Endonasal Management of Skull Base Defects: Meningoceles and Meningoencephaloceles

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ABSTRACT
Introduction: Meningoceles and menigoencephaloceles result from herniation of the meninges, with or without brain tissue, through defects in the skull base.
Objectives: To determine the success rate of skull base repair for meningoceles and menigoencephaloceles using an endonasal approach assisted by endoscopes, and to establish an algorithm for the preoperative topographic diagnosis of these lesions.
Methods: Patients treated for skull base meningoceles and menigoencephaloceles using anendonasal approach assisted by endoscopes between January 2010 and March 2024 wereincluded. Reconstruction of the defect was performed using a multilayer technique withautologous grafts and local flaps.
Results: Ten patients were treated: nine with meningoceles and one with a meningoencephalocele. Three were located in the sphenoid sinus, six in the cribriform plate of the ethmoid bone, and one in the frontal sinus.
The success rate we achieved in repairing the skull base defect was 90%.
Conclusions: The success rate we achieved in repairing skull base defects caused by meningoceles and meningoencephaloceles using an endonasal approach was 90%.
Computed tomography and contrast-enhanced magnetic resonance imaging (MRI) with a protocol to visualize the olfactory bulb were very useful in diagnosing the site of the lesions at the level of the cribriform plate of the ethmoid bone.

Keywords: meningoceles, meningoencephaloceles, endoscopic surgery, skull base reconstruction.

Manejo endonasal de defectos de la base del cráneo: meningoceles y meningoencefaloceles
RESUMEN
Introducción: los meningoceles y meningoencefaloceles se producen por la hernia de las meninges con o sin tejido encefálico a través de defectos en la base de cráneo. El objetivo fue determinar la tasa de éxito de la reparación de la base del cráneo por meningoceles y menigoencefaloceles mediante un abordaje endonasal asistido con endoscopios, y establecer un algoritmo para el topodiagnóstico preoperatorio de estas lesiones.
Métodos: se incluyeron los pacientes que fueron tratados por meningoceles y menigoencefaloceles de base de cráneo mediante un abordaje endonasal con endoscopios, entre enero de 2010 y marzo de 2024. La reconstrucción del defecto se realizó con técnica multicapa con injertos autólogos y colgajos locales.

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Resultados: fueron tratados 10 pacientes, 9 con meningoceles y uno con un meningoencefalocele. Tres se localizaron en el seno esfenoidal, seis en la lámina cribosa del etmoides y uno en el seno frontal. La tasa de éxito que obtuvimos en la reparación del defecto en la base del cráneo fue 90%.
Conclusiones: la tasa de éxito que tuvimos en la reparación de la base del cráneo por defectos ocasionados por meningoceles y meningoencefaloceles utilizando un abordaje endonasal fue 90%.
La tomografía computarizada de macizo facial y la resonancia magnética con contraste con protocolo para visualizar el bulbo olfatorio resultó muy útil para diagnosticar el sitio de las lesiones a la altura de la lámina cribosa del etmoides.

Palabras clave: meningoceles, meningoencefaloceles, cirugía endoscópica, reconstrucción de base de cráneo.

INTRODUCTION

Meningoceles (MC) and meningoencephaloceles (MEC) are protrusions of the meninges, with or without brain tissue, through a bony defect of the skull base into the nasal cavity or paranasal sinuses, forming a cystic lesion containing cerebrospinal fluid (CSF).

The brain tissue is usually nonfunctional.

Skull base defects may be congenital or acquired, including causes such as elevated intracranial pressure, trauma, expansive tumors, or iatrogenic injury related to surgical procedures.

The treatment of these lesions aims to repair the bony defect in order to prevent the risk of meningitis. Several surgical approaches have been described for the management of MC and MEC.

Endonasal endoscopic access using a multilayer reconstruction technique with grafts or local flaps is currently considered the approach of choice, given its high success rates in defect repair and its low morbidity.

OBJECTIVES

To determine the success rate of skull base repair for idiopathic meningoceles and meningoencephaloceles using an endonasal endoscopic approach, and to establish an algorithm for the preoperative topographic diagnosis of these lesions.

STUDY DESIGN

Descriptive and retrospective.

