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Pseudomeningoceles of the sphenoid sinus masquerading as sinus pathology

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Abstract

Objective—To describe the clinical presentation, pathophysiology and treatment of spontaneous cerebrospinal fluid (CSF) leaks of the sphenoid bone, with an emphasis on a previously undescribed form in this location, in which CSF is trapped under the mucosa of the sinonasal cavity or in the soft tissue of the skull base.

Study design—Case series and literature review. Level of evidence 4.

Methods—Analysis of cases through medical records and literature review.

Results—Four examples of unusual spontaneous CSF leaks of the skull base are presented. In each case, a CSF collection was contained behind the sinonasal mucosa of the sphenoid sinus, resembling a nasal polyp or mucocoele on exam or imaging. In one case, the fluid collection was also associated with significant bone resorption and extravasation into the soft tissue of the infratemporal fossa. In each case, small defects of the ventral skull base (sphenoid bone) were the source of the CSF leaks. Successful treatment was achieved after transnasal endoscopic repair of the skull base defects using a combination of free abdominal fat grafts, free fascial grafts and pedicled nasoseptal flaps. Postoperatively, a ventriculoperitoneal shunt was placed if the intracranial pressure was elevated.

Conclusions—Spontaneous CSF leaks arising in the sphenoid sinus may not always present with overt CSF rhinorrhea but with a submucosal fluid collection (pseudomeningocele) that may mimic a mucocoele or nasal polyp. These *bona fide* pseudomeningoceles of the skull base may be associated with elevated intracranial pressure and can be managed using endoscopic endonasal surgery.

Keywords

CSF leak; mucocoele; meningocele; skull base; sinus

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Introduction

Defects in the skull base can lead to spontaneous herniation of meningeal membranes and present as a meningocele or meningoencephalocele. The bony defect may be small and clinically silent until a breach in the meninges appears, leading to a cerebrospinal fluid (CSF) leak. In the anterior cranial base, typical presentations include CSF rhinorrhea and a pulsatile or compressible nasal mass covered by attenuated meninges and mucosa. In theory, in the context of a spontaneous CSF leak, if the CSF remains contained by mucosa or soft tissue of the skull base, it should form a pseudomeningocele, a collection of CSF in the soft tissue that is not surrounded by arachnoid membranes. While meningocele, meningoencephalocele, and spontaneous CSF leaks of the skull base have been recognized and are routine pathologies for the skull base surgeon and the rhinologist, spontaneous pseudomeningocele of the skull base has surprisingly not yet been reported. The cases reported here are unusual in that they presented on clinical examination or imaging as a nasal polyp or fluid collection simulating a mucocoele, but were filled with CSF and represent a *bona fide* pseudomeningocele.

Materials and Methods

Identification of Cases: A 3-year review of operative reports with billing code for CSF leak or meningocele repair in the Center for Skull Base Surgery at the University of Pittsburgh Medical Center was performed from 2006 to 2009. Case 4 was provided by a senior author at Charing Cross Hospital in London, UK.

Review of the literature: The PubMed database (www.ncbi.nlm.nih.gov/PubMed) was queried for “pseudomeningocele AND head and neck OR skull” in the English language. Pertinent literature resulting from this search, or found in the bibliographies of these articles, was reviewed.

Surgical techniques: Standard techniques of endoscopic endonasal approaches to the skull base are described in detail elsewhere.¹⁻³

Results

Case 1

This patient presented with nasal obstruction secondary to a mucosal covered “polyp” filling the posterior nasal cavity and radiographic expansion of the lateral recess of the sphenoid sinus and posterior maxilla. There was no visible bone defect of the skull base on CT scan and preoperatively the mass was diagnosed as a mucocoele. Imaging for this case was done at an outside hospital and is no longer available. The patient underwent an endoscopic endonasal transpterygoid approach to the lateral recess of the sphenoid. Intraoperatively, there was elevation of the mucosa from the lateral recess of the sphenoid with a contained collection of fluid. Following removal of the mucosa, drainage of CSF from a 2mm defect in the lateral recess of the sphenoid sinus was noted with no herniation of meninges. This was repaired successfully with free fascia and fat grafts. The postoperative course was uncomplicated; the patient was subsequently lost to follow up. This case, which lacks image documentation, led us to recognize a novel localization for pseudomeningoceles, which have yet not been described in the sinonasal cavity. Intraoperative image documentation was recorded on subsequent cases.

