Cerebrospinal Fluid Leaks Complicating Orbital or Oculoplastic Surgery

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Objectives: To report cerebrospinal fluid (CSF) leakage as an unusual complication following orbital or oculoplastic procedures and to describe its diagnosis and management.

Methods: Retrospective case review.

Results: Three cases of CSF leaks are described in patients following orbital or oculoplastic procedures. Two patients developed CSF leaks after orbital decompression surgery for compressive optic neuropathy and 1 patient had a CSF leak following endonasal dacryocystorhinostomy. In the first case, high-resolution computed tomography confirmed the site of the leak that required surgical repair. In the second case, a β2-transferrin test result confirmed the presence of CSF in the nasal drip, and coronal computed tomography identified a small fracture near the fovea ethmoidalis, but the leak resolved within 2 days of bed rest. In the third case, the patient reported several days of nasal dripping, but the problem had already resolved at the first follow-up appointment.

Conclusions: A CSF leak following certain orbital and oculoplastic procedures is a rare but well-recognized complication. This case report reviews the mechanisms, diagnostic techniques, and staged management of CSF leaks.

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CEREBROSPINAL FLUID (CSF) leak is a rare complication in orbital or oculoplastic surgery, but the surgeon should recognize its possible occurrence in certain procedures and be familiar with its diagnosis and management. We report 3 cases of CSF leaks manifesting in various ways and requiring different management following orbital decompression and an endonasal dacryocystorhinostomy (DCR).

REPORT OF CASES

CASE 1

A 57-year-old man demonstrated thyroid-associated orbitopathy with active inflammation, eyelid retraction with a left corneal ulcer, exophthalmometry of 25 OD and 27 mm OS, restrictive myopathy in all directions of gaze with constant diplopia, and bilateral compressive optic neuropathy (best-corrected visual acuity, 20/50 OU). His VISA (vision, inflammation, strabismus, and appearance) score was as follows: visual acuity: bilateral optic neuropathy; inflammation: 6 of 8; strabismus: 3 of 3; and appearance: severe. A course of 3 boluses of intravenous corticosteroids followed by radiation therapy did not resolve his optic neuropathy, and bilateral 2-wall orbital decompression was performed through a Lynch incision, removing the medial wall posteriorly to the sphenoid sinus and superiorly to the level of the ethmoid vessels and inferiorly along the orbital floor to the infraorbital nerve. The day after surgery, his visual acuity was 20/20 OU, and he had no afferent pupillary defect. Two days later after returning to his home in a remote community, he contacted us and reported clear fluid draining from his right nostril, particularly when leaning forward. With a suspected diagnosis of CSF leak, he was prescribed bed rest for a week and was asked to immediately report the development of fever or neurologic complaints. Headache and fluid leakage persisted during the next week, and he returned by airplane to the hospital. During the flight, his headache dramatically worsened, and he developed right leg weakness. On admission to the hospital, he was noted to have CSF rhinorrhea, while computed tomographic scan (CT) confirmed dehiscence on the floor of the anterior cranial fossa posterolateral to the cribiform plate and

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a small collection of intracranial air over the right middle cranial fossa. The expansion of this air during the flight was assumed to have worsened his headache and caused the paresis. Surgical repair was performed using an abdominal fat graft and tissue adhesive (Tisseal; Baxter Healthcare Corporation, Glendale, California) placed directly over the leaking spot through the original Lynch incision. There was no recurrence of the leakage, and paresis on the right side resolved.

CASE 2

A 60-year-old woman with hyperthyroidism diagnosed 1 year previously was initially seen with rapidly progressive thyroid-associated orbitopathy with the following VISA score: visual acuity: 20/50 OD and 20/40 OS, impaired color vision, and optic neuropathy present; inflammation: 5 of 8 and progressive; strabismus: bilateral limitation of gaze and diplopia with gaze; and appearance: exophthalmometry 23 OD and 24 mm OS. Her orbital CT scans confirmed severe apical crowding. A trial of oral prednisone (80 mg/d) did not improve her visual acuity, and bilateral orbital decompression of the medial wall and floor was performed. Following hospital discharge, she noted continuous drainage of fluid from her right nostril dripping to the back of her throat when lying down and associated with a dull right-sided headache. She was admitted to the hospital with a suspected CSF leak, and a consultation with neurosurgery was arranged. A CT scan showed a bony defect near the right fovea ethmoidalis (Figure), and a sample of the fluid demonstrated the presence of β₂-transferrin, confirming CSF rhinorrhea. She was treated with bed rest, and the leak spontaneously resolved 2 days later. Her visual acuity returned to 20/20 OU.