MATERIALS AND METHODS

All patients treated for skull base MC and MEC using an endonasal endoscopic approach between January 2010 and March 2024 were included. The diagnosis of cerebrospinal fluid leak was confirmed intraoperatively in half of the patients and, in the remaining cases, by preoperative determination of β-trace protein.

All patients underwent nasal endoscopy, computed tomography (CT) of the facial skeleton, and contrast-enhanced magnetic resonance imaging (MRI) of the facial skeleton. In cases in which CT did not clearly demonstrate a bony defect of the skull base, MRI was performed using a protocol designed to visualize the olfactory bulb (T2 fast spin-echo sequences with fat suppression and high-resolution isotropic 3D T2 sequences, allowing multiplanar reformatting and optimizing contrast between cerebrospinal fluid and the olfactory pathway).

Surgical procedures consisted of exposing the lesions through an endonasal endoscopic approach and reducing them using bipolar forceps. The mucosa surrounding the bony defect was then resected, followed by multilayer reconstruction using autologous grafts of septal or turbinate mucoperiosteum and fat harvested from the earlobe, or local flaps from the middle turbinate, inferior turbinate, or nasoseptal region. A biological adhesive (fibrin glue) or a dural sealant composed of a synthetic hydrogel with polyethylene glycol and polyethyleneimine (Adherus®) was applied as the final reconstructive layer, followed by absorbable gelatin sponge (Spongostan®) or hemostatic matrix (Surgiflo™), and a glove finger containing hydroxylated polyvinyl acetate sponge (Merocel®) was used as supportive nasal packing for three days.

Patients remained hospitalized for three days until removal of the nasal packing.

The endoscopes used were 0°, and standard instrumentation for endoscopic sinonasal surgery was employed. Neuronavigation was not used.

The study protocol was approved by the Institutional Ethics Committee of the Hospital Italiano de Buenos Aires (approval No. 7485; Priisa file number: 16207) and was conducted in accordance with the principles outlined in the amended Declaration of Helsinki.

RESULTS

Ten patients were treated: nine with MC and one with MEC.

Eight were women and two were men.

Eight patients presented with intermittent cerebrospinal fluid fistulas.

The etiology of the skull base defects was idiopathic, as none of the patients had a history of trauma, prior surgery, or other lesions involving the nasal cavity or paranasal sinuses.

Three lesions were located in the sphenoid sinus: two on the lateral wall and one in the sphenoid planum.

Intrathecal fluorescein was used in one patient (10 mL of cerebrospinal fluid were withdrawn via lumbar puncture and mixed with 0.2 mL of 5% fluorescein; the solution was injected at a rate of 1 mL per minute).

The surgical approach consisted of bilateral sphenoidotomy in two patients and a transpterygoid approach with sphenoidotomy in one patient.

Multilayer reconstruction using grafts was performed in all cases. A lumbar drain was placed in one patient due to elevated intracranial pressure.

In one patient, repair failed at four months and was successfully managed with temporal craniotomy and reconstruction of the defect (Fig. 1).

Six patients had MC located at the cribriform plate of the ethmoid bone. In five of these six patients, topographic diagnosis of MC and cerebrospinal fluid leak was confirmed by MRI using an olfactory bulb-specific protocol. Cerebrospinal fluid leakage resolved in all cases (Fig. 2).

One patient presented with a MEC in the left frontal sinus and was successfully treated using an endonasal Draf III frontal sinus approach. The defect was repaired using a multilayer technique (Fig. 3).

The overall success rate for skull base defect repair was 90% (Table 1).

DISCUSSION

The etiology of meningoceles (MCs) and meningoencephaloceles (MECs) may be traumatic, iatrogenic, congenital, or idiopathic. The latter has been associated with a possible increase in intracranial pressure (>25 cm H_2O). Although the pathophysiology is not fully understood, it has been proposed that venous hypertension may impair cerebrospinal fluid (CSF) reabsorption at the level of the arachnoid villi.

CSF overproduction may also contribute to increased intracranial pressure.[1]

Skull base defects are thought to result from progressive thinning of the bone in areas adjacent to the pulsatile dura mater, venous sinuses, or arachnoid granulations, which repeatedly impact the bone and eventually lead to dehiscence, meningocele formation, and CSF fistulas.

