperative Day 2, and in another patient a superficial wound dehiscence occurred on postoperative Day 48. During the follow-up period, the authors found no evidence of wound infections, neurovascular damage, or CSF leakage requiring reoperation.

Conclusions. The middle fossa approach involving a combination of hydroxyapatite cement, collagen-based dural substitute matrix, and polyethylene glycol hydrogel sealant is a safe, effective method for repairing MFCSF leaks. The combination of synthetic materials provides an alternative to existing materials for skull base surgeons.

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Key Words • middle fossa • otorrhea • cerebrospinal fluid leak • repair

Cerebrospinal fluid leaks are a significant concern for surgeons who treat skull base pathology. Cerebrospinal fluid leaks from the middle fossa can be especially challenging to evaluate and treat. Uncomplicated, minuscule amounts of CSF leakage may only cause discomfort and not require surgical intervention. However, symptoms such as hearing loss or recurrent meningitis mandate surgical exploration and repair of cranial defects. Appropriate management of CSF leaks requires a high index of suspicion and proper utilization and interpretation of diagnostic tools.

Middle fossa CSF leaks can be classified as acquired, congenital, or spontaneous depending on the etiology. Diagnosis of leakage can be challenging and depends on the type and presentation of the leak. Regardless of the origin, the common feature of MFCSF leaks is the presence of a defect in both the middle fossa floor and the overlying temporal lobe dura mater.

Repairs of MFCSF leaks have posed challenges to skull base surgeons for years. In 1903, Canfield first described the use of xenograft (canine) dura to repair a postoperative CSF leak with good results, and Dandy later established the use of autologous tissue for the repair of CSF otorrhea. Many authors have advocated the use of fibrin glue, bone cement, and autologous tissue, alone or in multiple combinations, to guarantee effective closure. No consensus exists on the ideal procedure and repair technique, but there is general agreement that multilayer closure significantly reduces the recurrence rate.

Current repair recommendations are largely based on surgeons’ personal experiences because there are few large studies for this uncommon condition. Our technique for repairing MFCSF leaks addresses the temporal bone defect by remodeling it with HAC, the dural defect by synthetic repair with collagen-based dural substitute matrix, and closure augmentation with PEG hydrogel sealant. While the strategy of bony and dural repair practiced at our institution is not unique, to our knowledge this is the first reported series of this combination of materials. It is our goal to provide an additional option for the surgical repair of MFCSF leaks.

Abbreviations used in this paper: EAC = external auditory canal; GSPN = greater superficial petrosal nerve; HAC = hydroxyapatite cement; MFCSF = middle fossa CSF; OCR = ossicular chain reconstruction; PEG = polyethylene glycol.
Methods

We performed a retrospective case review of all CSF leaks in patients in whom the middle fossa approach was used by the operating neurosurgeon (B.G.) between January 2009 and September 2011. Inclusion criteria included age greater than 18 years, neuroimaging-documented temporal bone defect, and symptoms consistent with CSF leaks or gross CSF otorrhea after myringotomy.

Surgical Technique

After proving informed consent, the patient is brought to the operating room, and general endotracheal anesthesia is induced. The patient is placed in a lateral decubitus position, and a lumbar drain is inserted under sterile conditions. The patient is rotated into the supine position, and 3-point Mayfield head fixation is applied. The patient’s head is then rotated toward the opposite shoulder and extended to position the temporal squama parallel with the floor. The facial nerve monitor leads are appropriately connected to the monitoring system. A standard reverse U or reverse question mark incision is planned for the middle fossa approach. The scalp is incised and standard subgaleal dissection is performed. The temporalis muscle is sharply incised and bluntly dissected from the calvaria. The zygomatic root is identified, and a bur hole is placed immediately above this. The second bur hole is placed slightly posterior to the EAC. A rectangular craniotomy is made with the superior border slightly superior to the squamosal suture, the rostral border two-thirds anterior to the EAC, and the caudal border one-third posterior to the EAC. To facilitate extradural dissection, the inferior edge of the craniotomy is drilled flush to the middle fossa floor. Lumbar drainage is begun once the craniotomy flap is removed.

