Treatment Strategies for Lateral Sphenoid Sinus Recess Cerebrospinal Fluid Leaks

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Objective: To highlight concepts critical to achieving successful repair and avoiding intracranial complications in the treatment of cerebrospinal fluid (CSF) leaks from the lateral recess of the sphenoid sinus (LRS).

Design: Outcomes study.

Setting: Tertiary referral university hospital.

Patients: Eleven patients with LRS CSF leaks from June 2008 to June 2010.

Interventions: Endoscopic transpterygoid approach and multilayer repair of skull base defect in the LRS.

Main Outcome Measures: Recurrence, graft techniques, postoperative intracranial pressure (ICP), and use of ventriculoperitoneal (VP) shunt.

Results: Thirteen CSF leaks originating in the LRS were surgically repaired in 11 patients; 2 patients required bilateral leak repair. The endoscopic transpterygoid approach was used in 12 of 13 repairs. Eight patients had failed attempts at repair prior to presentation (4 endoscopic sphenoidotomies and 4 middle cranial fossa [MCF] approaches). One patient presented with a temporal lobe abscess following hydroxyapatite “obliteration” to seal off the LRS. This required a combined MCF/transpterygoid approach to drain the abscess, remove the encephalocele and hydroxyapatite, and seal the skull base defect. In 2 cases, the LRS was left patent owing to concerns of inadequate mucosal extirpation. The median duration of follow-up was 10.8 months (range, 2-29 months). One patient experienced a failure (2 months after repair), which was successfully sealed on the second attempt. Postoperatively, 5 patients required VP shunts, and 5 were maintained on acetazolamide for elevated ICP (average, 26.7 cm H2O in 8 patients; presumed elevated in 2 patients).

Conclusions: The current study demonstrated a 92% success rate using the endoscopic transpterygoid approach for LRS skull base defects providing support for routine use in the treatment algorithm. Poor outcomes were observed with previous surgical attempts to obstruct the LRS without repairing the skull base defect.


TREATMENT STRATEGIES FOR repairing cerebrospinal fluid (CSF) leaks have changed dramatically over the past 20 years. Where open craniotomies for CSF leaks and associated encephaloceles were once standard procedures for skull base defects, transnasal endoscopic techniques have become the gold standard for CSF leak repair with success rates higher than 90% in most series. Cerebrospinal fluid leaks and encephaloceles are typically categorized into 4 general classifications based on etiology: congenital, neoplastic, traumatic (including iatrogenic), or idiopathic etiologies. The idiopathic, also referred to as “spontaneous” etiology, has in large part been attributable to the presence of increased intracranial pressure (ICP) and is often considered a variant of benign (also termed idiopathic) intracranial hypertension. The presence of a skull base defect can increase the risk of pneumocephalus, meningitis, and brain abscess. With the exception of some CSF leaks resulting from blunt trauma, surgical repair is typically required, and the exposure of a defect is crucial to the success of the repair, especially in the frequent context of increased ICP. The location of the skull base defect dictates the endoscopic approach and techniques necessary for surgical treatment. When present in the sphenoid sinus near the midline, standard transseptal or transnasal endoscopic tech-
these approaches are often insufficient for satisfactory repair into the lateral recess of the sphenoid sinus (LRS), there is a widely pneumatized sphenoid sinus extending medially to the pterygoid muscle attachments using a drill or Kerrison punches and a 15° diamond burr. Adipose tissue is meticulously removed, and blunt dissection defines the internal maxillary artery for ligation and allows for preservation of the vidian nerve, infraorbital nerve, and sphenopalatine ganglia. The posterior wall of the pterygopalatine fossa is encountered, and the face of the pneumatized pterygoid process is removed superomedially to the pterygoid muscle attachments using a drill or Kerrison rongeurs. The goal on completion of the approach is not only total exposure of the encephalocele and associated skull base defect, but also complete mucosal extirpation from the LRS. Surgeon confidence in the ability to remove all the mucosa dictates obliteration vs maintaining sinus patency.

When the encephalocele is exposed, the tumor is ablated to the level of the skull base defect using either radiofrequency coagulation (Coblator; ArthroCare ENT) or trans-sphenoidal bipolar cauter.