These lesions are more frequently observed in obese women –possibly due to impaired cerebral venous return associated with increased abdominal adiposity[2]–and in patients with sleep apnea, in whom hypoventilation leads to hypercapnia, cerebral vasodilation, and venous hypertension[3].

In our series, women predominated (8/10), and six patients were obese (body mass index >30).

Dural venous sinus stenosis has also been associated with elevated intracranial pressure[4].

Skull base defects and herniation of dural and encephalic tissue occur more frequently in areas of intrinsic bony weakness, such as the ethmoid roof, cribriform plate, olfactory cleft, and the lateral recess of the sphenoid sinus (lateral to the vidian nerve canal and foramen rotundum)[5-7]. In our series, lesions were more commonly located at the level of the cribriform plate/olfactory cleft (6/10).

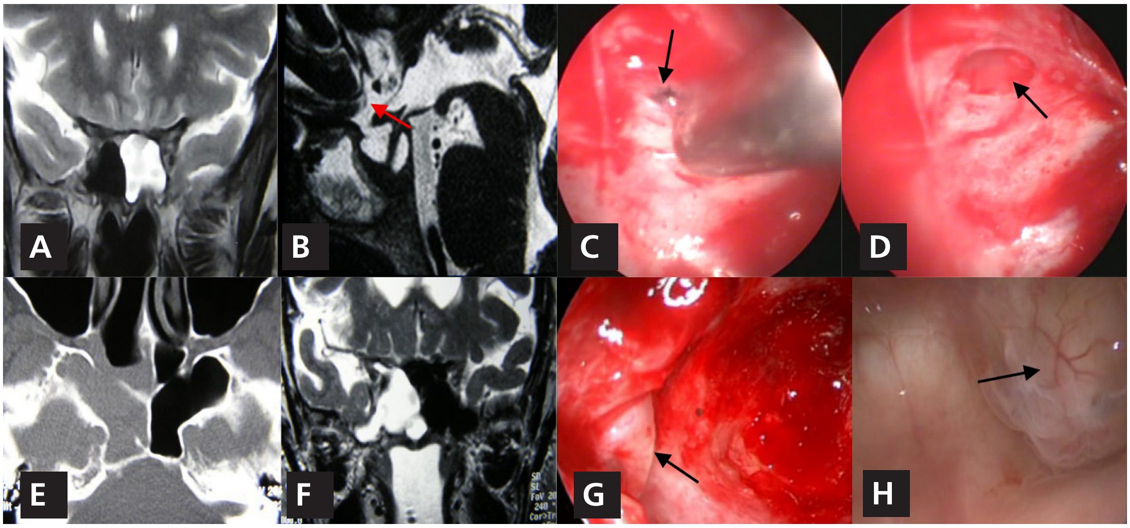

Figure 1. Meningoceles located at the sphenoid planum and sphenoid sinus with high-flow cerebrospinal fluid (CSF) leak. *Sphenoid planum meningocele.* (A) MRI showing accumulation of CSF in the left sphenoid sinus. (B) Sagittal MRI demonstrating CSF leakage with contrast through the defect in the sphenoid planum (arrow). (C) Endonasal endoscopic view after sphenoidotomy and bipolar reduction of the meningocele (arrow). (D) Osseous defect after resection of the surrounding mucosa (arrow). *Right lateral sphenoid sinus meningocele.* (E) CT scan showing laterally pneumatized and opacified right sphenoid sinus. (F) Occupation of the right sphenoid sinus by CSF. (G) Lateral sphenoid meningocele (arrow). (H) Sphenoid meningocele located on the left lateral wall of the sphenoid sinus (arrow).

