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Management of Multiple Spontaneous Nasal Meningoencephaloceles

Rodney J. Schlosser, MD; William E. Bolger, MD

Objectives/Hypothesis: **Multiple spontaneous nasal meningoencephaloceles in the same patient are rare lesions. Although many skull base defects occur after prior trauma or surgery, otolaryngologists must be aware of the potential for spontaneous encephaloceles. We present our experience with this unusual condition and discuss its pathophysiology and unique management issues.** ***Study Design:*** **Retrospective.** ***Methods:*** **Review of medical records, radiographic images, and cerebrospinal fluid pressures.** ***Results:*** **We identified 5 patients with multiple, simultaneous, spontaneous encephaloceles: 4 patients with 2 encephaloceles and 1 patient with 3 encephaloceles (11 in all). Locations of the 11 encephaloceles were sphenoid lateral recess (6), frontal sinus with supraorbital ethmoid extension (2), ethmoid roof (1), frontal sinus (1), and central sphenoid (1). Three patients had bilateral sphenoid lateral recess encephaloceles, accounting for all six in that location. All four patients with available radiographic studies demonstrated empty sella turcica. Surgical approaches included endoscopic transpterygoid approach to the lateral sphenoid recess (3), endoscopic approach to ethmoid and central sphenoid (3), and osteoplastic flap with frontal sinus obliteration (2). We had 100% success at latest endoscopic follow-up (mean period, 17 mo). Three patients had postoperative lumbar punctures with mean cerebrospinal fluid pressure of 28.3 cm water (range, 19–34 cm; normal range, 0–15 cm).** ***Conclusions:*** **Multiple spontaneous encephaloceles can be managed safely and successfully using endoscopic and extracranial approaches. A high index of suspicion for this diagnosis must be maintained, especially in patients with radiographic evidence of laterally pneumatized sphenoid sinuses or empty sella. Spontaneous encephaloceles and cerebrospinal fluid leaks represent a form of intracranial hypertension.** ***Key Words:*** **Spontaneous encephalocele, endoscopic sinus surgery, cerebrospinal fluid leak, empty sella.**

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INTRODUCTION

Nasal *meningoencephaloceles* (often shortened to *encephalocele* or *cephalocele*) are herniations of the meninges and brain parenchyma into the nose. Because of the associated skull base defect and cranionasal fistula, cerebrospinal fluid (CSF) leaks and the risk of meningitis can accompany the condition when the meningeal layer is breached.

Encephaloceles are uncommon and can arise from congenital, traumatic, or spontaneous origins. More experience has been reported with CSF leaks because they represent a smaller cranionasal fistula and are more common than encephaloceles. Most CSF leaks are caused by skull base fractures or surgical trauma. Spontaneous or nontraumatic encephaloceles or CSF leaks have been the least common in most series, accounting for only 3% to 5% of all CSF leaks.[1–4] Historically, spontaneous cranionasal fistulas were divided into high-pressure leaks, typically caused by an intracranial tumor and hydrocephalus, and otherwise idiopathic leaks thought to be in the normal-pressure category. Our experience is that these spontaneous, idiopathic encephaloceles and CSF leaks are probably a result of elevated CSF pressures exerting hydrostatic forces on anatomically fragile areas of the skull base.

Extracranial endoscopic repair of encephaloceles and CSF leaks has been shown to have a high success rate without the morbidity of traditional intracranial approaches.[2–4] Patients with spontaneous fistulas represent a unique patient population because of the underlying pathophysiology of their condition and the overall attenuation of their skull base. We present our series of patients with multiple spontaneous nasal encephaloceles and discuss diagnosis, management, and probable pathophysiology of the condition.

MATERIALS AND METHODS

Patient Data

We retrospectively reviewed medical records and imaging studies of all patients with multiple spontaneous nasal meningoencephaloceles and collected demographic and treatment information.

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From the Department of Otorhinolaryngology—Head and Neck Surgery, University of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.

