Recurrent Cerebrospinal Fluid Leaks in High-Risk Patients

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KEYWORDS

• Idiopathic intracranial hypertension • Cerebrospinal fluid • Glucagon-like-peptide-1 • Lumbar drain • Extended endoscopic approaches

KEY POINTS

- When available, vascularized flap reconstruction should be strongly considered for repair of recurrent cerebrospinal fluid leaks.
- Idiopathic intracranial hypertension (IIH) is highly associated with recurrent leaks; therefore, temporary or permanent intracranial pressure control should be considered in select patients.
- Prior cranial base irradiation increases delayed leak risk and complicates reconstruction.
- Vasculopathy (eg, diabetes and atherosclerosis) has not been proven to independently predict recurrence, though comorbidity optimization should be employed.
- Venous sinus stenting and ventriculoperitoneal shunting are options in select, high-risk patients with refractory IIH.

INTRODUCTION

Recurrent anterior and lateral cerebrospinal fluid (CSF) leaks are challenging scenarios that can result in pneumocephalus, risk of infection, increased morbidity, and extended hospital stays. These can occur perioperatively or in a delayed manner after extended endoscopic approaches (EEA) to the skull base for various pathologies. In a recent systematic review of 1083 cases of CSF leak, the recurrence rate was 10.3%. The most common location of recurrent leaks was at the anterior skull

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Abbreviations	
CSF	cerebrospinal fluid
EEA	extended endoscopic approaches
GLP-1	glucagon-like-peptide-1
ICP	intracranial pressure
IIH	idiopathic intracranial hypertension
LD	lumbar drain
sCSF	spontaneous CSF
VP	ventriculoperitoneal

base, reported at 55.9%, followed by the middle cranial fossa at 36.2%, and the posterior fossa at 1.7%.[1] With modern vascularized flaps and meticulous technique, successful primary closure rates are high, often in excess of 90%, though there are subsets of high-risk patients who continue to recur.[2] In the setting of EEA, the location of the surgical resection and presence of a high-flow intraoperative CSF leak can inform the likelihood of a postoperative leak, with suprasellar and clival lesions carrying a higher risk.[3] Patient factors such as immunosuppression or previous radiation may result in impaired wound healing and are plausible risk factors for recurrent CSF leak. Vasculopathy has historically been speculated to impair healing, but current evidence does not support it as an independent predictor of CSF leak.[4–6] In particular, spontaneous CSF (sCSF) leaks have a higher recurrence rate, ranging from 2.9% to 44.4% across various studies.[7] This may be due to untreated underlying idiopathic intracranial hypertension (IIH) in this patient population.[8] This article provides a comprehensive, evidence-based overview of the risk factors, diagnosis, and management of recurrent CSF leaks in high-risk patients.

RISK FACTORS

Idiopathic Intracranial Hypertension

IIH is a well-documented risk factor for the pathogenesis of spontaneous and recurrent CSF leaks. Elevated intracranial pressure (ICP) thins bone, promotes meningoencephaloceles, and drives repair failure.[9–11] Clinical suspicion for IIH arises with headache, papilledema, and pulsatile tinnitus, though patients with sCSF leaks rarely present initially with these symptoms.[12] Other risk factors for sCSF in patients with IIH include obesity, hypertension, and obstructive sleep apnea, with obesity being the most prevalent risk factor and reported in 73.9% of patients with sCSF otorrhea.[13] (This topic is discussed in detail in article “Idiopathic intracranial hypertension”).

Radiation

Radiation therapy, commonly employed for skull base malignancies, causes long-term tissue changes—fibrosis, hypovascularity, atrophy—leading to delayed breakdown and recurrent leaks.[14] Repair strategies must account for this compromised environment. Robust vascularized local flaps, such as nasoseptal flaps or pericranial flaps, or, where not feasible, free tissue transfer (eg, radial forearm and anterolateral thigh) are often required.[15]

Vasculopathy

Diseases affecting the microvasculature such as diabetes, hypertension, and atherosclerosis are known to impair wound healing and predispose to complications after

skull base surgery such as bleeding, sepsis, and urinary tract infections.[16–18] Inadequate microvascular perfusion due to long-standing atherosclerosis or diabetes can lead to thickening of the capillary basement membrane, endothelial dysfunction, and reduced tissue oxygen delivery. This can lead to slower fibroblast activity, delayed collagen deposition, and weaker scar formation, which can increase graft dehiscence. Vasculopathy can also impair angiogenesis and lead to defective remodeling. However, vasculopathies do not appear to independently predict CSF leak recurrence after endoscopic skull base surgery.[3,4] However, smoking, which causes vasoconstriction and oxidative endothelial injury, can increase flap complications and failure and is correlated with higher rates of return to the operating room after transsphenoidal skull-base procedures.[19] While optimization of glucose, nutrition, and vascular risk factors is appropriate perioperatively, vasculopathy should not be overemphasized as a primary determinant of leak recurrence.