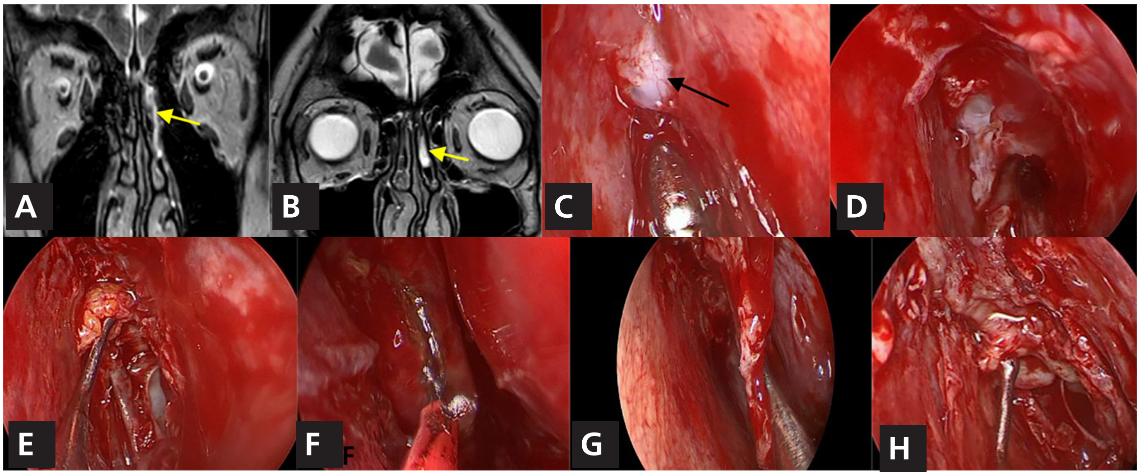

Figure 2. Meningocele located at the level of the ethmoidal roof. (A, B) MRI with an olfactory bulb visualization protocol, showing CSF leakage (arrow) and accumulation within the turbinate (arrow). (C) Endonasal endoscopic view of the ethmoidal meningocele (arrow). (D) Endoscopic view after bipolar reduction of the meningocele. (E) Placement of autologous fat in the intracranial/extradural space. (F) Dissection of the middle turbinate flap using a monopolar scalpel and resection of its medial half. (G) Rotation of the lateral half of the turbinate toward the defect. (H) Repair of the skull base defect using the rotated middle turbinate flap.

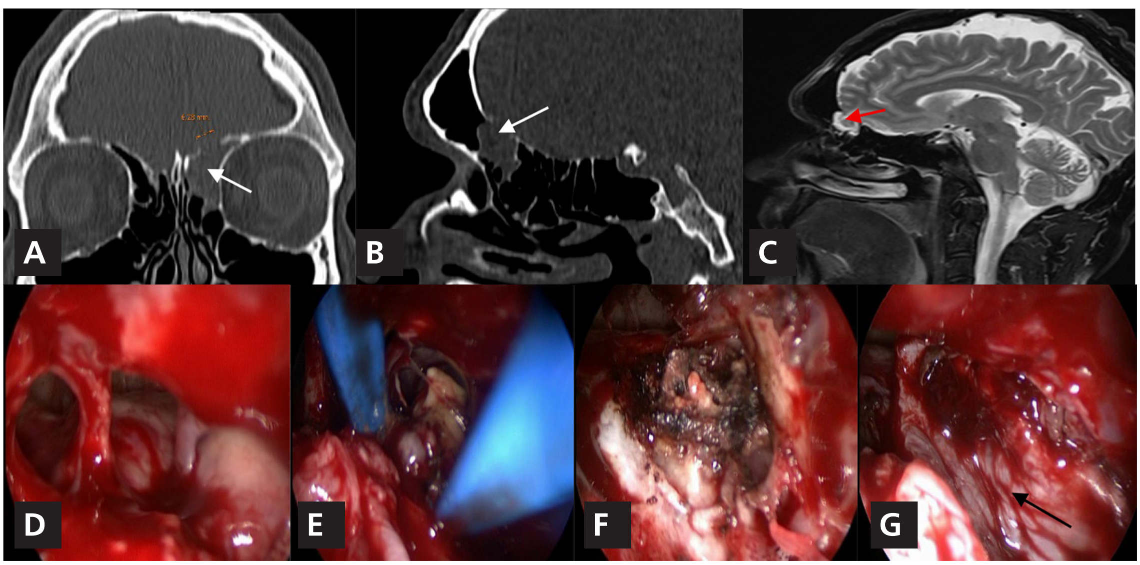

Figure 3. Meningoencephalocele of the left frontal sinus. (A, B) Computed tomography, coronal and sagittal views, showing a bony defect with protrusion of brain tissue (arrows). (C) MRI demonstrating the meningoencephalocele within the frontal sinus (arrow). (D) Exposure of the meningoencephalocele through an endonasal endoscopic approach extended to the frontal sinus (Draf III). (E) Reduction of the meningoencephalocele using bipolar forceps. (F) View of the defect to be repaired. (G) Multilayer reconstruction using a nasoseptal flap (arrow).