Extradural dissection along the floor of the middle fossa is performed using the facial nerve probe under magnification of the operating microscope. The middle meningeal artery is identified exiting the foramen spinosum and is coagulated and divided. This maneuver facilitates mobilization of the temporal dura and eliminates bleeding from the artery during dissection. Dural elevation is performed in a posterior-anterior direction, starting along the petrous ridge. Care is taken not to elevate the dura in a lateral-medial direction to avoid lifting the GSPN out of the major petrosal groove. Preventing unnecessary traction on the nerve and geniculate ganglion decreases the incidence of facial nerve damage. Anteriorly, the dissection is carried to the mandibular division of the trigeminal nerve. Medially, dural elevation is continued beyond the “false” petrous ridge, which is formed by the upper edge of the superior petrosal sinus groove, until the true petrous ridge is reached. Correct identification of the true petrous ridge allows for proper placement of retractors to elevate the temporal lobe, fully exposing the middle fossa floor. If our neuroimaging indicates that the defect is well defined, then our middle fossa floor exposure is carried out only until the defect is fully identified; thus, obligatory exposure of the petrous ridge is not necessary in all cases of MFCSF leak repair. This is done only when the bony defect is ill defined on preoperative imaging.

Following the exposure, the tegmen and temporal dura are inspected for defects. Dural defects, wherever possible, are primarily repaired with running 4-0 braided monofilament suture (Nurolon, Ethicon Inc.). We then place an appropriately sized piece the dural substitute matrix (DURAFORM, Codman & Shurtleff, Inc.) an onlay graft. For defects that cannot be primarily reapproximated (which is the majority of defects both in our series and in the literature), an inlay graft is placed subdurally through the defect and a second piece is positioned as an onlay. Finally, the dural reconstruction is augmented with PEG hydrogel sealant (DuraSeal, Confluent Surgical Inc.) applied around the edges of the onlay graft (Fig. 1). Hydroxyapatite cement with or without a split-thickness cranial bone graft is fashioned to cover the entire extent of the bony defect along the middle fossa floor. The craniotomy flap is replaced and fixed with titanium miniplates. The temporalis muscle is reapproximated with 20 Vicryl suture as is the subsequent galeal layer. The skin is reapproximated with staples.

Postoperatively, all patients are admitted to the intensive care unit with orders for lumbar drainage not to exceed 10 ml/hour. The patients remain in intensive care until the lumbar drain is removed, typically after 3–5 days. Subsequently, the patient is transferred to a regular hospital bed and monitored for otorrhea, rhinorrhea, or wound leakage for an additional 1 or 2 days before being discharged from the hospital.

Results

Patient Population

The demographic information of patients included in this investigation is summarized in Table 1. A total of 8 patients underwent operative repair of MFCSF leaks. One patient younger than age 18 years was excluded. No conservative therapies were attempted at our institution before surgical management. The sex was evenly distributed with 3 men and 4 women. Patients ranged in age from 22 to 81 years (mean 53 years). In 5 patients (71.4%) MFCSF leakage was acquired, and in 2 (28.6%) it was spontaneous. The most common presenting symptoms were otorrhea in 4 patients (57.1%) and hearing loss in 3 (48.9%). Only 1 patient (14.3%) exhibited CSF rhinorrhea. Six patients (85.7%) had defects of the tegmen tympani and 1 patient (14.3%) had a defect of the sphenoid bone. The mean bony defect was 6 mm (range 4–7 mm) on CT imaging. The size of the bony defects was not recorded in the operative notes. Two patients (28.6%) had a history of chronic otitis media and 5 patients (71.4%) had a history of previous otological surgical procedures.

Surgical Data

All patients underwent a middle fossa approach after placement of a lumbar drain at the start of the operation. Postoperatively, lumbar drainage was continued for 3–5 days. The mean duration of drainage was 4.2 days, and the mean volume of CSF drained was 1052 ml (range 680–1658 ml). The mean operative time was 202 minutes (range 175–223 minutes), and the mean blood loss was 370 ml (range 50–1200 ml).