Connective Tissue Disorders

Disorders that impair collagen or wound healing (eg, forms of Ehler-Danlos, Marfan syndrome, and other connective tissue abnormalities) can predispose to dural thinning and poor repair durability. Dura mater is a dense collagen-rich membrane; thus, conditions impacting fibrillar collagen can reduce collagen cross-linking, decrease tensile strength, and increase tissue elasticity of the dura.[20,21] Moreover, many connective tissue disorders impair wound healing and cause fibroblast dysfunction, resulting in suboptimal graft incorporation.[22,23] Some disorders, such a Marfan syndrome, can also impact structural bone fragility.[24] These can increase the likelihood of initial CSF leak formation and repair failure.

Immunosuppression and Impaired Healing

Chronic steroids, malnutrition, and immunosuppressive states plausibly impair remucosalization of the surgical site and resistance to infection. While direct evidence of an association with CSF leak risk is limited, perioperative optimization remains prudent. In these patients, the surgical team can more strongly consider a vascularized flap for skull base repair instead of a free mucosal graft.

Bone Disorders

Bone disorders linked to hyperparathyroidism and renal disease are classified under the umbrella term chronic kidney disease–mineral bone disorder. These diseases can disrupt mineral and hormone metabolism, including impaired activation of vitamin D, phosphate accumulation, secondary hyperparathyroidism, calcium mobilization from bone, leading to altered bone turnover, demineralization, thinning, deformities, and increased fragility.[25] Several bone or bone-related pathologies also appear to increase CSF-leak risk, particularly when they distort bone architecture, induce abnormal bone remodeling, or cause bone thinning. Some patients with spontaneous CSF leaks have significantly thinner calvaria/skull-base bone, even when controlling for obesity.[26,27] Thin skull base or tegmen can be associated with worse surgery outcomes, such as higher recurrence, second-site leaks, shunt requirement, and so forth.[28] Rare bone or renal disorders that cause bone loss or abnormal bone remodeling have been reported in association with spontaneous CSF leaks, demonstrating that bone pathology beyond pressure-induced thinning can lead to leak.[29] The relative contribution of pressure-induced remodeling on an intrinsic bone structural vulnerability (bone-remodeling, bone tinning, dysplasia, and bone-matrix disorder) remains unclear and likely varies by patient.

DIAGNOSIS

Diagnosis requires a combination of clinical suspicion and confirmatory tests. Direct visualization of the previous surgical site may be helpful in cases of significant CSF leak, which can be seen via nasal endoscopy. Beta-2 transferrin and beta-trace protein are gold-standard biochemical markers for testing in nasal secretions. Imaging with high-resolution computed tomography and MRI can localize defects and may also show pneumocephalus; cisternography is reserved for elusive cases. In suspected IIH, lumbar puncture, evaluation for papilledema, and venous imaging-guided ICP assessment[9–11,30–32] can be helpful. In many cases, intraoperative exploration may be required, and intrathecal fluorescein should be considered for difficult-to-localize defects or when multiple defects are suspected. (For detailed review, see article "Diagnostic Tools and Imaging for Skull Base CSF Leak.")

MANAGEMENT

Treatment of a recurrent CSF leak can be difficult due to the nature of the patient population and the natural challenges that revision surgery entails. Management should be tailored to each patient's clinical scenario and risk factors. In patients at higher risk of repair failure, every effort should be made to optimize their care before and after the repair. This optimization includes tapering medications that impair healing whenever possible, being vigilant about assessing potential infections, ceasing smoking in the perioperative period, and closely monitoring the repair over an extended period. Generally, the mainstay of treatment of a recurrent CSF leak is surgical repair, with strong consideration of adjunctive ICP-lowering strategies in a multidisciplinary manner with neurosurgery and neurology. In select cases, the use of perioperative lumbar drains (LDs) may be considered to reduce ICP, particularly in recurrent leaks that occur in the immediate perioperative period, provided that imaging supports the use of an LD without concern of CSF volume depletion or brain herniation. Surgical principles of leak repair include meticulous preparation of the defect site, watertight closure, multilayer reconstruction, and vascularized flap coverage for high-risk or high-flow leaks.[33–35]

Medical Treatments

Acetazolamide, a carbonic anhydrase inhibitor, reduces CSF production by decreasing bicarbonate ion formation in the choroid plexus, thereby theoretically reducing ICP. In the setting of recurrent CSF leak in a patient with IIH, acetazolamide could be considered, as it is generally well tolerated by patients. A review by Patel and colleagues[36] found that acetazolamide shows promise in preventing postoperative CSF leaks, but studies generally had small sample sizes and lacked high-quality randomized controlled trials, so further high-quality studies are still needed. There are currently no standardized protocols and dosing regimens for acetazolamide administration for this indication.