CASE 3

A 47-year-old woman underwent an uneventful left endonasal DCR and was discharged on the day of surgery. She returned 2 weeks later and reported resolution of tearing but development of left-sided headache and dripping of clear fluid from her left nostril the day after surgery. She discovered that the dripping and headache were reduced while lying on her back, and after confining herself to bed rest for 5 days, they spontaneously resolved. Because she had no symptoms at the time of follow-up, no further investigations were performed, but a diagnosis of suspected post-DCR CSF leak was made.

Cerebrospinal fluid leaks are a rare occurrence in ophthalmologic surgery and have seldom been reported. In the ear, nose, and throat literature, iatrogenic causes are responsible for 20% to 30% of all CSF leaks, typically following functional endoscopic sinus surgery. In a review of 1000 intranasal ethmoidectomies, Freedman and Kern reported 1 CSF leak. Dessi et al identified 2 postsurgical CSF leaks following 1192 endoscopic sinus surgical procedures. In ophthalmologic procedures, CSF leaks have been reported in 3 exenteration cases during separation of the periorbita from the orbital roof using monopolar cautery. In these cases, the CSF leak was noted during surgery and was repaired primarily with a suture through the dura. Neuhaus and Baylis reported 2 cases of CSF leaks during external DCRs that were successfully managed with bed rest. They surmised from subsequent cadaver dissections that the rotational movement during creation of the osteotomy could transmit forces to the cribriform plate, leading to fractures. Another case of CSF leakage during an external DCR in a patient with a meningocerebrocele was reported that resolved with observation and bed rest. Recently, Fayet et al reported a single case of a CSF leak complicating an endonasal DCR.

The position of the cribriform plate in relation to the medial orbital wall varies among individuals. It ranges from 10 to 16 mm behind the medial canthal tendon and anywhere from 11 to 30 mm above the insertion of the tendon, possibly lower in persons of Asian race.

In DCRs and medial wall orbital decompressions, the surgical site lies in proximity to the cribriform plate, but aside from unusual anatomical variants, there typically should be a safe margin so that violating the plate is rare. Exceptions likely occur in these procedures when rotational forces near the neck of the middle turbinate are transmitted to the cribriform plate, resulting in fractures, dural tears, and CSF leaks.

Of the cases performed by 1 of us (P.J.D.) during a 14-year period, there were 2 CSF leaks among 610 orbital decompressions (0.3%). There was 1 CSF leak among 2456 DCRs (0.04%) performed.
Management are instituted. Recent interviews of 135 patients with CSF leaks found symptoms of rhinorrhea in 64%, eventually associated with headache in 8% or meningitis in 10%, meningitis alone in 9%, and headache alone in 4%, as well as CT findings of pneumocephalus in 1%. The first step when suspecting a leak is to confirm that the fluid is CSF. A simple test in an office or bedside setting is to place a pledget soaked in fluorescein, 5%, intranasally. If it turns from yellow to green, this is suggestive of a CSF leak. In patients with insufficient fluid, asking the patient to perform a Valsalva maneuver and to lean forward may induce the leak. Glucose levels in CSF are two-thirds those of serum levels, and glucose strips have been used to diagnose CSF leaks because glucose concentrations in nasal secretions are low. However, upper respiratory tract infections or blood contamination of the nasal sample may give false-positive results. Because of poor specificity and only 80% sensitivity, this test is not recommended.