In all repair procedures, we used the combination of
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HAC, collagen-based dural substitute matrix, and PEG hydrogel sealant. Other materials used to augment the closure included split-thickness bone grafts and temporalis fascia and muscle (Table 2). Split-thickness bone grafts were used in addition to HAC to repair the temporal bone defect in 4 cases (57.1%). Dural substitute matrix was used for dural repair and was placed as either an onlay (2 patients [28.5%]) or an inlay/onlay (5 patients [71.4%]). The addition of temporalis muscle and fascia for dural defect repair was used in 4 cases (57.1%). Encephaloceles were identified and repaired in 5 (71.4%) of the 7 cases.

Postoperative Results and Complications

The mean follow-up duration was 12 months (range 5–33 months), and all patients were free of evidence of MFCSF leak, wound infections, or signs of neurovascular deficits at final follow-up appointment. Hearing loss improved in only 1 patient with preoperative hearing loss, whereas in the other 2 patients hearing status remained unchanged.

One patient returned to the hospital on postoperative Day 8 with an epidural hematoma requiring surgical evacuation. Intraoperatively, the repair site was inspected and showed no signs of CSF leakage. This patient was discharged to home without further complications. A second patient experienced a superficial wound dehiscence 48 days postoperatively without evidence of CSF fistula. The majority of our patients (71.4%) experienced no postoperative complications. The data are included in Table 2.

**Illustrative Case**

This 22-year-old man presented with right-sided con-
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Table 2: Middle fossa repairs*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Encephalocele</th>
<th>Repair</th>
<th>Lumbar Drain (days)</th>
<th>CSF (ml)</th>
<th>Complications</th>
<th>Follow-Up (mos)</th>
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<td>superficial wound dehiscence</td>
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<td>1310</td>
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</table>

* I = inlay; O = onlay; P = PEG hydrogel sealant; SB = split-thickness bone; TFM = temporalis fascia and muscle.

result from destructive pathology such as tumors, infection, trauma, and surgical procedures, the latter two being the most common. Congenital lesions such as patent Hyrtl fissure, Mondini dysplasia, and petromastoid canal fistulas occur much less frequently and typically occur in younger patients. At older ages (usually > 50 years) chronically enlarged arachnoid villi can cause pulsatile erosion of pneumatized segments of the temporal bone, leading to transmission of CSF through the defect. If there is no associated cause of a CSF leak, it is classified as spontaneous.

Cerebrospinal fluid leakage from the middle fossa can be challenging to diagnose and manage. Profuse fluid leakage from the ear canal allows for relatively straightforward diagnosis. Likewise, leaks following trauma or surgery may be anticipated, or even expected, allowing rapid establishment of the diagnosis and intervention. Conversely, if the MFCSSF leak is intermittent, subtle, or spontaneous in nature, it requires a higher index of suspicion to diagn-

**Discussion**

Middle fossa CSF leaks are uncommon pathological entities that can be classified as acquired, congenital, or spontaneous. Acquired MFCSSF leaks most commonly

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* I = inlay; O = onlay; P = PEG hydrogel sealant; SB = split-thickness bone; TFM = temporalis fascia and muscle.

Fig. 2. Coronal CT scan showing a defect of the right tegmen (arrow) with fluid in the middle ear.
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nose. Persistent middle ear fullness, with or without sensorineural, conductive, or mixed hearing loss; progressive drainage of clear fluid after myringotomy; and recurrent meningitis can all be signs of an MFCSF leak. Also, fluid in the mastoid air cells can be indicative of an MFCSF leak resulting from a tegmen mastoideum defect. Persistent drainage of fluid following myringotomy can be confused with postprocedural infection. However, if drainage does not resolve with adequate antibiotic treatment, a CSF leak should be suspected. Beta-2 transferrin is a confirmatory test that is highly sensitive and specific for CSF identification, as transferrin is only converted into the beta-2 isoform within the nervous system. Historical use of glucose strips has been subject to a high false-positive rate of 45%–75% due to mucus or blood contamination.

Prompt diagnosis and treatment require a basic understanding of the structural anatomy and pathology of the middle fossa. A defect in the middle cranial fossa bone and breach of the overlying temporal dura is required to develop an MFCSF leak. Politzer first referred to the “wanting” composition of the tegmen tympani of the temporal bone in 1896. Ahren and Thulin later studied the temporal bones in 96 cadavers and found approximately 20% to have some form of tegmen defect, a finding confirmed by Kapur and Bangash in 1986. Obviously, it is important to note that isolated tegmen defects, without a dural defect, do not lead to MFCSF leaks.