Case 2

A 42 year old woman was referred to our institution with a two month history of left-sided clear rhinorrhea positive for beta-2-transferrin. She complained of worsening headaches

over the past month, and intermittent blurred vision. She recalled a history of blunt head trauma with concussion 20 years ago. The remainder of the medical history was significant for a gastric bypass for obesity.

Clinical examination and imaging. Nasal endoscopy revealed a pulsatile mass of the left sphenoid sinus without evidence of a CSF leak. The ocular fundus examination showed normal appearing optic nerve papillae. The rest of the physical examination was within normal limits. Computed tomography (CT) showed a total opacification of the left sphenoid sinus without bony erosion (Figure 1 A, B). Magnetic resonance imaging (MRI) revealed that the sphenoid sinus opacification was a fluid collection hyperintense on T2-weighted images (Figure 1C) and hypointense on T1-weighted images.

Operation. An endoscopic endonasal approach to the skull base was performed¹. A right sided nasoseptal flap pedicled on the posterior nasal artery³ was elevated, and bilateral wide sphenoidotomies with a left transmaxillary transpterygoid approach provided access to the mid-clivus and lateral sphenoid recess². The CSF leak was found at the posterior aspect of the sphenoid via a very small opening at the mid-clivus. Abundant fluid was arising from the defect and CSF collected under the mucosa. The small opening was obstructed with artificial dural graft (Duragen, Integra Lifescience), and covered with the nasal septal flap.

Postoperative course. Postoperative lumbar puncture confirmed elevated intracranial pressure. A programmable ventriculoperitoneal shunt was placed. Nineteen months after surgery, neither the CSF rhinorrhea nor the sphenoid sinus mass have recurred.

Case 3

A 37 year old man was referred with recurrent right CSF rhinorrhea. He had sustained blunt head trauma at age 2 in the left frontal area, from which he fully recovered. He remained asymptomatic until 4 years prior to his visit. At that time, he developed a spontaneous CSF leak with intractable headache. He underwent a bifrontal craniotomy for meningocele repair, and was described to have a sphenothmoid meningocele. Thereafter, he had continuing CSF leakage and underwent a transnasal endoscopic repair of a left posterior ethmoid CSF leak, followed by placement of a programmable ventriculoperitoneal shunt. The symptoms recurred despite an appropriately functioning shunt, and the headaches worsened. He was referred to us at this point in his care.

Clinical examination and imaging. Nasal endoscopy revealed changes compatible with a previous limited endoscopic sinus surgery of the anterior and posterior ethmoid sinuses. The anterior face of the sphenoid sinus was intact, demonstrating that the sphenoid sinus was uninvolved in the prior surgery. There was no evident CSF leak. The rest of the head and neck, and cranial nerve exam was normal. Computed tomography revealed a cystic expansion of the body and right greater wing of the sphenoid sinus with complete dissolution of bone in some areas (Figure 2 A, B). Fluid appeared to have extravasated into the soft tissues below the skull base (Figure 2 A-C).

Operation. An endoscopic endonasal approach to the skull base was performed¹. A left sided nasoseptal flap pedicled on the posterior nasal artery was elevated³. Close intraoperative examination confirmed absence of prior surgery in the sphenoid sinus. Wide sphenoidotomies were then performed, revealing a ballooning of the mucosa of the floor and posterior wall of the sphenoid sinus; the mass resembled a mucocoele. The mucosa was removed and clear CSF was drained. There was significant expansion and destruction of bone of the sphenoid region, especially in the right lateral recess. On this side, the vidian nerve and artery were completely exposed. Similarly, the bone of the medial orbital apex and the middle fossa were eroded. A 2 mm breach in the dura with leakage of CSF was

noted at the right superior orbital fissure, superior to the second division of the trigeminal nerve. A small collagen graft (Duragen, Integra Lifescience) was placed through the dural defect to control the leak. Abdominal fat grafts were used to obliterate the clival recess and the septal mucosal flap was transposed over the fat grafts and the medial and inferior walls of the right middle cranial fossa.