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Send Correspondence to William E. Bolger, MD, Department of Otorhinolaryngology, University of Pennsylvania, 5 Ravdin/Silverstein, 3400 Spruce Street, Philadelphia, PA 19104, U.S.A. E-mail: william.bolger@uphs.upenn.edu

Preoperative Management

All patients were evaluated with preoperative computed tomography (CT) and magnetic resonance imaging (MRI) scans to assist in localization of the skull base defects. When clear rhinorrhea was present, nasal drainage was sent for β-2 transferrin analysis to confirm the diagnosis. Additional radiographic studies such as radioactive cisternograms or CT cisternograms were obtained when additional information was needed to identify the sites of the encephaloceles. Preoperative prophylactic antibiotics were not used.

Lumbar Drain

All procedures were started by placing a lumbar drain before the induction of general anesthesia. This was performed with the patient awake and in a sitting position to fill the lumbar cistern with CSF. Prior experience placing lumbar drains with the patient in the decubitus position has shown us that, at times, patients with long-term CSF leaks can have a relative depletion of CSF, and it can be difficult to withdraw CSF and subsequently place the drain. Intrathecal fluorescein (0.1 mL 10% solution diluted in 10 mL CSF) was slowly injected through the spinal needle just before drain insertion. This aids in precise identification of the encephalocele and associated bony defect. Use of fluorescein also assists in confirmation of watertight closure at the end of the procedure. Informed consent for the use of intrathecal fluorescein was obtained in all patients. The risks, benefits, and lack of U.S. Food and Drug Administration (FDA) approval for intrathecal use were disclosed.

Once the drain is in place, it is clamped and general anesthesia is induced. Care is taken during induction to avoid masking the patient and using positive-pressure ventilation because this carries the risk of causing pneumocephalus through the cranionasal fistula. Once the skull base defect is exposed, just before bone graft placement, 15 to 25 mL CSF is removed over a 15-minute period to aid in reduction of the encephalocele. The lumbar drain is kept open and draining 5 to 10 mL per hour for the remainder of the procedure and for the initial postoperative period. This is especially useful in avoiding large spikes in intracranial pressure (ICP) should the patient cough or strain during extubation or have nausea and vomiting during the postoperative period.

Diligent care of the lumbar drain is required during the immediate postoperative period. The drain should be kept open at all times to prevent elevations in ICP from pushing against the graft and dislodging it. Adjustments in the height of the drain are used to keep the drainage rate between 5 and 10 mL CSF per hour. If the drain height is too low, an excessive amount of CSF can be removed, leading to intracranial herniation. Conversely, if the drain height is too high, air may flow retrograde into the intrathecal space and lead to pneumocephalus. Nursing staff must understand that the rate of drainage is to be adjusted by changing the height of the drain, not by clamping it temporarily.

Surgical Techniques

Patients were treated with a variety of surgical techniques based on the location of their encephaloceles as outlined in Table I. All skull base defects were repaired using a multilayer closure consisting of cortical bone grafts (typically, mastoid bone) and fascia grafts as described later in this section.

Transnasal endoscopic repair was performed as previously described,[3] whenever possible, depending on the location of the encephalocele. The endoscopic approach frequently requires a total sphenoethmoidectomy and possible middle turbinectomy to properly identify and expose the skull base defect. All endoscopic procedures are started by topical decongestion with oxymetazoline (0.05%), followed by injections using 1% lidocaine with 1:100,000 epinephrine. Injections are performed in the lateral nasal wall, turbinates, and transoral and transnasal sphenopalatine blocks, depending on the site of the encephalocele and the approach needed. The nose is irrigated with clindamycin solution to decrease bacterial contamination within the operative field. Encephaloceles are reduced or amputated using monopolar and bipolar cautery. Meticulous hemostasis is required, to avoid intracranial hemorrhage. Once the encephalocele is adequately reduced and several millimeters of mucosa surrounding the bony defect is removed, reconstruction of the defect is started. A postauricular approach is used to harvest a thick layer of temporalis fascia and the outer layer of cortical bone overlying the mastoid air cells. The repair is performed by gently elevating the dura above the bony skull base defect and then using the bone graft in an underlay fashion. This requires great care in bone graft design and placement because the entire skull base in these patients is attenuated and can easily fracture or break off, creating an even larger defect. In addition, these defects often have complex three-dimensional configurations, requiring precise sculpting of the bone graft. Once the underlay bone graft is of satisfactory size and shape, it is soaked in clindamycin solution to minimize any intracranial seeding of bacteria. After positioning the bone graft, the temporalis fascia is placed in an overlay fashion. The fascia graft is intentionally kept thick, in contrast to the thin fascia graft used for tympanoplasties, to decrease the chance of having a small pinhole develop. The fascia graft is followed by multiple layers of absorbable packing.