In some patients, multiple skull base defects may coexist[8].

The diagnosis of a CSF fistula has two main objectives: to determine whether watery rhinorrhea corresponds to CSF and to establish the precise topographic location of the skull base defect responsible for the leak.

The first objective can be achieved by measuring β-trace protein levels, which are present in CSF and can be quantified in serum and urine. Alternatively, β_2-transferrin can be measured in nasal secretions.[9]

Using these assays, as little as 5 µL and 100 µL of CSF per 1 mL of nasal secretion can be detected by β-trace protein and β_2-transferrin measurement, respectively[10].

β_2-transferrin testing has a sensitivity and specificity exceeding 90%, whereas β-trace protein measurement is faster and more cost-effective, with higher concentrations

Table 1. Patients treated using an endonasal endoscopic-assisted approach for skull base meningoceles and meningoencephaloceles

Age	Sex	Localization	Lesion type and size	Surgical reconstruction	Lumbar drain	Outcome	Follow-up
64	F	Lateral sphenoid sinus	Meningocele (10 mm)	TMultilayer technique (failed after two endonasal surgeries; subsequently closed via craniotomy)	no	no	4 months (CSF leak resolved after craniotomy); no recurrence at 84
59	F	Lateral sphenoid sinus	Meningocele (15 mm)	Periosteal graft over sphenoid fat	no	sí	months10 months
54	F	Planum sphenoidale	Meningocele (0,8 mm)	Intracranial/extradural cartilage + intradural cartilage	yes	sí	8 months
48	F	Frontal sinus	Meningoencefalocele	Intracranial/extradural cartilage + nasoseptal flap	no	sí	7 months
62	F	Left cribriform plate	Meningocele (0.8 mm)	Intracranial/extradural fat + middle turbinate flap	no	sí	80 months
27	M	Right cribriform plate	Meningocele (0.7 mm)	Intracranial/extradural fat + lateral nasal wall flap	no	sí	10 months
34	F	Left cribriform plate	Meningocele 0.8 mm)	Intracranial/extradural fat + middle turbinate flap	no	sí	15 months
37	F	Right cribriform plate	Meningocele (0.7 mm)	Intracranial/extradural fat + middle turbinate flap	no	yes	46 months
52	F	Left cribriform plate	Meningocele (0.8 mm)	Intracranial/extradural fat + middle turbinate mucoperiosteal graft	no	yes	12 months
65	F	Left cribriform plate	Meningocele (0.8 mm)	Intracranial/extradural fat + middle turbinate flap	no		6 months

in CSF than in serum, making it an excellent marker of CSF leakage (sensitivity >90% and specificity of 100%)[11].

In four of the patients included in this series, the presence of CSF in rhinorrhea was confirmed preoperatively by β-trace protein measurement.

This diagnostic test was not available in our country prior to 2015.[12]

Localization of the fistula site is performed by outpatient nasal endoscopy (the fistula site was identified in only one patient with a frontal meningoencephalocele) and by imaging studies.

Non-contrast maxillofacial computed tomography (CT) with thin sections can identify bony defects at the level of the ethmoidal roof, sphenoid, or frontal sinus, a fluid level within the sphenoid sinus due to cerebrospinal fluid (CSF) accumulation, or herniation of the meninges into the nasal cavity. When CT failed to identify the fistula site (most frequently at the level of the cribriform plate), contrast-enhanced magnetic resonance imaging (MRI) using a protocol designed to evaluate the olfactory bulb proved to be essential. Comparison of signal characteristics on FLAIR and CISS sequences may help differentiate CSF (hyperintense on CISS and hypointense on FLAIR) from inflammation or edema (hyperintense on both CISS and FLAIR)[13,14] (Fig. 4A-C).

DIAGNOSTIC ALGORITHM FOR NASAL CEREBROSPINAL FLUID LEAK AND TOPOGRAPHIC LOCALIZATION OF SKULL BASE DEFECTS

Imaging studies may also identify indirect signs of intracranial hypertension, including an empty sella, dilated lateral ventricles, increased tortuosity of the optic nerve, and distension of the perioptic subarachnoid space (visualized as a wide ring of cerebrospinal fluid surrounding the optic nerve)[15]. None of the patients described in this series showed indirect imaging signs of elevated intracranial pressure.