Immediate postoperative course. The initial postoperative course was uncomplicated, with resolution of headaches. However, the patient developed recurrent right-sided CSF rhinorrhea associated with excruciating headaches on postoperative day eighteen. Re-exploration revealed a healthy mucosal flap. The site of the previously identified CSF leak remained effectively repaired. Therefore, to identify an additional source of CSF leak which might have escaped our scrutiny during prior surgery, we widened the exposure via a right transmaxillary, transpterygoid approach² and explored the lateral recess of the sphenoid sinus and the ventral aspect of the infratemporal fossa. The pseudomeningocele extended into and expanded the pterygopalatine space. We then noticed a small breach in the dura lateral to foramen ovale, with drainage of clear CSF. Again, a collagen graft was placed over the dural defect, encompassing the entire infratemporal skull base. The lateral recess of the sphenoid was then covered with fat grafts, and the septal mucosal flap was transposed over the fat.

Postoperative course. With eighteen months postoperative follow up, the patient has had no recurrence of a CSF leak or submucosal CSF collection. His headaches have resolved.

Case 4

A 55 year old woman presented with a longstanding history of unilateral right nasal obstruction which had recently worsened. She denied any rhinorrhea or blunt head injuries. Past medical history included diabetes, hypertension and obesity.

Clinical examination and imaging. Nasal endoscopy revealed a deviated septum towards the right with an ipsilateral spheeno-ethmoidal polypoid mass (Figure 3C). Cranial nerve function and the rest of the head and neck examination were normal. Computed tomography (CT) defined the right nasal polyp which extended into the post nasal space and obstructed the spheeno-ethmoidal recess with complete opacification of the sphenoid sinus (Figure 3A). On the MRI, the mass had low-intensity signals on T1 weighted images (Supplemental Figure) and a high T2 signal consistent with an inflammatory polyp (Figure 3B).

Operations and initial postoperative course. A septoplasty and endoscopic excision of the unilateral spheenoethmoidal polypoid tissue was undertaken. Excision of the presumed nasal polyp resulted in clear fluid drainage that was later shown to be beta-2 transferrin positive. Postoperatively the patient had persistent CSF rhinorrhoea necessitating a second procedure. Intrathecal injection of fluorescein localized the origin of the CSF leak in the right sphenoid. A septal mucoperichondrial flap was raised to repair the defect in two layers, using fibrin glue. However, the right-sided rhinorrhoea recurred shortly after the procedure and the patient was referred to author (HAS) for a revision. An endoscopic endonasal approach with wide sphenoidotomy was performed. Ballooning of the mucosa of the floor and lateral wall of the sphenoid was identified and stripped from the underlying bone. The CSF leak originated from a 3 × 3 mm bony defect in the infero-lateral aspect of the sphenoid sinus (Figure 3D). A layered repair of the defect was undertaken with fascia lata, septal cartilage and mucoperichondrium (Figure 3E). The fat was used to fill the sphenoid sinus cavity, and fibrin glue and Gelfoam were placed. Since the CSF pressure was not elevated, the patient did not require a lumbar drain.

Postoperative course. The patient was discharged home on the second postoperative day and had no recurrence of her CSF leak four months postoperatively.

Discussion

Here we describe, to our knowledge, the first reported cases of spontaneous pseudomeningocele of the skull base. In all of these cases, CSF leaked through the ventral meninges at the skull base and accumulated in the local soft tissue and behind the mucosa of the paranasal sinuses. Clinically, some of these cases were associated with CSF rhinorrhea. But in the absence of CSF rhinorrhea, a pseudomeningocele may be difficult to differentiate from a mucocoele radiographically.