Figure 1. Algorithm for the approach to lateral recess sphenoid (LRS) cerebrospinal fluid (CSF) leaks. *Bed rest, stool softeners, and avoidance of strenuous activity for a period of 5 days before and after lumbar drain placement.

Figure 2. (A) Lateral recess sphenoid sinus (LRS) transpterygoid approach. (B) Endoscopic transpterygoid approach.

Figure 3. An illustration of the surgical technique for repair of LRS CSF leaks.

METHODS

The skull base defect following ablation of the encephalocele dictates our reconstructive technique (Figure 3). Because 12 of 13 of the CSF leaks in our cases were spontaneous, multilayered closure with septal bone and tissue inlay grafts were used in nearly all of these defects. Inlay tissue grafts used in this series were either Alloderm (LifeCell), Duragen (Integra), or Surgisis (Cook Medical). This was followed by placement of an overlay tissue graft with or without a free fat graft with an additional layer of a pedicled septal flap in certain cases for definitive closure of the recess.
RESULTS

Thirteen CSF leaks in 11 patients originating in the LRS were surgically repaired. Given the location of the defects and guided by our algorithm for the approach (Figure 1), the endoscopic TPTG procedure was used in 12 CSF leaks. The demographic variables and history are included in Table 1. The median age at presentation was 56 years (range, 43-65 years). Eight of the 11 patients were female. The median BMI was 35.6 (range, 28.0-46.6). The median duration of follow-up was 10.8 months (range, 2-29 months), with a 92% success rate on first attempt. The presenting symptom in all patients included a component of CSF rhinorrhea. Three patients noted a history of meningitis prior to presentation. Computed tomographic scans of the sinuses confirmed the diagnosis suspected by history and physical examination in all patients; magnetic resonance imaging (MRI) was also useful in operative planning and diagnostic confirmation in 8 patients. Seven patients had empty sella syndrome on MRI.

All skull base defects were associated with an encephalocele and were approached using a TPTG technique as described. Only 3 patients had no prior surgical attempts to repair the CSF leak (patients 1, 5, and 11) (Table 2). Three patients (patients 6, 8, and 10) had undergone a total of 4 prior middle cranial fossa craniotomies that had failed to control the CSF leak. Three patients (2, 4, and 7) had received prior treatment with sphenoidotomy packing without success; these procedures did not achieve the endoscopic access to the lateral recess and directly address the defect. One such patient (patient 7) had her sphenoid sinus packed with hydroxyapatite; this led to a temporal lobe abscess, necessitating a middle cranial fossa and TPTG (above and below) approach for abscess drainage and defect repair. One patient was taken to surgery for repair of a possible ethmoid roof CSF leak prior to presentation at our institution, but the LRS defect had not been identified preoperatively on the MRI scans by the primary surgeon. Another patient had undergone 4 prior attempts at packing the LRS before referral for continued failure (Figure 4 and Figure 5). A TPTG approach was attempted in patient 9 at an outside institution, but no attempt was made to remove the encephalocele, repair the defect, or remove the mucosa of the recess. Instead, an in-
inferior turbinate mucosal graft was placed over the TPTG area in an attempt to seal the LRS, which resulted in a recurrent CSF leak within 24 hours.

We had 1 patient in our series (patient 6) with a recurrent CSF leak. Interestingly, this was a late failure at 2 months postoperatively and was in the context of a func-