The best test available is β2-transferrin, a desialated form of transferrin. This enzyme is found almost exclusively in CSF and is present in only a few other body fluids such as cochlear perilymph and in low concentrations in aqueous and vitreous humor. Its levels are not affected by lacrimal or nasal mucous contamination, and only a small volume is required for its assay (depending on the laboratory, from 2 μL to 0.5 mL). Care must be taken to avoid contamination with blood, and bacterial infection may cause its decomposition. Some laboratories require a simultaneous serum measurement of β2-transferrin because liver dysfunction may increase its systemic levels. This test has 99% specificity and 97% sensitivity for CSF. In patients with a high suspicion of CSF leak but a negative β2-transferrin result, a second test is advised. Even in the face of a negative test result, its positivity is dependent on accurate sampling of the nasal discharge.

Another marker that is sensitive and specific for CSF is β-trace protein (prostaglandin D synthase), present in higher concentrations in CSF compared with the rest of the body. However, this test is not widely available.

Following laboratory confirmation of a CSF leak or in patients in whom a high suspicion exists, radiographic studies can aid in finding the bony defect and establishing the source of the leak. High-resolution coronal CT scanning using 1.5-mm sections with bone windows focusing on the ethmoidal roof is the procedure of choice and demonstrates a bone defect in more than 80% of cases. If the test result is inconclusive, CT cisternography can be ordered, in which contrast medium is injected intrathecally and highlights the leakage site; other options are magnetic resonance cisternography for suspected encephaloceles or meningoceles or nuclear medicine cisternography, with intrathecal injection of radioisotopes. In some cases, the site of leakage is not found until an endoscopic procedure for exploration and repair is performed.

Management of CSF leaks follows a graded pathway. In a review of 101 patients with traumatic CSF leakage, the leak spontaneously resolved within the first 24 hours in half of the patients. In the remaining 51 patients, 53% of leaks spontaneously resolved within 5 days from onset. Therefore, initial management for small CSF leaks should be bed rest with elevation of the head to reduce venous back pressure, as well as advice to avoid blowing the nose, which could force air or nasal secretions retrogradely into the intracranial cavity. Antifungal agents and stool softeners may be considered to reduce Valsalva maneuvers, and placement of a lumbar shunt might be used to reduce the CSF pressure. A neurosurgical consultation should be considered to detect neurologic complications and to monitor management.

A major complication of CSF leakage is meningitis, typically caused by Streptococcus pneumoniae. The use of prophylactic antibiotics is controversial; while some authors discourage their use, others report success in halving the incidence of infection (from 20% incidence of meningitis without antibiotics to 10% with prophylaxis). Should antibiotic prophylaxis be chosen, a drug that passes through the blood-brain barrier is recommended such as a third-generation cephalosporin (eg, ceftriaxone sodium), or trimethoprim-sulfamethoxazole or levofloxacin in allergic patients.

If conservative measures fail, surgical intervention is the definitive treatment. An extracranial approach is the traditional route through a Lynch incision, with reported success rate of 89% to 95%, but the endoscopic endonasal route has become popular because of good outcomes and low morbidity, with success ranging from 80% to 93.3% on the first attempt. Many materials are used to seal the leak, including fibrin glue, cyanoacrylate, or oxidized cellulose (Surgicel; Johnson & Johnson, New Brunswick, New Jersey), as well as mucosal grafts from the turbinate, septum, bone, fascia, fat, pericardium, mucoperiosteum, or cartilage. If these approaches to seal the leak fail, an open craniotomy with direct repair may be necessitated.

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REFERENCES

Eye on the Web

Frog Eyes

Did you know that frogs focus their eyes not by changing the shape of the lens but by moving it, much like a modern accommodating intraocular lens?1 Frog eyes can be brown, green, red, bronze, silver, and even gold; pupils can be round, horizontal, or vertical, triangular, star-shaped, or even heart-shaped! The large, protruding eyes of most frogs give them a very wide field of vision, which is useful for looking around without moving and scaring off prey (Figure). Frogs have good night vision and depth and movement perception. Unfortunately, they cannot see stationary objects well and can starve even when surrounded by insects if they are not moving.2 Another fascinating fact is that a frog retracts its eyes down toward the oropharynx when swallowing. When a frog swallows, its eyes help push food down its throat, since there is no bone between the eyes and the esophagus.3

Figure. A green frog with brown eyes. Photograph by Ilya Rozenbaum, MD.

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