In nine patients, the probable site of the meningocele or meningoencephalocele was identified preoperatively. In only one patient with a history of prior open surgeries in whom the defect could not be localized, preoperative intrathecal fluorescein injection was used. However, this was ultimately unnecessary, as sphenoidotomy allowed straightforward identification of the meningocele on the lateral wall of the sphenoid sinus.

The endonasal endoscopic approach is the surgical technique of choice for the treatment of MCs and MECs with sinonasal extension. There is consensus that skull base defects should be repaired as early as possible[16,17]. Reduction of the MC/MEC using bipolar forceps and resection of the mucosa surroundingthe bony defect are common steps in all procedures.

Multilayer reconstruction is preferred whenever feasible. At the level of the cribriform plate, placement of an intradural intracranial graft is technically challenging, as it may injure olfactory nerve fibers and enlarge the defect. Reconstruction of defects smaller than 2 cm and with low-flow CSF leaks may be performed using autologous grafts –preferably harvested from the contralateral septal mucoperiosteum or the middle turbinate– with outcomes comparable to those achieved using local flaps.

When the defect is located in the ethmoid region, we prefer the use of a bipedicled middle turbinate flap; for other locations, a nasoseptal flap or a lateral nasal wall flap with either anterior or posterior pedicle is preferred. As the first reconstructive layer (intracranial-extradural), we favor the use of fat harvested from the abdomen or the ear lobule.

The use of lumbar drainage in idiopathic CSF fistulas remains controversial. In an international consensus on the management of idiopathic CSF leaks, 59% of experts agreed that lumbar drainage may be necessary to allow preoperative fluorescein injection, measure intracranial

