

The transnasal endoscopic management of spontaneous cerebrospinal fluid rhinorrhea from the lateral recess of the sphenoid sinus

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Background

Spontaneous cerebrospinal fluid (CSF) rhinorrhea from the lateral recess of the sphenoid sinus is surgically challenging. Sternberg's canal has stirred great controversy as the potential source. The aim of this study was to present our experience with endonasal endoscopic repair, the possible etiopathology, and the outcomes.

Study design

This prospective study comprised 10 patients (seven female and three male) with spontaneous CSF rhinorrhea from the lateral recess of the sphenoid sinus, which was not related to trauma, previous surgery, tumors, irradiation, or meningitis. CSF rhinorrhea was confirmed with $\beta 2$ transferrin test and high resolution CT scan (HRCT) and MRI cisternography. All patients were treated with the endonasal endoscopic conservative retrograde trans-sphenoidal approach.

Results

The mean BMI was 35.55 ± 2.84 kg/m². Elevated intracranial pressure was present in all cases confirmed directly (with a mean intraoperative lumbar drain pressure of 27.5 ± 3.84 cm H₂O), and indirectly [with the presence of primary empty sella (100%), arachnoid pits (30%), and attenuated skull base (40%)].

Osteodural defect was constantly present in the superior wall of the lateral recess, lateral to the foramen rotundum, none above the foramen rotundum or below the vidian canal orifice. The mean follow-up was 46.9 ± 8.26 months.

Conclusion

The endonasal endoscopic repair is a safe and effective method. The etiopathology is multifactorial. The management of elevated intracranial pressure is crucial. The potential source is not Sternberg's canal but persistent cartilaginous vascular channels at the ossification center of the alisphenoid, cartilaginous precursor of the greater wing of the sphenoid bone.

Keywords:

intracranial pressure, lateral recess of the sphenoid sinus, meningoencephalocele, skull base, spontaneous cerebrospinal fluid rhinorrhea

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Introduction

Spontaneous cerebrospinal fluid (CSF) rhinorrhea with or without a meningoencephalocele from the lateral recess of the sphenoid sinus (LRSS) represents a unique challenge due to its anatomical relations to the internal carotid artery, cavernous sinus, optic nerve, and extreme variations in the pneumatization of the sphenoid sinus. The location of meningoencephaloceles in the sphenoid sinus may be medial/planum type, perisellar type, and a lateral recess type (which is the pneumatization of the sphenoid sinus lateral to the foramen rotundum and the vidian canal orifice) [1]. Generally, CSF leaks are classified on the basis of the underlying etiology [2]. Leaks without an identifiable cause, such as those caused by trauma, surgery, malformation, tumor, and previous radiation therapy, are generally referred to

as 'idiopathic' [2–5]. The underlying etiology in the majority of individuals in this category has been elevated intracranial pressure (ICP) and is postulated to represent a manifestation of benign intracranial hypertension and thus the term spontaneous is preferred [6,7]. Forty-one percent of spontaneous CSF rhinorrhea is present in the LRSS [2,8].

Sternberg's canal [9] has stirred great controversy in the literature as to whether or not it is the potential

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source of the osteodural defect in spontaneous CSF rhinorrhea with [7,10,11] or without [2,4,12] a meningoencephalocele from the LRSS and whether the canal is medial [4,12] or lateral to the foramen rotundum [7,11–13].

Surgical management of spontaneous CSF rhinorrhea in the LRSS is a distinctive entity with respect to treatment and has changed over the last 20 years [4,12]. Different approaches for this area have been proposed: external endocranial, external extracranial, and endoscopic [14,15]. In the past, the external approaches were the standard procedures to get access to the LRSS. With the use of rigid-angled telescopes, blind angles and recesses in the nose and sinuses are visible clearly with minimal bone removal (Fig. 1). The endonasal endoscopic techniques have become the gold standard for CSF leak repair with success rates higher than 90% in most series [16]. The endonasal endoscopic transtethmoid–sphenoid pterygoid approach (transpterygoid approach) is the surgical corridor of choice for the treatment of spontaneous CSF rhinorrhea with or without a meningoencephalocele from the LRSS [17–19] (Fig. 1).

The aim was to present our experience with the endonasal endoscopic repair of these cases, the possible etiology, and the outcomes.