Another adjunct treatment to consider, after addressing the immediate CSF leak, is weight management. Obesity and elevated body mass index are both known risk factors for spontaneous CSF leaks and some poor outcomes, but the direct relationship between weight and recurrent CSF leak has not been established.[37,38] However, a mechanistic rationale for recommending weight loss to prevent recurrent leaks clearly exists. If the patient has been unable to lose weight despite other glucagon-like-peptide-1 (GLP-1) receptor agonists. GLP-1 agonists are a promising novel therapeutic option for patients with intracranial hypertension secondary to obesity. A randomized controlled trial found a significant reduction of ICP in patients with IIH: a 5.6 cm H_2O

decrease in 3 months.[12] Vesole and colleagues found that patients with IIH on GLP-1 agonists were 24% less likely to have sCSF leaks and 72% less likely to undergo primary anterior or lateral skull base leak repair.[39–41] To date, no studies have specifically examined the impact of GLP-1 agonists on CSF leak recurrence rates, warranting further investigation.

Revision Surgical Repair

Revision surgery can be technically demanding in cases of a recurrent CSF leak. Scar tissue, distorted anatomy, and prior graft failure complicate closure. Choice of repair is highly contingent upon the clinical scenario and available options left for the surgeon. Immediate postoperative leaks in healthy, low-risk patients may just be due to improper placement or shifting of the primary closure material and/or sinus packing. Kinks, folds, or lifted edges in a graft or flap may allow CSF to egress. In these cases, a simple adjustment or replacement of the repair material may be all that is needed to ensure a tight, waterproof multilayer closure.

Meticulous evaluation of the repair site in revision surgery is essential. The area around the defect should be carefully prepared with the removal of any nearby bony partitions and native mucosa to ensure the repair will lay flat with no tenting. Pedicled flaps should be tension-free and may need to be repositioned to ensure all aspects of the flap are placed on bone or appropriate tissue for adherence without "floating portions." The repair should be reinforced with well-placed sinus packing, such as a Merocel sponge placed in a gloved finger dressing, to maintain pressure on the area and promote adherence to the bone. In high-risk patients repaired primarily with a free mucosal or synthetic graft, revision surgery with multilayer repairs that include a vascularized flap or free tissue transfer is usually preferable.[14,15,33–35] Image-guided navigation and fluorescein can aid with localization of the defect and the site(s) of repair failure.[31] Outcomes improve when the closure strategy is tailored to individual patient anatomy, flow dynamics, and tissue quality.

Cerebrospinal Fluid Diversion

The need for perioperative and long-term CSF diversion for patients with recurrent CSF leaks is highly patient-dependent. Some previous studies showed the use of a perioperative LD reduced leak recurrence, while recent data support only selective use in high-risk patients.[36,42–44] A recent meta-analysis of endonasal CSF leak repair did not find a difference in postoperative improvement between those with and without LD.[45]

For patients with CSF leak and refractory IIH, ventriculoperitoneal (VP) shunting is associated with decreased leak recurrence after primary surgical repair.[8] However, the procedure carries the risk of infection, bleeding, hardware malfunction, headache from overdrainage, and abdominal issues.[2,46] High-risk features such as papilledema, obesity, prior CSF leak, multiple defects, and inadequate response to medical management may further inform decision-making as to whether to pursue a VP shunt. Patient selection, shared decision-making, and multidisciplinary follow-up are essential.

Venous sinus stenting offers an alternative in select patients with IIH and bilateral venous stenosis on magnetic resonance venogram (MRV), which has been shown to improve IIH symptoms but has not been studied in the context of CSF leak.[47–49]

SUMMARY

Recurrent CSF leaks require precise diagnosis, identification of risk factors, and tailored surgical and medical management that is patient-centered. The strongest

evidence supports robust vascularized repair, ICP management in IIH, and specialized approaches in irradiated patients. Vasculopathy should not be overstated as an independent risk factor. Multidisciplinary care remains the cornerstone of durable outcomes and requires dialog with appropriate colleagues.

CLINICS CARE POINTS

- Evaluate IIH in all spontaneous or recurrent leaks.
- Vascularized flaps reduce recurrence and should be preferred in high-risk cases.
- Irradiated patients require robust vascularized or free flap strategies.
- Optimize systemic comorbidities (eg, diabetes and nutrition) even if they are not independent predictors.
- Use LDs selectively, guided by intraoperative risk.
- Control ICP alongside repair in patients with IIH.
- Consider VP shunt or venous sinus stenting for refractory IIH.

DISCLOSURE

The authors have nothing to disclose.

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