### Table 1. Preoperative Data

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>BMI</th>
<th>Location</th>
<th>Etiology</th>
<th>Presenting Signs and Symptoms</th>
<th>Prior Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/65</td>
<td>30.0</td>
<td>L LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>None</td>
</tr>
<tr>
<td>2/F/56</td>
<td>28.7</td>
<td>R LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>Sphenoidotomy to pack sinus</td>
</tr>
<tr>
<td>3/F/46</td>
<td>31.9</td>
<td>R LRS, R cribriform</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>ESS with graft placed over posterior ethmoid, CSF leak not identified</td>
</tr>
<tr>
<td>4/F/60</td>
<td>31.9</td>
<td>R LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>Sphenoidotomy to pack sinus ×4</td>
</tr>
<tr>
<td>5/M/58</td>
<td>43.3</td>
<td>Bilateral LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>None</td>
</tr>
<tr>
<td>6/F/47</td>
<td>32.6</td>
<td>L LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>MCF; transpterygoid (late failure)</td>
</tr>
<tr>
<td>7/F/48</td>
<td>42.3</td>
<td>L LRS</td>
<td>Spontaneous</td>
<td>Meningitis, temporal lobe abscess</td>
<td>Sphenoidotomy with hydroxyapatite</td>
</tr>
<tr>
<td>8/F/58</td>
<td>38.3</td>
<td>Bilateral LRS</td>
<td>GSW and spontaneous</td>
<td>CSF leak</td>
<td>MCF × 2 sphenoidotomy/partial transpterygoid</td>
</tr>
<tr>
<td>9/M/43</td>
<td>46.6</td>
<td>Bilateral LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>None</td>
</tr>
<tr>
<td>10/M/62</td>
<td>38.6</td>
<td>Bilateral LRS</td>
<td>Spontaneous</td>
<td>CSF leak, meningitis</td>
<td>MCF 10 y prior; infected shunt</td>
</tr>
<tr>
<td>11/F/56</td>
<td>28.0</td>
<td>Bilateral LRS</td>
<td>Spontaneous</td>
<td>CSF leak</td>
<td>None</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CSF, cerebral spinal fluid; ESS, endoscopic sinus surgery; GSW, gunshot wound; L, left; LRS, lateral recess of the sphenoid sinus; MCF, middle cranial fossa; R, right.

### Table 2. Operative Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Defect 1</th>
<th>Operation</th>
<th>Defect 2</th>
<th>Operation</th>
<th>Acetazolamide</th>
<th>Shunt</th>
<th>Graft*</th>
<th>ESS</th>
<th>ICP, cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>L LRS</td>
<td>TPTG</td>
<td>None</td>
<td>ND</td>
<td>Yes</td>
<td>No</td>
<td>Duragen/bone</td>
<td>Yes</td>
<td>24</td>
</tr>
<tr>
<td>2</td>
<td>R LRS</td>
<td>TPTG</td>
<td>None</td>
<td>ND</td>
<td>Yes</td>
<td>No</td>
<td>Durarepair/bone</td>
<td>Yes</td>
<td>27</td>
</tr>
<tr>
<td>3</td>
<td>R LRS</td>
<td>TPTG</td>
<td>R ethmoid</td>
<td>Repair</td>
<td>No</td>
<td>Yes</td>
<td>Alloderm</td>
<td>Yes</td>
<td>ND</td>
</tr>
<tr>
<td>4</td>
<td>L LRS</td>
<td>TPTG</td>
<td>None</td>
<td>ND</td>
<td>Yes</td>
<td>No</td>
<td>Surgisis/bone</td>
<td>Yes</td>
<td>28</td>
</tr>
<tr>
<td>5</td>
<td>L LRS</td>
<td>TPTG</td>
<td>R LRS</td>
<td>TPTG/Sep Flap</td>
<td>No</td>
<td>Yes</td>
<td>Surgisis/bone/fat</td>
<td>Yes</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>L LRS</td>
<td>TPTG</td>
<td>None</td>
<td>ND</td>
<td>No</td>
<td>Yes</td>
<td>Surgisis/bone/fat</td>
<td>No</td>
<td>ND</td>
</tr>
<tr>
<td>7</td>
<td>L LRS</td>
<td>TPTG/MCF</td>
<td>None</td>
<td>ND</td>
<td>Yes</td>
<td>Yes</td>
<td>Surgisis</td>
<td>Yes</td>
<td>ND</td>
</tr>
<tr>
<td>8</td>
<td>L LRS</td>
<td>TPTG</td>
<td>R LRS</td>
<td>Not repaired</td>
<td>No</td>
<td>Yes</td>
<td>Surgisis/fat/septal flap</td>
<td>No</td>
<td>32</td>
</tr>
<tr>
<td>9</td>
<td>L LRS</td>
<td>TPTG</td>
<td>R LRS</td>
<td>Not repaired</td>
<td>Yes</td>
<td>No</td>
<td>Surgisis/bone/fat</td>
<td>Yes</td>
<td>35</td>
</tr>
<tr>
<td>10</td>
<td>L LRS</td>
<td>TPTG</td>
<td>R LRS</td>
<td>TPTG</td>
<td>No</td>
<td>Yes</td>
<td>Surgisis/bone/fat</td>
<td>No</td>
<td>32</td>
</tr>
<tr>
<td>11</td>
<td>R LRS</td>
<td>TPTG</td>
<td>L LRS</td>
<td>Not repaired</td>
<td>No</td>
<td>No</td>
<td>Surgisis/bone/fat</td>
<td>No</td>
<td>11</td>
</tr>
</tbody>
</table>