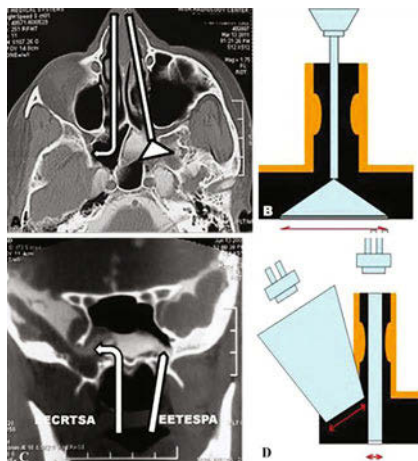
Patients and method

After approval from the university ethical committee for the design and protocol, consent of all patients toward the method of treatment was obtained. In this

prospective study, 10 patients (seven female and three male) with spontaneous CSF rhinorrhea in the LRSS, which was not related to trauma, previous surgery, tumors, irradiation, and meningitis, attending Ain Shams University Hospitals (Cairo, Egypt) between January 2009 and June 2014 were included. Diagnostic workup included endoscopic examination, laboratory tests, and imaging. CSF was confirmed using chemical analysis and $\beta 2$ transferrin test in all patients. Every patient underwent HRCT cisternography and MRI cisternography (Fig. 2).

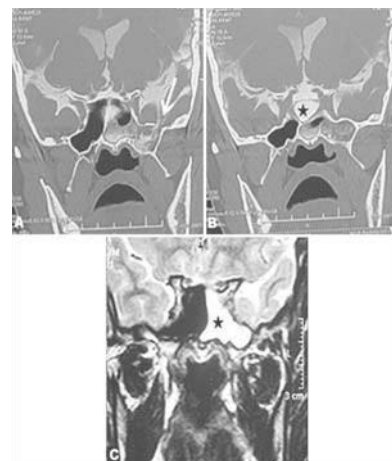
Evaluation of ICP was carried out through direct intraoperative measurement of lumbar drain pressure and through indirect radiological evaluation of CT and MRI scans. Radiological signs evaluated include the presence of CSF leak or a meningoencephalocele in the LRSS, the site of the osteodural defect, and the signs of indirect intracranial hypertension (consisting of attenuated scalloping of the anterior or middle cranial fossa bone, arachnoid pits, skull-base defects, empty sella, dural ectasia, and abnormalities of the optic nerve sheath complex). Table 1 shows the demographic data, the clinical symptoms of the patients at presentation, and the BMI. BMI was graded as follows: normal, 18.5–24.9 kg/m²; overweight, 25–29.9 kg/m²; and obese, greater than 30 kg/m². BMI is a measure of body fat based on height and weight. All patients were treated by means of the endonasal endoscopic conservative retrograde trans-sphenoidal approach (retrograde). Table 2 evaluates the intraoperative variables, including size of the defect (measured intraoperatively), shape of the defect, length of the operation, and value of the ICP. Table 3 evaluates postoperative

Figure 1



(a) The advantage of a 45° degree lens. (b) Tunneling with endoscope. (c) Left-side endoscopic endonasal transtethmoid–sphenoid pterygoid approach (EETESPA), right-side endoscopic endonasal conservative retrograde trans-sphenoidal approach (EECRTSA). (d) Funneling with microscope.

Figure 2



Case no. 3: (a) HRCT with contrast showing osteodural defect in the lateral recess of the left sphenoid sinus lateral recess (arrow) with cerebrospinal fluid (CSF) leak. (b) HRCT with contrast showing empty sella (asterisk). (c) MRI cisternography showing CSF leak in the left lateral recess of the sphenoid sinus (asterisk).

Table 1 Preoperative data of the patients

Case nos	Sex	Age	Side	Duration of symptoms	Presenting symptoms	BMI (kg/m ²)
1	F	51	RT	12	SCSFR/headache/vertigo/tinnitus	39.5
2	F	34	RT	6	SCSFR/headache/tinnitus/balance	34.3
3	F	49	LT	9	SCSFR/headache/tinnitus/visual/balance	38.3
4	M	44	LT	14	SCSFR/headache/meningitis	32.2
5	F	32	LT	5	SCSFR/headache/tinnitus/balance	38.4
6	M	30	LT	6	SCSFR/headache/visual	36.6
7	F	41	LT	8	SCSFR/headache/balance	35.3
8	F	46	LT	9	SCSFR/headache/tinnitus/balance	32.32
9	F	43	RT	10	SCSFR/headache/tinnitus/visual/balance	31.67
10	M	37	LT	11	SCSFR/headache/vertigo/tinnitus	36.90
Mean ± SD		40.7 ± 7.21		9 ± 2.87		35.55 ± 2.84

F, Female; L, left; M, male; Rt, right; SCSFR, spontaneous cerebrospinal fluid rhinorrhea.