TABLE I.
Patient Data.

Patient	Encephalocele Location	Operation	Follow-up (mo)	Empty sella	BMI (kg/m^2)	ICP (cm H_2O)
LA	L frontal/SOE L lateral sphenoid R lateral sphenoid	OPF Transpterygoid Observation	12	+	29.8	19
EH	R posterior ethmoid L frontal	Endoscopic repair OPF	30	+	32.8	Refuses
SS	L lateral sphenoid R lateral sphenoid	Transpterygoid Observation	15	N/A	N/A	Lost
FL	R frontal/SOE L central sphenoid	Endoscopic repair Endoscopic repair	5	+	36.0	32
IY	R lateral sphenoid L lateral sphenoid	Transpterygoid Observation	22	+	32.9	34

SOE = supraorbital ethmoid; OPF = osteoplastic flap and obliteration.

Encephaloceles located in the lateral recess of the sphenoid sinus required an endoscopic transpterygoid approach.[5] This approach begins with a total sphenoethmoidectomy and maxillary antrostomy. The posterior wall of the maxillary sinus is then removed, and the pterygopalatine fossa is entered. The internal maxillary artery and its branches are identified and transposed inferiorly or clipped and divided to expose the deeper areas of the pterygopalatine fossa. The vidian nerve and the maxillary division of cranial nerve V are dissected free and preserved if possible. The anterior wall of the sphenoid sinus is drilled away to gain access to the lateral recess of the sphenoid sinus. This provides the exposure needed to reduce the encephalocele and repair the skull base defect with bone and fascia grafts as described earlier in this section.

An external approach using an osteoplastic flap and ipsilateral frontal sinus obliteration was used for encephaloceles with significant extension into the frontal sinus. All visible mucosa is removed from the frontal sinus, followed by meticulous drilling with a diamond burr to remove mucosal remnants. Underlay bone and overlay fascia grafts are placed before obliterating the sinus with abdominal fat.

Postoperative Care

All patients were kept on a regimen of strict bedrest while lumbar drains were in place (typically, for 2 or 3 d postoperatively). After the lumbar drains were removed, patients gradually resumed ambulation and light activity for 6 weeks after surgery. Intravenous antibiotics, typically ceftriaxone because of its CSF penetration, were used while patients were in the hospital. Oral antibiotics were used for 2 or 3 weeks after discharge.

Patients were seen every 1 or 2 weeks postoperatively. Conservative endoscopic debridement was performed to maintain patency of sinuses surrounding the defect and to avoid stasis of secretions and bacterial infections. By 6 weeks after surgery, most patients had returned to relatively normal activity levels and little packing remained.

RESULTS

Five patients had multiple simultaneous encephaloceles treated between 1996 and 2001. One of the 5 had three separate encephaloceles, and there were 11 encephaloceles in the current series (Table I). Four of five patients were women. Average age was 49.2 years (age range, 39–60 y) with an average of 0.6 prior attempted repairs (range, 0–2). Three of the women had body mass index (BMI) greater than 30 (obese), and the fourth woman had a BMI of 29.8 (overweight). Presenting symptoms included CSF rhinorrhea (4 patients), seizure (2 patients), meningitis (1 patient), headache (1 patient), and dizziness (1 patient). Three encephaloceles in the ethmoid roof or central sphenoid were repaired using the standard endoscopic approach (Fig. 1). Three were located in the lateral sphenoid recess and were repaired using the endoscopic transpterygoid approach (Fig. 2A and B). Two were approached using an osteoplastic flap with frontal sinus obliteration, and three encephaloceles were asymptomatic without CSF leak and are being followed with serial MRI scans every 6 to 12 months. Complications occurred in only one patient and consisted of decreased sensation in the distribution of the intraorbital nerve and decreased sense of smell. To date, average follow-up on all patients is 17 months after encephalocele repair with 100% success. Radiographic studies demonstrated an empty sella in all four patients with available scans (Fig. 2C and D). No patients had radiographic or clinical evidence of hydrocephalus or dilated ventricles. Postoperative CSF pressures were measured in three of five patients after successful repairing of the leaks. The mean ICP was 28.3 cm water (range, 19–34 cm; normal range, 0–15 cm). One patient refused lumbar puncture and another has been lost to follow-up because of military service.