The pathophysiology of a pseudomeningocele involves both dural and arachnoid tears with leakage of CSF into the surrounding soft tissue, forming a fluid collection often surrounded by a fibrous capsule⁴. Most reports of pseudomeningoceles published in the English literature are localized at the level of the thoracic or lumbar spine. The lesion is causally linked to an acute dural and arachnoid tear induced by surgery or trauma. Such tears occur most commonly at the junction of the theca with the nerve root sleeve, causing a pseudomeningocele in up to 2% of laminectomies⁴⁻⁵. However, pseudomeningoceles rarely occur in the head and neck area. Again, trauma or iatrogenic intervention were the primary causes of most reported cases in this region. For instance, CSF extravasation into the soft tissue occurs in 4.5% of neurotologic procedures⁶ and is described as a rare complication of an atlanto-occipital dislocation⁷⁻¹⁰ or as a rare cause of post-traumatic orbital swelling^{4, 11}. Herein, we report, to our knowledge, the first cases of spontaneous pseudomeningocele affecting the skull base. While it is possible that pseudomeningoceles have been previously observed in the skull base or paranasal sinuses, and may even have been included in series of CSF leaks, the entity has not been recognized or reported as such.

We believe that pseudomeningoceles of the skull base originally develop like spontaneous CSF leaks. Small arachnoid tears lead to CSF leaks through small dural defects or “pitholes” located at the skull base¹²⁻¹³. The defect may be of developmental origin. However, unlike most spontaneous CSF leaks where CSF extravasates directly into the sinus cavity, in spontaneous pseudomeningocele of the skull base, the CSF accumulates behind the mucosa of the paranasal sinus and may extend into the soft tissue of the infratemporal fossa or pterygopalatine space. Importantly, no recent trauma preceded the development of these lesions; previous surgical trauma likely did not contribute to the development of a pseudomeningocele in case 3, as the lesion appeared outside of the surgical field. However, two patients did have a remote history of blunt head trauma that may have played a role in the pathophysiology of the pseudomeningocele.

In the four cases reported here, the CSF leak responsible for the pseudomeningocele originated in the sphenoid sinus. Pseudomeningoceles may develop preferentially in the sphenoid sinus rather than the anterior ethmoid and cribriform regions because the dura overlying the sphenoid sinus may be less prone to herniation, and the mucosa of the sphenoid sinus more likely to be elevated without rupture.

The third case of pseudomeningocele that we report was associated with significant bony erosion of the sphenoid bone. Interestingly, bone erosion occurs with spontaneous pseudomeningocele and not with CSF leaks secondary to craniotomy¹⁴. While it is possible that the skull base erosion precedes the CSF leak, we propose that bone erosion happens as a consequence of a leak, in the same way that pseudomeningoceles of the cranial vault or intradiploic arachnoid cysts of the cranial vault or spine are known to erode bone. In an intradiploic pseudomeningocele, whether of spontaneous or post-traumatic origin, a small

defect of the arachnoid and dura lets CSF leak into the diploic space via a small defect of the inner table^{11, 15-16}. It is postulated that the hydrostatic pressure of the CSF trapped between the diploe increases via a ball-valve mechanism¹¹ or as a consequence of CSF pulsation¹⁶, leading to bone resorption. Intradiploic arachnoid cysts are similar lesions that can develop without obvious trauma and are connected to the subarachnoid space via a small round dural defect of a few millimeters, but the CSF is contained by arachnoid membrane. But they are thought to expand in a similar fashion^{5, 14, 17-20}. Clinically, intradiploic arachnoid cysts have been reported at the skull base²¹ and may cause significant bone deformity²².

With spontaneous pseudomeningoceles of the skull base, we hypothesize that CSF may enter the sphenoid bone or the basilar part of the occipital bone via preexisting dehiscences or preformed pathways. Increased hydrostatic pressure due to fluid accumulation by a ball-valve or slit-valve mechanism results in bone resorption and subsequent enlargement of the bone defects. If the pseudomeningocele develops in the clivus, which contains a diploic space, the erosion of the trabecular bone ultimately breaches the external table. The CSF then accesses the submucosal space and potentially the soft tissue of the skull base where it accumulates. The pseudomeningocele may then masquerade as a nasal polyp or mucocoele. This is in contrast to intradiploic pseudomeningoceles or intradiploid arachnoid cysts, where CSF is sequestered within the bone.