Table 2 Intraoperative surgical variables of the patients

Case nos	Size of the defect (mm in diameter)	Operative time (min)	ICP (mm H ₂ O)
1	8	170	24
2	6	180	26
3	7	150	29
4	8	135	25
5	9.5	165	27
6	7	200	21
7	9	185	33
8	6	175	32
9	7.2	150	30
10	7.4	145	28
Mean ± SD	7.55 ± 1.14	165.5 ± 20.34	27.5 ± 3.84

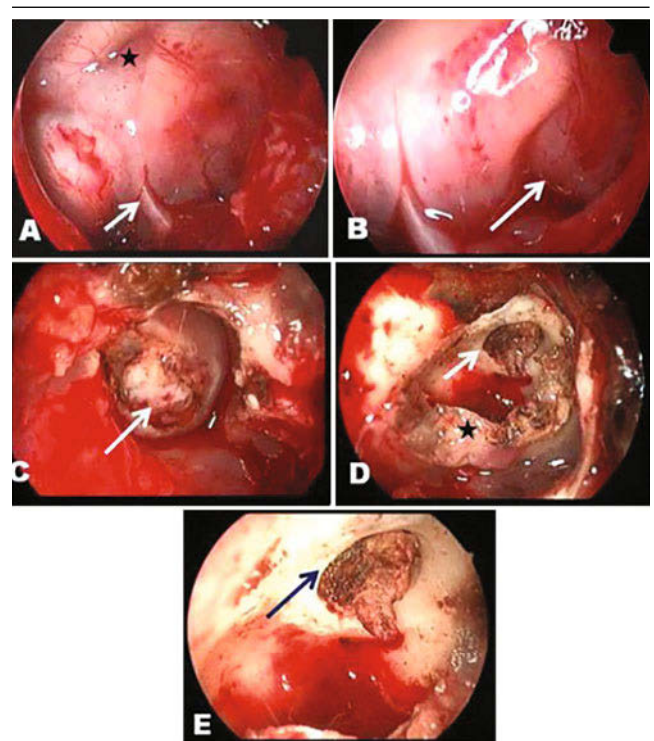
ICP, intracranial pressure.

data, stay in hospital, complications, follow-up period, recurrences, and the time of the postoperative CT and MRI. CT and MRI were performed at 6 months postoperatively. The lumbar drain was left for 5 days postoperatively while the patients were in the hospital. Antibiotics were administered for 2 weeks. One tablet (500 mg) of Diamox (Amdipharm Mercury Company, London, UK) (acetazolamide), a carbonic anhydrase inhibitor diuretic that crosses the blood–brain barrier, was given twice daily for 6–12 months as all patients had increased ICP. Every 3 months routine endoscopic examination was carried out throughout the follow-up period.

Surgical technique

Surgical technique followed was ‘endonasal endoscopic conservative retrograde trans-sphenoidal approach’ (retrograde approach) (Figs 1 and 3).

All patients were operated under total intravenous anesthesia. The anesthesiology team performed the procedure with the lumbar drain placed intrathecally in all patients in the operating room. The team obtained an opening pressure (in cm H₂O) immediately after insertion with a manometry attached to the lumbar

Figure 3

Intraoperative endoscopic view of the left sphenoid sinus with a 45° lens in case no. 3. (a) Carotico-optical recess (asterisk) and vidian canal (arrow). (b) Meningoceles in the lateral recess sphenoid sinus (arrow). (c) Meningoencephalocele after removal of the sphenoid sinus mucosa (arrow). (d) Osteodural defect (arrow) and elevated mucoperiosteum of sphenoid sinus (asterisk). (e) Close-up view of (d).

drain and zeroed at the spine (or external auditory canal) in the lateral decubitus position with the body level. Drains provide a means to measure the ICP directly intraoperatively and localize the site of the defect intraoperatively through saline/fluorescein (in two patients only) injected intrathecally (we have used a mixture of 0.1 ml of 10% fluorescein diluted in 10 ml of the patient’s CSF injected slowly over 10–15 min and have had no complications). Withdrawal of 5–10 ml of CSF during the ablation of the encephaloceles assists the retraction of the brain and facilitates the placement