CASE REPORT

The case of one patient (L.A.) demonstrates some of the challenges encountered in treating this population. This 60-year-old woman was diagnosed with seizure disorder in 1979. She developed meningitis in 1997, and a right-sided sphenoid CSF

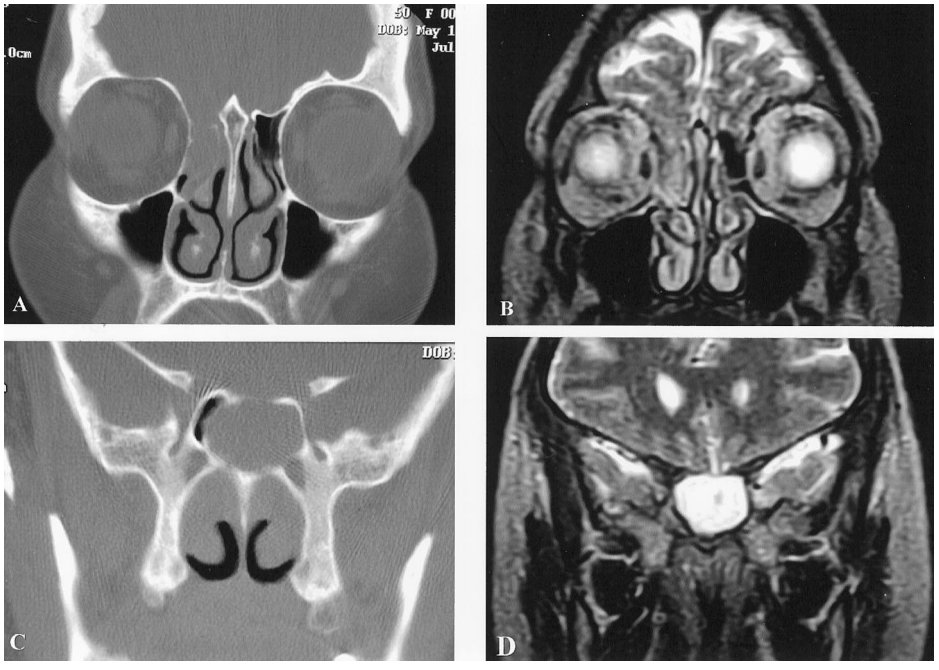

Fig. 1. Coronal computed tomography (CT) scan (**A**) and T2-weighted magnetic resonance imaging (MRI) scan (**B**) demonstrating a right-sided supraorbital encephalocele. The same patient also had a large left-sided sphenoid meningoencephalocele (**C** and **D**) repaired during the same operation.