Abbreviations: Ecele, encephalocele; ESS, empty sella syndrome; ICP, intracranial pressure; L, left; LRS, lateral recess of the sphenoid sinus; MCF, middle cranial fossa; ND, not done; R, right; TPTG, transpterygoid.

*Alloderm, LifeCell; Duragen, Integra; Surgisis, Cook Medical.

Figure 4. Patient 4. This patient had undergone 4 prior sphenoidotomies with “packing” without identification and repair of the associated encephalocele and skull base defect. The preoperative (A) and 6-month postoperative (B) coronal computed tomographic scans are depicted following a transpterygoid approach. The opacification seen postoperatively is a result of obliteration of the recess. Note the complete integration of the bone graft and thickened skull base (neo-osteogenesis) from removal of the mucosa.
On secondary repair, the defect was repaired again using Surgisis dural underlay, septal bone underlay, Surgisis dural substitute overlay, obliteration of the free fat graft, and, finally, a contralateral pedicled septal flap to completely cover the obliterated lateral recess. Graft materials are shown in Table 2.

Six patients had a second, synchronous skull base defect. Patient 3 had a skull base defect in the ipsilateral ethmoid region, which was repaired concurrently. Patients 5, 8, 9, 10, and 11 had synchronous contralateral LRS defects; in patients 5 and 10 these were repaired concurrently (Figure 6 and Figure 7). Patient 8 had a bullet from a self-inflicted gunshot wound lodged in the left LRS but was noted to have a contralateral spontaneous LRS defect. Because a contralateral pedicled septal flap was used to repair the large defect caused by the bullet, performing a concurrent TPTG approach would have sacrificed the blood supply to the septal flap. When this patient had a VP shunt implanted to treat high ICP, her right “spontaneous” CSF leak was not clinically present, and she has elected to have careful clinical follow-up.

Eight patients had their ICPs measured postoperatively, and the average ICP was 26.7 cm H2O (range, 11-35 cm H2O). Three patients did not have lumbar pressure measurements recorded owing to (1) failure to obtain recordings prior to placement of a VP shunt for presumed idiopathic intracranial hypertension (subjective neurosurgical assessment was high ICP during ventriculostomy catheter placement) in patient 3, (2) replacement of a known malfunctioning VP shunt the day before surgery in patient 6, and (3) pressure readings following com-
bined endoscopic plus MCF approach for temporal lobe abscess that were felt to be an inaccurate representation in the setting of infection in patient 7. In all, acetazolamide therapy was initiated in 5 patients, whereas 5 patients required replacement for malfunctioning shunt (patients 6 and 10) or elective placement of a VP shunt for severely elevated ICP. Only 1 patient (patient 11) did not require postrepair intervention because she was found to have a postoperative ICP of 11 cm H₂O.

**COMMENT**

Overall, the LRS is a rare location to encounter a CSF leak, but our prior studies have demonstrated that the most common location for spontaneous sphenoid CSF leaks was the LRS (35%). Although some authors have felt that LRS CSF leaks are secondary to a patent lateral craniopharyngeal (Sternberg) canal, the predominant leak location is lateral to the sites of fusion of ossification centers and lateral to the second branch of the trigeminal nerve. The lateral craniopharyngeal canal is positioned medial to the superior orbital fissure, implying that an encephalocele originating through this theoretical canal must not only traverse the cavernous sinus but also penetrate 2 layers of dura mater before exiting the skull base. The current study and our prior work suggest that these leaks are indeed acquired rather than directly the result of a patent Sternberg canal. Nine of 11 patients in the present investigation had undergone prior unsuccessful attempts to address the disease process in the LRS. This allows an understanding and reiteration of certain key principles in the strategies for effectively treating this region.