Table 3 Postoperative data and outcome in the patients

Case nos	Hospital stay (days)	Recurrence	Complication (operative or postoperative)	Follow-up duration (months)	Time of postoperative/CT/MRI (months)
1	7	None	None	39	8
2	6	None	None	52	6
3	10	None	None	48	9
4	7	None	None	44	11
5	8	None	None	36	9
6	6	None	None	38	7
7	8	None	None	47	12
8	10	None	None	60	10
9	12	None	None	59	12
10	13	None	None	46	10
Mean \pm SD	8.7 \pm 2.59			46.9 \pm 8.26	9.4 \pm 2.01

CT, computed tomography.

of the multilayer grafts. It decreases the CSF pressure to stabilize the multilayer grafts in place postoperatively. We used circular bite forceps, Kerrison punch, and Citelli forceps. Intranasal angled drills are essential. Double-angled and malleable instruments facilitate the maneuvers. Surgical steps were performed with a 45° telescope lens from the beginning. Image-guided navigation was used in three cases only.

The first step is endonasal endoscopic bilateral paraseptal sphenoidotomy. Reset the superior turbinate totally on the ipsilateral side and the middle turbinate partially. Ipsilateral posterior ethmoidectomy is performed through the superior meatus (natural ostium), which aids in early identification of the medial orbital wall. Contralateral superior and middle partial turbinectomies are individualized to each case on the basis of the extent of sphenoidotomy. The sphenopalatine vessels are cauterized when encountered on the ipsilateral side. Resect the posterior 1 cm of the septum (perpendicular plate of the ethmoid bone and the vomer bone) and the rostrum of the sphenoid bone, allowing space for instrumentation, exposure of key anatomic landmarks, and four-hand and two-nostril surgery. Remove the ipsilateral anterior wall and inferior walls of the sphenoid sinus until the foramen rotundum and the vidian canal orifice appear clearly. The contralateral anterior and inferior walls are removed according to each case. The posterior limit of the dissection is now visible and identified: the carotico-optical recesses, the internal carotid artery, the sellar prominences, and the clivus (Fig. 3a).

Coming from the medial and posterior to the lateral and anterior, the area lateral to the foramen rotundum and the vidian canal orifice in the LRSS is visualized clearly with a 45° lens exposing blind angles and recesses that normally cannot be seen with a 0° or a 30° lens. Thus, a full visualization of the defect is achieved without the bone removal at the posterior wall of the maxillary sinus and the pterygoid process base through

tunneling (the advantages of the optical properties of the 45° angled telescope) and not through funneling as in microscopic surgery. In extreme pneumatization of the LRSS or the pterygoid process base, the retrograde subperiosteal piecemeal-tailored bone removal of the anterior wall of the LRSS can be performed with Kerrison forceps or double-angled instruments without jeopardizing important structures in the pterygopalatine fossa. Full visualization of the defect allows multilayer reconstruction and eliminates the need for sinus obliteration.

When fully exposing the meningoencephalocele (Fig. 3b), verify the presence of CSF at the osteodural defect by injecting saline or fluorescein in the lumbar drain and/or by applying pressure on the internal jugular vein. Remove the mucosa over the meningoencephalocele completely to prevent submucosal cyst formation by bipolar cautery (Fig. 3c). Reduce the meningoencephalocele by bipolar coagulation and amputate the rest until the bony defect in the skull base is fully exposed (Fig. 3d). Dissect the sinus mucosa by 1 cm around the defect to maximize the area of bony contact with graft and later use the mucosa as an augmentation layer (Fig. 3e). The bony defect was present constantly in the superior wall of the LRSS in all patients. The edges of the osteal defect were smooth, regular, and round (punched-out) suggesting a developmental origin, and not acquired (Fig. 3e). Smooth the surface of the bones around the defect to promote the nourishment and stability with a diamond burr. Administer mannitol intravenously intraoperatively before the reconstruction to reduce the CSF pressure before the proper placement of the multilayer graft. Dissect the dura from the brain and the cranium.

We advise multilayer reconstruction: first intracranial (subdural and extradural) and second extracranial. Fibrin glue after each layer acts as a watertight layer. Fat and fascia lata are placed subdurally as the first layer. Cartilage is placed extradurally and subcranially

(tragal/septal cartilage, tragal cartilage is our choice as it is one graft with three layers and was performed in five patients only and its greater diameter should be greater than that of bone defect by sufficient amount). A piece of bone and fascia lata was placed extracranially. Reflect the elevated sinus mucosa over the multilayered graft complex. The multilayer reconstruction graft complex is augmented by a layer of Surgicel (Ethicon Somerville, New Jersey, United States) and then gel foam for support and hemostasis in sinus without sinus obliteration.