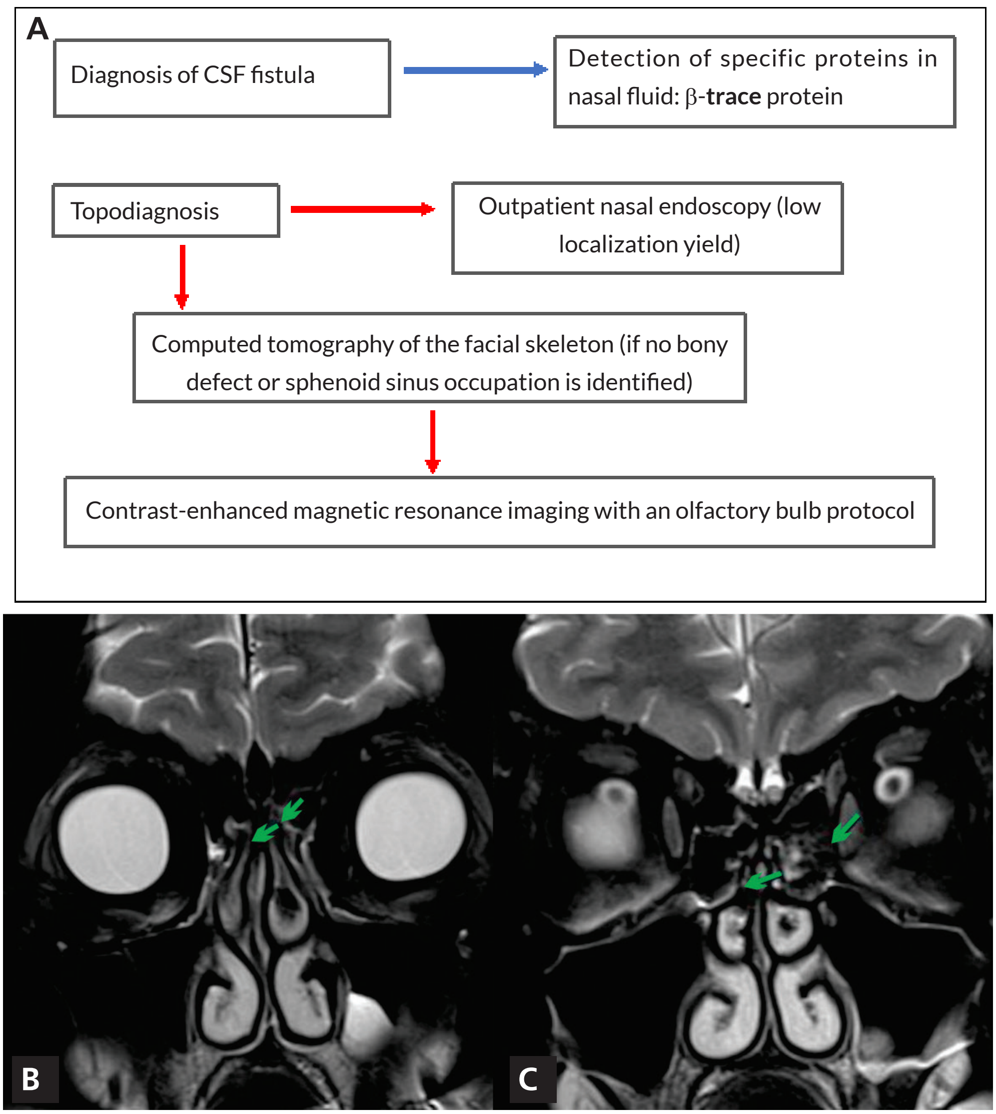

Figure 4. A. Diagnostic algorithm for skull base defects. B, C. Magnetic resonance imaging with an olfactory bulb protocol, showing cerebrospinal fluid leakage and accumulation in the left anterior ethmoidal cells (between green arrows

pressure, and reduce it after skull base repair[10]. Another review reported that lumbar drainage was avoided whenever possible, except in cases of high-flow leaks and revision surgeries[18].

In a prospective study of 150 patients, lumbar drainage was placed intraoperatively in 50% of cases undergoing CSF leak repair. The success rate was 77% for spontaneous fistulas versus 96-97% for iatrogenic and traumatic fistulas. The authors concluded that lumbar drainage did not reduce recurrence rates in patients with elevated intracranial pressure[19]. Other studies have similarly suggested that lumbar drainage should not be routinely used in all CSF leaks[20,21].

In our series, postoperative lumbar drainage was used in one patient with a meningocele located at the sphenoid roof who presented with elevated intracranial pressure and a high-flow leak. We believe that lumbar drainage may be useful in cases of high-flow fistulas, revision surgeries, and when imaging demonstrates signs of intracranial hypertension.

A Medline review including 1,178 patients compared outcomes of skull base repair for MCs and MECs using an endonasal endoscopic approach versus an external approach. No significant differences were found in repair success rates (90%) between the two techniques. However, complications were significantly lower in patients treated via the endonasal route (meningitis: 1.1% vs. 3.9%; wound infection or abscess: 0.7% vs. 6.8%; sepsis: 0% vs. 3.9%). Perioperative mortality was also lower in the endonasal group (0% vs. 1.4%). The authors concluded that the endonasal endoscopic approach is a safe and effective technique[22].

In one study, 17 patients with meningoencephaloceles of the anterior cranial fossa and parasellar region were treated endoscopically. In 15 patients, repair was successful after the first procedure; in two patients with persistent fistulas associated with hydrocephalus, a second intervention and ventriculoperitoneal shunt placement were required[23].

In another study including 141 patients treated for meningoceles or meningoencephaloceles via an endonasal endoscopic approach, a 96.5% success rate in skull base repair was reported[24]. The success rate achieved in our series (90%) was comparable to those previously reported in the literature.

Additional measures that may complement surgical repair in order to prevent recurrence include weight reduction in obese patients, treatment of obstructive sleep apnea, and placement of venous sinus stents, most commonly in the sigmoid and transverse sinuses[25].

CONCLUSIONS

The success rate achieved in the repair of skull base defects caused by meningoceles and meningoencephaloceles using an endonasal approach was **90%**.

Computed tomography of the facial skeleton and contrast-enhanced magnetic resonance imaging with an olfactory bulb protocol proved to be highly useful in identifying lesion sites at the level of the ethmoid cribriform plate.

Author Contributions: Conceptualization, Methodology, Formal analysis, Investigation, Original draft writing, Writing-review and editing (FFL, CS, VR, CSR).

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