Our current approach to treating pseudomeningoceles of the skull base is identical to the endoscopic repair of CSF leaks²³⁻²⁶. Pre-operatively, we obtain a fine cut (1.5mm or less) CT of the skull base to identify small erosions of the skull base. We also obtain an MRI to evaluate for an associated meningo-encephalocele. Intra-operatively, we remove the pseudomeningocele and identify all the skull base and dural defects causing the CSF leak. This requires a wide exposure, as illustrated by the second case. We then perform an endoscopic duroplasty followed by coverage with a vascularized mucosal pedicled flap (nasoseptal flap) or free tissue grafts^{3, 26}. If the mucosal flap does not reach the dural defect, a fat graft, temporoparietal fascia flap or extracranial pericranial flap are alternatives to be considered.

The initial success rate of endoscopic repair of CSF leaks is greater than 90% in expert hands²⁷, in contrast to this report in which the initial success rate was 50%. While this difference may be secondary to the peculiar pathology of spontaneous pseudomeningocele, we attribute this discrepancy to a combination of factors. First, the two cases which failed initial treatment (cases 3 and 4) were both recurrent cases with multiple repair attempts before referral. Previous surgery has been identified as an independent risk factor for postoperative CSF leak after endoscopic skull base surgery (Snyderman, unpublished retrospective review of 1000 cases). Second, pseudomeningoceles may present with multiple erosions of the skull base, as observed in case 3. This further substantiates our recommendation for a wide intraoperative exposure to facilitate identification of additional sources of CSF leak.

Elevated CSF pressure may play a role in the development of pseudomeningoceles of the skull base and patients should be investigated for occult hydrocephalus before or following repair of the defect²³. At our institution, we perform a lumbar puncture after surgery or following removal of a lumbar spinal drain. If CSF pressure is elevated, medical (acetazolamide) or surgical (ventriculoperitoneal shunt) management may be considered. However, the relationship between elevated CSF pressure and the development of a pseudomeningocele remains to be established. In case 3, for instance, the pseudomeningocele developed despite a normal functioning ventriculo-peritoneal shunt.

Conclusion

A spontaneous pseudomeningocele is a novel entity that may masquerade as a unilateral polypoid mass or a mucocele, with or without rhinorrhea. It occurs when CSF becomes trapped behind the paranasal sinus mucosa or extracranial soft tissues, and may be associated with bony erosion of the sphenoid bone or the clivus. For successful treatment, we recommend a wide exposure to address all potential skull base defects. Successful treatment can be obtained with traditional endonasal skull base surgical techniques. Increased recognition of this pathological entity is necessary to ensure proper management.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Pre- and post-operative radiographic imaging of Case 2. Preoperative CT (A, B) and MRI (C) were obtained immediately prior to surgery. The left sphenoid sinus is opacified by the pseudomeningocele (asterisk). The clivus (arrowhead) is unusually thin but intact in this patient. The opacification is fluid-filled and hyperintense on T2-weighted MRI imaging (MRI-T2). The postoperative axial CT (D) shows resolution of the pseudomeningocele. The nasoseptal flap is visible over the clivus.

Figure 2.

Pre- and post-operative radiographic imaging of Case 3. Preoperative CT (A, B) and MRI (C) were obtained immediately prior to surgery. The sphenoid sinuses are opacified (asterisk), and a fluid collection fills part of the right infratemporal fossa (arrowhead). Note the extensive erosion of the sphenoid bone. The opacification is fluid-filled and hyperintense on T2-weighted MRI imaging (MRI-T2). The postoperative axial CT (D) shows resolution of the pseudomeningocele with the fat graft filling the right infratemporal fossa.

Figure 3.

Pre- and post-operative imaging of Case 4. A preoperative CT (A), identifies bilateral sphenoid opacification with a defect of the bony skull base (arrow). The opacification is fluid filled and hyperintense on T2-weighted images, as seen on a preoperative MRI (B). The preoperative nasal endoscopy (C) shows a pseudomeningocele (arrow head). Intraoperatively (D & E), the CSF leak is identified after intrathecal injection of fluorescein (D, white arrowhead). It is successfully repaired with a dural plug (E, black arrowhead). Middle Turbinate (MT), Inferior Turbinate (IT), and Septum (S).