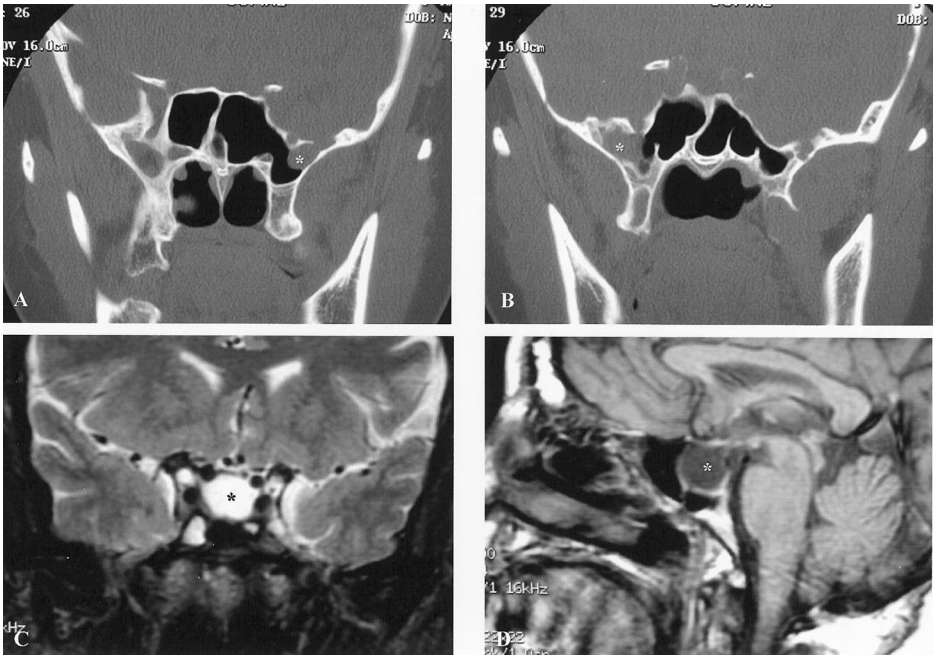

Fig. 2. Coronal CT scans demonstrating a left-sided sphenoid lateral recess encephalocele (**A**) that is being observed and a right-sided sphenoid lateral recess encephalocele (**B**) after endoscopic transpterygoid repair. Neo-osteogenesis and residual soft tissue fibrosis after obliteration of the right-sided lateral recess are evident. Coronal (**C**) and sagittal (**D**) MRI scans of the same patient demonstrate the empty sella (*). The sella turcica is filled with cerebrospinal fluid, and the residual pituitary gland is compressed.

leak was diagnosed. She underwent a sublabial approach by an outside neurosurgeon with closure of her leak. Nearly 3 years later, she presented with left-sided CSF rhinorrhea and underwent a transnasal and external ethmoidectomy approach combined with a lumbar–peritoneal shunt to close the leak. However, the leak persisted, and she presented to our institution with left-sided rhinorrhea and a β-2 transferrin–positive test result for CSF. Imaging studies demonstrated bilateral encephaloceles of the lateral sphenoid recesses, a left-sided frontal/supraorbital ethmoid encephalocele, and an empty sella. She underwent an osteoplastic flap procedure, repair of skull base defect with mastoid bone graft and temporalis fascia, and left-sided frontal sinus obliteration. Postoperative radionucleotide cisternogram demonstrated a functioning lumbar–peritoneal shunt. One month later, she underwent a successful left-sided transpterygoid approach to close the lateral sphenoid recess encephalocele. Follow-up studies have included a normal Schirmer test result for lacrimation, normal-sized ventricles on CT, a lumbar puncture with opening pressure of 19 cm H_2O (with the functioning shunt), and a small, asymptomatic stable encephalocele in the right-sided lateral recess of the sphenoid. She has not exhibited any further CSF leaks or neurological symptoms.

DISCUSSION

Spontaneous or nontraumatic, "normal pressure" encephaloceles are uncommon entities. Three conditions are necessary to have a cranionasal fistula: a bony defect, rupture of the dural/arachnoid membrane, and a pressure differential across the defect. The pathophysiology of spontaneous encephaloceles or CSF leaks is unclear, but a variety of explanations have been offered. Congenital skull base defects are unlikely because pneumatization of the frontal sinus and lateral sphenoid sinuses does not occur until later in childhood. Another proposed theory is that focal atrophy of the normal contents of the cribriform plate or sella turcica may create potential spaces that can be filled with enlarged pouches of normal arachnoid space extensions.[6] It seems more likely that significant pneumatization of the bony sinus walls creates sites of inherent structural weakness within the skull base. Hydrostatic pulsatile forces within the CSF may then lead to the formation of small pits within the bone at the sites of arachnoid villi. It is known that certain patients have impaired CSF absorption and thus have accentuated spikes in ICP. These significant fluctuations in the peak CSF pressure at the site of the bony weakness permit subsequent herniation of dura and brain tissue with the development of encephaloceles and CSF leaks at these points of attenuation.