Several theories have been proposed regarding the pathogenesis of MFCSF leaks, especially those seen in the absence of trauma or congenital defects. Ommaya has proposed a theory that leaks result from erosion of the tegmen due to chronic variations in intracranial pressure. Others have implicated the enlargement of aberrant arachnoid granulations located over thin or pneumatized bone as a cause of adult-onset CSF otorrhea. In the absence of a precipitating event or an increased level of suspicion, MFCSF leaks often have a significant delay in diagnosis or are misdiagnosed altogether. Persistent MFCSF leaks can lead to considerable morbidities, including hearing loss, pneumocephalus, temporal lobe seizures, cerebral abscess, and meningitis. Secondary meningitis is well documented, with an incidence in the literature ranging from 4% to 50%. Thirty percent to 60% of postoperative meningitis and 10%–27% of post–traumatic brain injury meningitis can be directly attributed to CSF leaks. Those leaks originating from temporal bone pathological entities that persist more than 7 days have been reported to have a significantly increased risk of developing meningitis, compared with leaks repaired within 1 week.

Evidence supporting the use of prophylactic antibiotics in the prevention of meningitis has been conflicting at best. Findings from an early double-blinded study by Klustersky et al. failed to support the routine usage of prophylactic antibiotics in traumatic CSF leaks. However, the findings also failed to establish a detrimental effect. Similarly, in a recent meta-analysis Villalobos and colleagues found that prophylactic antibiotics did not decrease the risk of developing meningitis with basilar skull fractures. Conversely, Friedman et al. found that prophylactic antibiotics decreased the risk of developing meningitis by half, a finding further supported by the meta-analysis published by Brodie et al. Given the equivocal effect of prophylactic antibiotics, early surgical intervention has been strongly recommended. Repair of the temporal bone and dural defects and elimination of the CSF fistula are the ultimate goals.

Neuroimaging is critical in the evaluation of MFCSF leaks. High-resolution, thin-cut CT scans obtained through the suspected temporal bone can be extremely valuable in localizing a bony defect and thus allowing for accurate surgical planning. Computed tomography cisternography, with or without injection of radioactive isotopes, may be an option if the bony defect is not evident on CT scans; however, meaningful results are limited to those obtained in patients with active CSF fistulas, and the modality has been shown to have a significant false-negative rate with low-volume or intermittent leaks. The decision to undertake cisternography should also be made with consideration of the increased morbidity associated with lumbar puncture and contrast reactions. Computed tomography cisternograms were acquired in 5 of the 7 patients in the present study, and in all cases the studies were deemed necessary when initial CT scans did not definitively define the full extent of the temporal bone defect. All defects were clearly delineated on the CT cisternogram. Magnetic resonance imaging has proven useful in assessing the presence of dural defects and concomitant encephaloceles. Both T1- and T2-weighted MRI sequences are useful in evaluating the presence and content of herniated cerebral tissue, but their usefulness in delineating the bony defects has been shown to be limited. In a retrospective review of 8 cases of MFCSF leakage, Pappas et al. did not use MRI to identify and correct the site of the fistula. Likewise, Lundy et al. used MRI in 2 of 19 consecutive cases and found the information acquired to be equivocal. In our patients, MRI scans were obtained in 4 of the 7 patients we treated. Scans correlated with the intraoperative presence or absence of an encephalocele in 3 of 4 patients. The fourth had an encephalocele identified intraoperatively that was not visualized on MRI. Magnetic resonance imaging did not alter our surgical approach or repair method in any case.

Conservative Treatment

Conservative treatment, such as avoiding Valsalva maneuvers and straining, head of bed elevated to 30°, or placement of a lumbar drain, was not performed at our institution for MFCSF leak management. Savva et al. reviewed the cases of 92 patients with CSF leaks through the temporal bone. They found that in 82 patients the leak was caused by trauma—head injury in 29 and surgical procedure in 53. Conservative measure worked in 26 of 29 patients in whom CSF leaks were caused by a head injury. The remaining 3 patients required surgical intervention. Conversely, conservative measures worked in only 1 of the 53 patients in whom the leak resulted from surgical procedures. Of note, all patients with nontraumatic leaks in the Savva et al. series required surgical intervention.