**CONTROL OF INCREASED ICP**

All 11 patients in our study had a spontaneous CSF leak with associated encephaloceles, and 7 patients had radiographic evidence of empty sella syndrome on MRI, a finding often seen in the context of idiopathic intracranial hypertension. These data are consistent with those in other published reports. The most important factor for a successful repair is decreasing ICP through any means necessary, including nutritional (significant weight loss), medical (acetazolamide), or surgical (VP shunt) means. Five of the patients in our series had elective placement of a VP shunt. Two of these patients had received prior VP shunts that had failed and were the foundation for persistence or recurrence of their CSF leak. Three patients had elevated ICP (> 30 cm H₂O) through lumbar pressure monitoring postoperatively, despite the use of acetazolamide, a carbonic anhydrase inhibitor that decreases the production of CSF. Four patients have been maintained on acetazolamide alone.

**GRAFT TECHNIQUE AND PLACEMENT**

While the graft material used varies among patients, the principle of underlay and overlay grafting is represented throughout our repairs in this series. As demonstrated in our reconstructive algorithm (Figure 3), our typical method of free graft repair involves using a dural substitute graft placed through the defect intracranially as an underlay graft. This is followed by a small bone graft placed in the defect. A graft (mucosa/fascia/cadaveric or xenograft) is held in place initially with Evicel tissue sealant (Ethicon) (Figure 8). If we feel that the mucosa can be completely extirpated from the LRS, obliteration is performed after the skull base is repaired with the 3-layer repair. A free fat graft is placed in the LRS, and an absorbable packing and a finger cot sponge pack are placed in the maxillary sinus against the obliterated wall for 1 to 2 weeks. It is our opinion that the LRS should be completely obliterated with fat only if all of the mucosa can be accessed and extirpated. If the mucosa cannot be confidently removed, it is
Five patients in our cohort had bilateral skull base defects in the LRS. One patient (patient 10) underwent a bilateral TPTG approach. The other patient (patient 5) underwent a TPTG approach to 1 defect but had a contralateral defect that was accessible through a sphenoidotomy owing to a sagittal orientation. He received an ipsilateral septal flap repair for this defect (Figure 6 and Figure 7). Patient 9 did not have evidence of a CSF leak from a contralateral encephalocele. During a prior attempt to repair the actively leaking side via a TPTG approach at an outside institution, the infraorbital nerve had been damaged. The patient did not want the contralateral defect repaired at the time of his surgery. He elected to continue therapy with acetazolamide following definitive repair of the leaking side for treatment of his intracranial hypertension and is currently undergoing weight reduction management. Another patient (patient 8) had a CSF leak from a self-inflicted gunshot wound with a bullet lodged in the left LRS. Owing to the enormity of the defect, a contralateral septal flap was placed following repair and fat obliteration. We also identified a contralateral defect preoperatively, consistent with arachnoid pits and elevated ICP. This was leaking intraoperatively, but a TPTG approach would have sacrificed the blood supply to the septal flap used to seal off the recess on the left. This patient's incidental defect and associated CSF leak were intended to be repaired in a staged fashion; however, to date, she has not experienced clinical leakage and prefers to have this managed through careful observational follow-up.

In conclusion, this series highlights several key principles regarding repair of skull base defects in the LRS. Extirpation of sphenoid sinus mucosa prior to obliteration should help prevent the formation of mucoceles and decrease the potential for temporal lobe abscesses or other intracranial complications. Transpterygoid exposure and identification of the entire skull base defect following encephalocele ablation seems to be an excellent approach for successful repair of LRS defects with low failure rate, as demonstrated in this case series.

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Author Contributions: All authors had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Alexander, Riley, and Woodworth. Acquisition of data: Alexander, Riley, and Woodworth. Analysis and interpretation of data: Alexander, Chaaban, Riley, and Woodworth. Drafting of the manuscript: Alexander and Woodworth. Critical revision of the manuscript for important intellectual content: Riley and Woodworth. Statistical analysis: Alexander and Chaaban. Obtained funding: Woodworth. Administrative, technical, and material support: Chaaban and Woodworth. Study supervision: Riley and Woodworth.

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REFERENCES


