Sternberg’s Canal: a Rare Cause of Cerebrospinal Fluid Rhinorrhea

Basheer Elsolh1; D.J. Cook, MD, FRCS(C)1,2

1Queen’s University School of Medicine, Kingston, Ontario, Canada
2Division of Neurosurgery, Kingston General Hospital, Kingston, Ontario, Canada

ABSTRACT

Cerebrospinal fluid (CSF) rhinorrhea, or leakage of CSF through the nasal cavity, is often seen with skull base fractures following massive head trauma or as a post-operative complication of skull base surgery. However, certain congenital osseous defects in the skull floor can also lead to CSF rhinorrhea. Sternberg’s canal results from an incomplete closure of the sphenoidal sinus during fetal and child development, forming a rare abnormal lateral craniopharyngeal canal connecting the middle cranial fossa and sphenoidal sinus. This creates the potential for formation of CSF rhinorrhea, meningocele, or encephalocele with temporal lobe herniation.

We present a case of a 66-year-old male presenting with a 3-month history of clear watery discharge from his left nostril. This was identified to be CSF following a β-2 transferrin assay. He denied prior similar episodes, head trauma, previous rhinological or neurosurgical procedures, headaches, cranial neuropathy, tinnitus, deafness, or focal neurological deficits. There was no current or historical evidence of meningitis.

A persistent left Sternberg’s canal with meningocele was diagnosed with computed tomography and magnetic resonance imaging. The patient successfully underwent open repair of the defect with autologous fat graft and had no postoperative complications.

Sternberg’s canal is a rare congenital malformation of the sphenoidal sinus through which meningocele or encephalocele can form. In the presence of an active CSF leak, it necessitates treatment, as it can significantly increase the risks of meningitis, brain abscess, intracranial hypotension, and seizures.

CASE PRESENTATION

A 66-year-old man presented to his family physician, complaining of a three-month history of clear liquid discharge from his left nostril. He experienced the rhinorrhea only at nighttime. He woke up nightly due to partial airway obstruction, and upon sitting up, had copious rhinorrhea soaking at least two facial tissues. He also complained of persistent post-nasal drip and an isolated episode of vertigo lasting 24 hours a few weeks prior to his presentation. He had no history of massive head trauma or prior neurosurgical intervention, and he denied headaches, neck stiffness, vision changes, weakness, numbness, paresthesia, convulsions, cognitive changes, fevers, chills, or night sweats. His past medical

Corresponding Author: Basheer Elsolh, Queen’s University School of Medicine, 15 Arch Street, Kingston, ON, K7L 3N6, Canada.
Email: belsolh@qmed.ca
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history was unremarkable, and his only medication was perindopril for hypertension control.

Upon physical examination, he appeared well and his neurological exam was unremarkable aside from watery nasal discharge. His pupils were equal and reactive to light and accommodation bilaterally, and the cranial nerve exam was normal. The patient had 5/5 power and normal reflexes in both upper and lower extremities bilaterally. His muscle tone, coordination, and gait were normal, and his sensation was intact.

Laboratory testing of the nasal discharge for β-2 transferrin confirmed it to be CSF. Computed tomography (CT) imaging showed a 4.5 mm bony defect in the left middle cranial fossa (Fig. 1, left panel). This was communicating with the left lateral pterygoid recess of the sphenoidal sinus and was deemed consistent with a persistent lateral cranioharyngeal (Sternberg’s) canal. Magnetic resonance imaging (MRI) confirmed the meningocele extending through the canal contained no brain parenchyma (Fig. 1, right panel).