In our series the 5 patients with acquired MFCSF leaks had previous surgical procedures. The other 2 patients had MFCSF leaks that were classified as sponta-
neous. Therefore, we chose not to attempt conservative treatments before surgical repair.

Choice of Approach

Following confirmation of an MFCSF leak, we advocate prompt surgical repair of the bony and dural defects to prevent associated morbidities. In the literature, the approaches and materials used to repair MFCSF leaks vary widely.

The middle fossa, transmastoid, and combined middle fossa/transmastoid approaches are the procedures most commonly described in the literature. Individual selection, however, is highly dependent not only on the advantages and disadvantages of each approach with regard to the specific location of the fistula but also the surgeon’s personal experience and comfort level with the approach in question (Table 3).

The transmastoid approach has been reported to be the least technically demanding route. It also confers the additional advantages of eliminating brain retraction and providing access to fistulas originating in the posterior fossa. The disadvantages include decreased exposure of anterior tegmen defects, risk of hearing loss, and difficulty in approaching large or multiple defects.

The middle fossa craniotomy provides a wide exposure of the entire tegmental plate, allowing identification and repair of large or multiple defects. Repair of tegmental defects can be performed without risk to the ossicular chain, thus preserving hearing. Increased exposure allows more accurate and secure placement of sealant materials, thereby decreasing the possibility of recurrence. However, many authors find the middle fossa craniotomy more technically demanding, and its disadvantages include an inability to approach defects of the posterior fossa.

In addition, multiple critical structures are potentially encountered during this approach. Mobilization of the temporal lobe and associated vascular structures such as the vein of Labbé can lead to postoperative seizures or venous infarction. Extradural dissection along the floor of the middle fossa also risks damage to the geniculate ganglion and GSPN.

We prefer the middle fossa approach for MFCSF leak repairs. Preoperative collaboration between the neurotologist and neurosurgeon is essential to ensure optimal surgical outcome. We advocate 3 steps to facilitate extradural dissection and protection of vital neurovascular structures. First, proper head positioning is critical; the head must be rotated 90° toward the opposite shoulder and extended so the temporal squama is parallel to the floor. This position allows gravity to aid in temporal lobe dissection. Second, lumbar drainage should be started only after the craniotomy is performed. Drainage of CSF prompts cerebral relaxation and allows for increased mobilization of temporal lobe dura from the middle fossa floor. Third, facial nerve monitoring is undertaken with the probe used to dissect the temporal dura from the floor of the middle fossa. This technique allows simultaneous dissection and stimulation of the GSPN and geniculate ganglion, conferring additional protection. We perform dural dissection in the traditional back-to-front manner to decrease the incidence of GSPN injury, although some authors have advocated safe dissection from front to back.

Repair Materials

Reconstruction in all cases involved a minimal combination of HAC, collagen-based dural substitute matrix, and PEG hydrogel sealant. In 4 cases we used additional native materials, including split-thickness bone graft, temporalis muscle, and fascia. Intraoperative inspection of the defect’s size and dimensions determined whether a split-thickness bone graft would be included.

Hydroxyapatite cement has been successfully used for the remodeling and repair of temporal bone defects by several authors, with Kveton et al. reporting a success rate greater than 97%. The cement is composed of tetracalcium and dicalcium phosphate salts reacting to form hydroxyapatite, the major component of skeletal bone.

There are numerous physiological properties that make HAC an ideal material for temporal bone repair. Its high biocompatibility limits postoperative inflammatory response and fibrosis, while its osteoconductive and osteointegrative properties allow for formation and intercalation of new bone within implants. Finally, the product remains malleable for approximately 20 minutes, allowing for easy remodeling into bony defects, and afterward it is resistant to CSF pulsation. Complications have been reported to range from 0% to 11%, with postoperative infections occurring in up to 5% of patients when cases of nasal sinus cavities are included. Kveton and Coelho reported infections in 2.8% of their 109 consecutive temporal bone repairs. Conductive hearing loss due to ossicular chain fixation has been reported up to 33%, with the majority of deficits occurring after transmastoid repairs. Thus, the application of HAC in patients with exposed ossicles must be carefully done. None of the patients in our series had an exposed ossicular chain, but

<table>
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<th>TABLE 3: Comparison of approaches</th>
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<td><strong>Approach</strong></td>
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<td>middle fossa</td>
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our recommendation for such cases would be to fashion a split-thickness bone graft large enough to cover the defect with or without careful placement of HAC.