In examining the presence of potential bony defects as the first condition needed for CSF leak/encephalocele, Shetty et al.[7] studied the pneumatization of the sphenoid sinus in 11 patients with spontaneous CSF leaks. The authors found that 10 of 11 (91%) had pneumatization of the lateral recesses in comparison with 23% to 43% of normal subjects.[8] In addition, 63% of their patients demonstrated arachnoid pits within the bony skull base. Ohnishi[9] has shown that the ethmoid roof is dehiscent 14% of the time. Detailed studies on the pneumatization of the frontal sinuses and associated potential sites of skull base defects have not been made.

Given the prevalence of significant paranasal sinus pneumatization with associated potential bony skull base defects in the general population, patients with spontaneous CSF leaks/encephaloceles also need to have significant variations in CSF pressure. Normal CSF pressures vary significantly depending on age, activity, and time of day. Normal intracranial pressures increase nearly three times from infancy to adulthood.[6] Pressures are also known to rise from a level of approximately 15 mm Hg in normal, awake patients to as high as 25 mm Hg during rapid-eye-movement sleep.[10] Thus, diagnostic lumbar punctures performed routinely in awake patients represent only one data point and do not detect fluctuations that occur

throughout the night and during activities such as sneezing or coughing. In addition, measures of ICP taken in patients with active CSF leaks are probably artificially lowered because of the chronic loss of CSF. For these reasons, we have begun to consider the role of obtaining postoperative ICP data after successfully repairing active spontaneous CSF leaks. This may be especially useful in patients with symptoms possibly attributable to elevated intracranial pressures, such as headaches and pulsatile tinnitus. Measuring the ICP postoperatively allows us to obtain a more realistic idea of the patient's true CSF pressure, rather than obtaining pressures preoperatively or intraoperatively that would be falsely lowered because of the active leaks.

Cerebrospinal fluid pressures have demonstrated transient elevations in other disease states, and there is probably a spectrum of mild to severe disease similar to that seen with vascular hypertension or glaucoma. Patients with benign intracranial hypertension (BIH) and empty sella syndrome (ESS) may have an altered physiology similar to that seen in patients with spontaneous CSF leaks or encephaloceles. Patients with BIH are typically obese women who present with elevated CSF pressures, papilledema, and headaches. Clark et al.[11] reported four patients treated for BIH who subsequently developed spontaneous CSF rhinorrhea. All leaks occurred at the cribriform plate, and the authors postulated that exaggerated CSF pulsatile flow leads to expansion and eventual rupture of the arachnoid sleeve surrounding olfactory filaments passing through the cribriform plate. Although the four female patients in our series did not present with typical BIH symptoms, all four were overweight or obese, according to their BMI. It is possible that these patients do not exhibit the symptoms of BIH when they are actively leaking CSF and do not develop elevated ICP with associated findings. It is our impression that they seem to develop BIH symptoms after their CSF leaks have been successfully patched.

Empty sella syndrome occurs when a weakened sellar diaphragm permits herniation of the subarachnoid space and its contents down into the sella turcica. This can lead to compression of the pituitary gland and give the radiological appearance of an empty sella. Although ESS occurs most commonly after necrosis of a pituitary tumor, it can be idiopathic and may represent a normal variant, especially in elderly patients.[12,13] Autopsy studies have shown that 5.5% to 26% of people have a rudimentary sellar diaphragm,[12,14] but radiographic studies demonstrate only a 5% to 6% incidence of empty sella in otherwise normal patients.[7,15] Primary, idiopathic ESS probably represents a milder form of intracranial hypertension and may even occur with normal fluctuations in CSF pulsations. Only 8% to 15% of patients with ESS have BIH[12,16] but, conversely, up to 94% of patients with BIH can have ESS.[17]

The majority of patients with ESS do not have typical signs or symptoms of elevated intracranial pressures,[12,18] but Maira[10] performed continuous ICP monitoring on 11 patients with primary ESS. Three patients had elevated ICP while awake, and an additional five patients had high intracranial pressures while in rapid-eye-movement sleep. Elevated ICP was accompanied by an absent or blunted nocturnal prolactin level in every patient. Only 3 of 11 patients had normal intracranial pressures at all times, and all of these had normal nocturnal prolactin spikes. Davis and Kaye[19] also used continuous ICP monitoring in a patient with ESS and spontaneous CSF rhinorrhea to show a slightly elevated ICP at baseline, but considerable peaks. Garcia-Uria et al.[20] reported on seven patients with spontaneous CSF rhinorrhea and ESS. Six of seven were women with no signs of elevated ICP.