The patient elected to have his Sternberg’s canal surgically corrected to resolve his persistent rhinorrhea symptoms and mitigate the risks of not treating a communicating cranioharyngeal defect, such as meningitis, brain abscesses, intracranial hypotension, and seizures. An endoscopic approach was considered for repair; however, due to the lateral position of the defect, an open craniotomy for reduction of the meningocele and repair was selected. An otolaryngologist consulted preoperatively supported the decision for an open rather than endoscopic approach. A small anterior temporal craniotomy was planned using neuro-navigation to optimize the positioning of the approach on the anterior middle fossa floor. An interfascial dissection was performed to isolate temporalis muscle inferiorly to give an adequate view of the middle fossa floor. Slight superior retraction of the temporal lobe revealed the bony defect in the skull floor. Foramen rotundum was visualized with the maxillary branch of the trigeminal nerve (V2) passing through it. The abnormal canal’s communication with the sphenoidal sinus and the defect in the dura were both visualized adjacent to this. A graft of fascia lata was excised from the patient’s left lateral thigh, along with a 3 cm³ piece of fat. The fat graft was used to plug the hole in the floor of the skull (Fig. 2). Fascia lata was used to cover the fat graft, and this was sealed with Tisseel, an artificial hemostatic fibrin agent (Baxter Healthcare Corporation, Deerfield, IL). The temporal lobe was released from retraction and set over the repair site. A 4x4 cm

Figure 1. Pre-operative imaging. CT scan (left panel) showing communication between left middle cranial fossa and lateral pterygoid recess of sphenoidal sinus. Meningocele herniating through persistent lateral cranioharyngeal canal as seen on T2-weighted MRI (right panel).
fascia lata graft was used as a duroplasty over the lateral temporal dura, a Jackson-Pratt 10 Fr. Round drain (Medline Industries, Mundelein, IL) was left in the extra-dural space, and the craniotomy was closed.

Post-operatively, the patient was instructed to remain supine for 72 hours while wearing compression stockings to reduce the risk of venous thromboembolism. The drain was removed two days post-operatively, and by the third day, the patient was eating and ambulating normally. He was discharged on post-operative day four. The patient had decreased sensation over the left V2 distribution of the trigeminal nerve post-operatively, and there was no evidence of recurrent CSF leak in the post-operative period. A postoperative MRI showed complete reduction of the meningocele and a fat plug sealing the location of the defect (Fig. 2). At a 15-month follow-up, the patient reported complete resolution of his V2 sensory loss, no CSF leak, and no wound complications.

**DISCUSSION**

Embryologically, the sphenoid bone forms from several precursor cartilaginous pieces that only fully fuse around four years of age. In 1888, Maximilian Sternberg described an incomplete fusion between the posterior basisphenoid and the lateral greater wing, leading to a persistent lateral craniohypophyseal canal connecting the intracranial space and sphenoidal sinus. This osseus malformation became known as Sternberg’s canal. It is not always symptomatic, but it has been linked to CSF rhinorrhea, visual field defects due to CN II pathway compression, and brain parenchymal herniation. CSF rhinorrhea results from CSF entering the sphenoidal sinus and draining into the nasal cavity through the superior nasal meatus.

Congenital causes of CSF rhinorrhea are rare but have been described in several locations in the skull base, including the sphenoid sinus. The prevalence of Sternberg’s canal in the general adult population is unclear in the literature. In the 19th century, Maximilian Sternberg described anatomical studies that postulated that his eponymous canal is present in around 4% of the adult population. Modern literature casts doubt on the prevalence being so high. A 2009 study by Barañano et al. found just a single case of Sternberg’s canal during a serial analysis of the CT scans of 1000 patients representative of the general adult population. Sternberg’s canal
is occasionally diagnosed incidentally in children with concomitant cleft facial dysplasias. While there is no consensus on the absolute incidence of Sternberg’s canals in the population, much of the literature classifies it as a rare or extremely rare condition.

The exact proportion of Sternberg canals that become symptomatic is also unknown. It is postulated most Sternberg canals do not form meningoceles or encephaloceles, and remain asymptomatic and likely undetected. Meningocele formation makes CSF leakage into the sphenoid sinus more likely due to increased likelihood of penetration of the dura and arachnoid. The incidence of congenital cranial meningocele/encephalocele in the general population is 1 in 35,000 people, with just 10% of those occurring at the skull base, and what is likely a much smaller proportion being attributable to Sternberg’s canal.