In our series, temporal dura repairs involved a combination of dural substitute and PEG hydrogel sealant, with or without the addition of temporalis muscle and fascia. Whenever possible, our initial preference is primary repair with sutures; however, for defects that cannot be approximated, we prefer synthetic, autologous, or combination inlay grafts, supplemented by onlay grafts (inlay/onlay technique). In cases in which the dural defect cannot be repaired primarily because the dura is often thin and frayed and the edges cannot be cleanly reaproximated—because the location of the defect is too difficult to reach, or because the defect is too small to allow insertion of an inlay graft—a stand-alone onlay graft is placed. All repairs are subsequently augmented with PEG hydrogel sealant.

The safety and efficacy of dural reconstruction with synthetic collagen-based dural substitutes are well described. The structure of the synthetic collagen-based dural substitute presents a low-pressure surface for CSF absorption, promoting a graft–dura interface and chemical signaling for native fibroblasts providing mechanical scaffolding. Litvack et al. reported a CSF leakage rate of 6.7%, as well as a 4.2% postoperative infection rate, when using collagen-based substitutes in 425 consecutive patients.

In the literature the PEG hydrogel sealant is well described as an adjunct for the prevention of CSF leaks. Its strong adherence to tissue, biological compatibility, and absorbability make it an ideal choice for the treatment of CSF leaks. A study by Boogaarts and associates reported an intraoperative watertight seal rate of 100% and a CSF leak recurrence rate of 4.9% at 3 months in 41 patients who underwent various intracranial procedures. Later, in a large multicenter study, Cosgrove et al. reported a 1.8% postoperative CSF leak rate with PEG hydrogel augmentation of primary suture closure in 111 intracranial procedures; the incidence of infection was 7.2%.

Postoperative Care

We believe that the decreased CSF pulsation and pressure along the extent of the dural repair facilitates more effective healing, despite reports to the contrary. To that end, all patients in the present series underwent lumbar drainage of 10 ml/hour for 3–5 days and remained in intensive care for the duration. Taking into consideration the intraoperative benefits of CSF diversion, we recommend the routine use of lumbar drainage in all patients undergoing repair of an MFCSF leak. Following removal of the lumbar drain, patients are monitored for an additional 1–2 days for any signs of wound leakage, otorrhea, or rhinorrhea.

Complications

Common complications seen in MFCSF leak repairs include recurrence of CSF leaks, graft failure, and wound infections. Savva et al. reported a 14.3% recurrence rate following optimal repair in 92 consecutive patients. Gacek et al. reported a repair rate of 93% in 16 patients with 1 patient requiring an additional surgery for a recurrent leak. The repair materials were not specifically described in their series.

Comparison

The repair rate in our small series was 100%, which compares favorably with larger middle fossa craniotomy series. Stenzel et al. reported a repair success rate of 91% in a series of 11 patients in whom the reconstruction was conducted exclusively via the middle fossa craniotomy. One patient in their series required reoperation for a recurrent leak. However, the exact materials used for repairs were not described. In their series of 15 patients, Gubbels et al. described the use of bone grafts, fat grafts, HAC, and fascia. All repairs were done via a middle fossa craniotomy, and the repair success rate was 95%. One patient required an additional surgical procedure for a recurrent CSF leak. Saw et al. reported a repair rate of 93% in 41 patients with 1 patient requiring an additional surgery for a recurrent leak. The repair materials were not specifically described in their series.

Conclusions

There are many alternatives described in the literature for repairing CSF leaks. Many decades of advancements in materials and approaches have allowed neurologists and skull base neurosurgeons to vastly improve outcomes after MFCSF leak repair, but consensus regarding the best combination is lacking.

Based on our experience, we believe that the use of HAC, collagen-based dural substitute matrix, and PEG hydrogel sealant via the middle fossa approach, along with routine postoperative lumbar drainage, provides an effective combination for the repair of MFCSF leaks. In our small series, no patient required reoperation for a current CSF leak, which justifies continued application and further prospective evaluation of this novel technique.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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