Results

The present study comprised 10 patients, three male and seven female. The mean age of the patients was 40.7 ± 7.21 years and the presenting symptoms of the patients are presented in Table 1. The mean delay between the onset of the symptoms and the diagnosis was 9 ± 2.87 months. The mean BMI was 35.55 ± 2.84 kg/m² conforming that all patients were obese (Table 1).

Radiological findings revealed that all patients had primary empty sella (100%); two patients had the leak on the right side, whereas eight had on the left side. Meningoencephalocele was present in eight (80%) patients. Arachnoid pits were present in three (30%) patients and thinned/attenuated skull base was present in four (40%) patients. The site of the osteodural defect was in the superior wall of the LRSS, lateral to the foramen rotundum and the vidian canal, neither above the foramen rotundum nor below the vidian canal (Figs 2 and 3).

The mean time of the operation was 165.5 ± 220.34 min and the mean ICP was 27.5 ± 3.84 cm H₂O measured directly intraoperatively (Table 2). The mean size of the defect was 7.55 ± 1.14 mm and the shape of the defect was punched-out, smooth, regular, and rounded (Fig 3d and e).

There were no intraoperative complications. The mean hospitalization time was 8.7 ± 2.59 days. The mean follow-up period was 46.9 ± 8.26 months. There was no incidence of postoperative complications (Table 3). No recurrences occurred, which was confirmed with HRCT and MRI cisternography.

Discussion

Spontaneous CSF rhinorrhea with or without a meningoencephalocele in the LRSS is a diagnostic challenge as it may be occult (asymptomatic and or

diagnosed accidentally by radiology), or intermittent, or present by vague symptoms such as headache, vertigo, visual (blurring of vision with or without diplopia), and/or meningitis [20].

The evidence presented in this study lends strong support that the etiology is multifactorial. The chronically elevated ICP evidenced with the direct intraoperative measurement of lumbar drain pressure and the indirect radiological assessment of the presence of empty sella (100%), arachnoid pits (30%), and thinned/attenuated skull base (40%) in patients and the presence of a weak point in the skull base are the most important underlying causes of spontaneous CSF rhinorrhea with or without a meningoencephalocele in LRSS. Elevated ICP in the presence of a strong skull base may manifest as benign intracranial hypertension, whereas in the presence of a weak skull base it manifests as spontaneous CSF rhinorrhea with or without a meningoencephalocele [2]. Increased ICP may result from obesity [21] and the natural fluctuations or spikes of ICP [2,22–25]. The mean BMI in our patient population was greater than 30 kg/m² indicating obesity.

Weak point in the skull base may result from elevated ICP, excessive/or wide pneumatization, and abnormal ossification of the skull bones and at the sites of inherent structural weakness (i.e. the dura of the sellar diaphragm, perforations in the cribriform plate and adjacent to the natural foramina of the skull base). Elevated ICP exerts constant pulsatile pressure to the skull base, and over the time ultimately leads to the erosion and thinning of the skull base. It is also possible that patients, who manifest with meningoencephaloceles and/or spontaneous CSF leak, have a focal increase in ICP, in which arachnoid villi may develop into aberrant arachnoid granulations outside the dural venous sinuses, causing arachnoid pits that could eventually erode the skull base [2,12,25].

Wide pneumatization may result in thinning and dehiscence of the roof of LRSS and the greater wing of the sphenoid bone, and eventually may result in CSF leakage and herniation of the temporal lobe into the sphenoid sinus, especially if the underlying sphenoid bone is thin to begin with. At birth, when sphenoid sinus is not developed, and, in adults, in the absence of the lateral recess, spontaneous lateral sphenoid cephaloceles may present with seizures and/or headaches without rhinorrhea and/or can also be found incidentally and may result in diagnostic confusion [25]. Therefore, elevated ICP, obesity, and thinned skull base, all contribute in the etiology and pathogenesis of spontaneous CSF rhinorrhea and meningoencephalocele from the LRSS, similar to those reported in prior case studies [6,10,11,13,18].