It is important to note that congenital basal skull deformities like Sternberg’s canal are not the most prevalent cause of CSF rhinorrhea. The presentation is more commonly due to traumatic or iatrogenic causes. It is crucial to identify a history of head trauma or prior skull base surgery in the patient, as these are the most common causes of such defects. CSF leaks secondary to trauma or skull base surgery may close on their own, and conservative management is recommended initially. Conservative management is also recommended for asymptomatic Sternberg’s canals discovered incidentally, with close monitoring for CSF leakage. In contrast, a symptomatic Sternberg’s canal with active CSF leaking may require surgical treatment in apprehension of the risks posed by CSF leakage, which include meningitis, intracranial hypotension, brain abscesses, and seizures. Since it is a congenital bony defect, there are no expectations for a Sternberg’s canal to close on its own, making CSF leakage potentially more long-term and problematic than in the context of trauma or skull base surgery. The natural history of untreated active CSF leakage includes meningitis and brain abscesses due to the communication between the cranial cavity and external environment through the nasopharynx. In addition, there is an increased risk of seizures and intracranial hypotension. The latter can manifest in lower cranial nerve deficits and pituitary dysfunction, among other issues. Some cases of intracranial hypertension have also been reported following CSF leakage, likely due to a secondary increase of CSF production by the choroid plexus in reaction to the leakage.

Certain sphenoidal defects can be repaired endoscopically through trans-nasal, trans-septal, or trans-pteroidal sphenoidal access. An endoscopic approach has the advantage of avoiding a craniotomy, thus making the operation less invasive. The neurosurgeon in this case deemed this patient’s lateral craniofaryngeal canal to be too laterally placed for such an approach to be technically achieved without expanded endoscopic sphenoidal instrumentation. Consultation with an otolaryngologist supported the view that the extreme lateral location of the sphenoidal sinus defect would necessitate a larger basal sphenoidal opening that would increase the risks and severity of endoscope-related rhinological complications such as epistaxis, hypomiosis, excessive conchal loss, and dry nasopharynx. Alternatively, an open intracranial approach would effectively expose the bony defect from above, allowing for full visualization of the affected structures, reduction of the meningocele, and repair. Disadvantages to this approach include the need to expose the temporal lobe laterally, which is associated with postoperative seizures as well as a higher incidence of stroke related to Vein of Labbé injury. Open craniotomy also incurs the risks of infection, bleeding, and CSF leak postoperatively. There are currently no universal guidelines to dictate whether an open or endoscopic approach should be used depending on the laterality of the defect.
The preference of an open approach was based on the experiences of the neurosurgeon and otolaryngologist after considering both approaches. The patient in this case was informed of the natural history and possible complications of the different approaches. Following the recommendation of the neurosurgeon and otolaryngologist, he opted for the open repair.

An autologous fat graft was used in this case to seal the bony defect. This is a common and reliable method to obliterate the sphenoidal sinus canal, as the graft can be molded to fully seal the defect and form a watertight seal with minimal morbidity to the donor site. Various other materials, both synthetic and autologous, have been trialed to plug skull base defects, but fat grafts have been the most successful. Autologous fascia lata from the patient’s thigh was used to cover the fat graft and as a duroplasty patch to repair the temporal lobe dura following meningocele excision. No bony or titanium mesh scaffolding was required in this case; however, this may be needed in cases with larger defects.

The case presented here is notable for the perceived rarity of the presenting condition and the technically challenging intervention necessary to repair the defect. The procedure immediately resolved the patient’s symptoms and there were no significant post-operative complications.

### REFERENCES


### LEARNING POINTS

1. Though its exact prevalence is unclear in the literature, Sternberg’s canal is a rather rare craniofacial deformity that results from incomplete fusion of the sphenoid bone.

2. While it may be asymptomatic, a Sternberg’s canal has the potential to form meningocele/encephalocele and CSF leakage. The latter puts the patient at increased risk of meningitis, brain abscesses, intracranial hypotension, and seizures.

3. To mitigate these risks, surgical intervention may be necessary in CSF leakage from a Sternberg’s canal. Open intracranial or endoscopic trans-sphenoidal approaches may be considered for repair.