In addition to a weakened sellar diaphragm, patients with ESS appear to have impaired circulation of CSF. Abnormal CSF absorption occurs in 80% to 84% of patients with ESS. This probably occurs because of blockage of the arachnoid villi over the convexities.[21,22] Clinical signs of papilledema and CSF leaks appear to correlate with actual increases in ICP and may be relieved after shunting procedures.[18] Patients with ESS and no clinical signs of elevated ICP may simply have a milder form of intracranial hypertension that we are unable to detect.

Spontaneous cranionasal fistulas represent a unique and challenging population. In contrast to patients with traumatic or postsurgical CSF leaks, patients with spontaneous encephaloceles and CSF leaks usually have broadly attenuated skull bases with large defects, probably because of fluctuations in their CSF pressure as described earlier in the present study. For these reasons, we are more aggressive in the placement of lumbar drains during the initial perioperative period and in limiting their activity postoperatively. Although only one patient in our series had a shunt in place, additional consideration may be given to long-term shunt placement in an attempt to minimize large fluctuations in ICP. Recently, we have started using acetazolamide for 6 weeks after surgery to decrease CSF production and hydrostatic pressures during the early healing process.

Treatment of patients with nasal encephaloceles and CSF leaks has changed since the early 1980s. Successful endoscopic repair of these lesions allows the otolaryngologist to treat most of these defects without the morbidity of intracranial procedures. Traditional neurosurgical approaches in this population have demonstrated good initial success of 89% but a recurrence rate of up to 50% with some cases recurring years after their initial repair.[15] This emphasizes the need for long-term follow-up for these patients because of their potential to develop subsequent leaks at the repair site or other areas of the attenuated skull base. The issue also arises in these patients as to which encephalocele to repair first and whether all encephaloceles need to be repaired. It was our treatment philosophy to repair all actively leaking encephaloceles to avoid the risk of meningitis or pneumocephalus. Encephaloceles were also repaired if they were a likely source of symptoms such as seizures. We typically only repaired one encephalocele during each surgery because of the significant length of time needed for this complex operation. The one exception was the repair of two encephaloceles in patient F.L., who had an active leak from a right-sided supraorbital encephalocele and a large left-sided encephalocele filling the entire sphenoid (Fig. 1). The repair of the right-sided leaking defect was made relatively quickly,

and adequate grafting material had been harvested for repair of the second encephalocele; therefore, we opted to perform both repairs at the same time. We have elected to observe three small, nonleaking encephaloceles in the series and follow them with serial imaging. It is not yet clear whether patching a defect at one site increases the likelihood of developing a subsequent CSF leak at another site. We are also investigating long-term intracranial pressures in this population by obtaining opening pressures during lumbar punctures after successful patching and after patients have been given adequate time to achieve a homeostatic state. This may enable us to more accurately determine the need for long-term shunts or acetazolamide in these patients.

CONCLUSION

Patients with spontaneous encephaloceles and CSF leaks represent a challenging population given the underlying nature of their disease. The pathophysiology of this disease probably begins with impaired CSF absorption that enhances the pulsatile nature of the intracranial pressures. These elevated forces widely attenuate the skull base and lead to subsequent herniation of the intracranial contents through inherently weakened areas, such as the sellar diaphragm or widely pneumatized sinuses. Radiographic evidence of empty sella and extensive pneumatization of the paranasal sinuses should raise the suspicion that multiple spontaneous cranionasal fistula may be present. Endoscopic repair and postoperative management in these patients is more difficult than in patients with encephaloceles or CSF leaks from other causes, and a comprehensive treatment plan may eventually require shunting or acetazolamide to prevent further recurrences. Long-term follow-up and investigation of the intracranial pressures in this population will enable us to better care for these patients.

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