The sphenoid bone has a complex ontogeny formed from multiple precursors (Fig. 4a). Nonfusion due to defective ossification and synchondroses can lead to osteodural defects at the skull base through several mechanisms [26]:

- (1) Persistence of craniopharyngeal canal [27,28];
- (2) Lateral craniopharyngeal canal (Sternberg's canal); and
- (3) Cartilaginous vascular channels at or near ossification centers of the sphenoid bone [26,29–31] (Fig. 4b).

In this study, the shape of the defects was regular, smooth, and rounded (punched-out), and the size of the defect was less than 10 mm in maximum diameter supposing it to be congenital and not acquired. All bony defects between the extracranial and intracranial space were constantly present in the superior wall of the LRSS and lateral to the foramen rotundum and the vidian canal. None of the defects were above the foramen rotundum or below the vidian canal, indicating that the potential source is not through Sternberg's canal. Sternberg [9] defined the canal to be medial to the foramen rotundum and inferomedial to the optic canal. Thus, our findings are in agreement with some studies [2,4,12], yet disagrees with others [7,10,11] who postulated that the canal is lateral to the foramen rotundum.

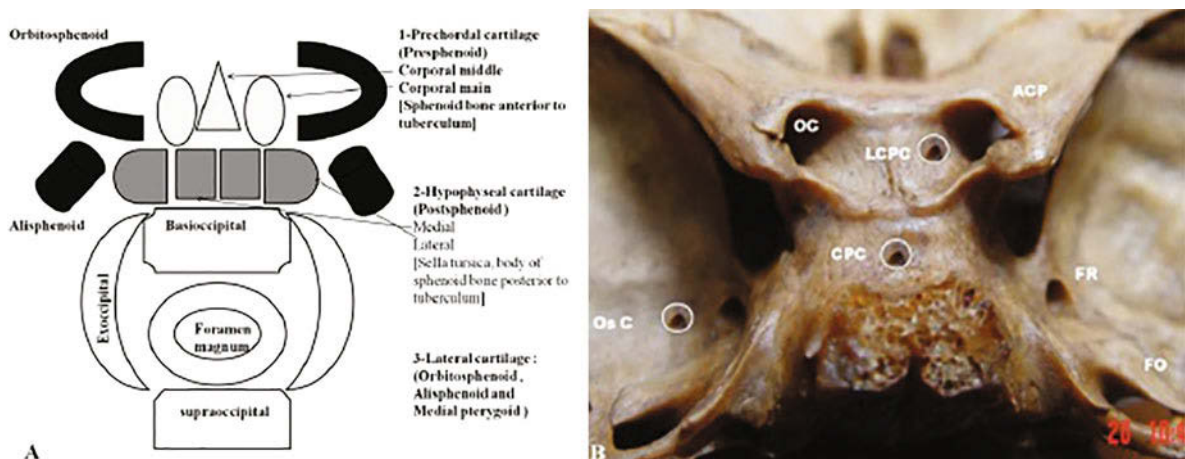
The ossification center of the greater wing of the sphenoid bone is the first center to ossify in the sphenoid bone at 9 weeks of intrauterine life and is located lateral to the foramen rotundum and anterior to the foramen oval [26,30–32]; the same specific site of the bony defect was reported in the present study. During development, cartilaginous vascular channels

appear at ossification centers whereby nutrients pass to the centers. These channels normally replace with bone during normal ossification. In defective ossification of the alisphenoid, these cartilaginous vascular channels are not filled with bone, but filled with fibrous tissue. This fibrous tissue acts as a weak point in the skull base, and in the presence of elevated ICP may lead to osteodural defect with or without meningoencephalocele in the LRSS. Thus, the most likely explanation for the osteodural defect is the persistence of cartilaginous vascular channels at or near the ossification center of the alisphenoid and not Sternberg's canal. Previous studies found that the site of the defect is located in the same site as found in the present study, but suggested it to be the result of aberrant arachnoid granulations [2,15,25,33].

Treatment of spontaneous CSF rhinorrhea with or without meningoencephalocele accomplishes two primary goals: prevents CSF leakages and repairs the osteodural defect to prevent ascending meningitis [15]. Endoscopic treatment increasingly replaces the transcranial or the extracranial techniques due to its advantages [5,23]. The transpterygoid approach (Table 4) is very challenging; in this technique, one has to open and skeletonize the pterygopalatine fossa by removing the posterior maxillary sinus wall and drilling the pterygoid process base, encountering the sphenopalatine and maxillary arteries, the maxillary nerve and the pterygopalatine ganglion [10,12,15].

We present our retrograde approach for the surgical management of spontaneous CSF rhinorrhea in the LRSS. Table 4 shows the main differences between the retrograde and the transpterygoid approach. Our procedure involved modifications to access the LRSS

Figure 4



(a) Diagram illustrating the precursors of the sphenoid bone. (b) Superior view of a dried skull showing the ossification center (OsC) of the greater wing of the sphenoid (alisphenoid), craniopharyngeal canal (CPC), Lateral craniopharyngeal canal (LCPC) or Sternberg's canal. The foramen rotundum (FR), foramen oval (FO), optic canal (OC), and anterior clinoid process (ACP).

Table 4 The main differences between the endonasal endoscopic transtethmoid–sphenoid pterygoid approach (transpterygoid) and the endonasal endoscopic conservative retrograde trans-sphenoid approach (retrograde)

Transpterygoid	Retrograde
Unilateral sphenoidotomy	Bilateral wide sphenoidotomy
Complete ethmoidectomy	Ipsilateral posterior ethmoidectomy through superior meatus natural ostium
Middle meatal antrostomy	No
Removal of the posterior wall of the antrum	No
Entry into pterygopalatine fossa	No
Removal of the pterygoid process base	No
—	Resection of posterior 1 cm of the nasal septum for binostril and four-hand surgery
—	45° lens to visualize blind angles and recesses
Routine anterior wall removal	Subperiosteal removal of anterior wall of the lateral recess may be performed in cases of extreme pneumatization

without entering the pterygopalatine fossa through a wide extension of the sphenoidotomy and the use of a 45° lens from the start. The near-total removal of the anterior and the inferior walls of the sphenoid sinuses achieves wide sphenoidotomy. This helps in the early identification of the posterior limit of our dissection (clivus, sellar prominences, internal carotid arteries, and carotico-optical recesses) and the important vital structures (maxillary nerve, vidian canal, and optic nerve), and the medial orbital wall. The alea of the vomer, rostrum of the sphenoid, and the posterior 1 cm of nasal septum are removed for two-nostril and four-hand surgery. Here, the surgeon approaches the lateral recess from the medial to the posterior through the sphenoid sinus (retrograde) and not from anterior as in the transpterygoid approach through the posterior wall of the maxillary sinus (Table 4). A 45° lens is used to visualize the blind recesses and angles lateral to the foramen rotundum with minimal bone removal. Posterior ethmoidectomy through natural ostium is performed to identify the medial orbital wall early. We agree with many reports that for achieving a reliable exposure of the LRSS, it is not always necessary to remove the pterygoid process completely [1,10,11].

The retrograde approach allows the multilayer reconstruction of the defect under full vision without the need for sinus obliteration. It decreases the amount of the bleeding and the time of surgery. On avoiding the anterior drilling, the pterygoid process base creates a smaller cavity, which helps in less crustations, earlier healing, and minimal affection of function.

In extensive pneumatization of the LRSS, minimal, tailored retrograde subperiosteal removal of the anterior wall of the LRSS may be performed on the basis of the site of the bony defect and the extent of LRSS pneumatization. We used multilayered reconstruction of osteodural defect, which allows good control of the leak with minimal morbidity because of the associated elevated ICP; this is in agreement with the findings of [10]. Tragal cartilage has advantages of being three layers in one graft (perichondrium, cartilage, and perichondrium).

Although our average follow-up is for 46.9 ± 8.26 months in this series, it must be pointed out that future failures are possible if intracranial hypertension is not effectively managed. Continued management of the intracranial hypertension will result in better outcomes.

Conclusion

Management of spontaneous CSF rhinorrhea with or without meningoencephalocele in the LRSS is surgically challenging. The etiology is multifactorial. Elevated ICP is the most important etiologic factor and is crucial in the management and the prevention of recurrences. The potential source is not Sternberg's canal but persistent cartilaginous vascular channel at the ossification center of the alisphenoid, as the site of the defect was specifically and constantly present at the site of the ossification center of the greater wing of the sphenoid bone. The retrograde approach is safe and effective. It utilizes the benefits of the optical properties of the 45° lens to access and visualize the blind recesses and angles of the LRSS with minimal bone removal through tunneling and not funneling. Thus, one can achieve early functional healing, shorter operative time and hospital stay, yet experience with the use of the 45° lens is needed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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