Published in final edited form as:

Laryngoscope. 2014 November ; 124(11): 2645–2650. doi:10.1002/lary.24661.

Obstructive Sleep Apnea in Patients Undergoing Endoscopic Surgical Repair of Cerebrospinal Fluid Rhinorrhea^{†,‡,§,¶}

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Abstract

OBJECTIVE—To examine the relationship between Cerebrospinal Fluid (CSF) Rhinorrhea and Obstructive Sleep Apnea (OSA).

STUDY DESIGN—Retrospective chart-review of patients who underwent surgical repair of encephaloceles and/or CSF rhinorrhea at a tertiary medical center over a 12 year period.

METHODS—Pertinent demographic, clinical and surgical data including age, sex, medical and surgical history was obtained. Patients were classified by etiology of CSF leak into a spontaneous leak group and a non-spontaneous leak group, which included patients with documented trauma, malignancy, or known iatrogenic injury.

RESULTS—We retrospectively identified 126 patients who underwent repair of encephalocele or CSF rhinorrhea. Of these, 70 (55.5%) were found to have a spontaneous etiology, while 56 (44.4%) had a non-spontaneous cause. Patients with spontaneous CSF rhinorrhea were more likely than their non-spontaneous counterparts to have a diagnosis of obstructive sleep apnea (OSA, 30.0% versus 14.3%, $p=0.0294$) and radiographic evidence of an empty sella on MRI (55.4% vs. 24.3%, $p=0.0027$). Overall, patients in the spontaneous CSF rhinorrhea group were more likely to be female compared to the non-spontaneous group (84.3% versus 41.1% female, $P=0.0001$).

[†]Presented at the North American Skull Base Surgery Conference, February 15-17, Miami, FL

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[§]GMF gratefully acknowledges support from the NIH National Research Service Award Institutional Training Grant 5T32DC005360, to the University of North Carolina for support of this research.

[¶]The authors have no other funding, financial relationships, or conflicts of interest to disclose.

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CONCLUSIONS—Our study shows that patients with spontaneous CSF rhinorrhea are significantly more likely to have a diagnosis of OSA compared to those with non-spontaneous causes of CSF leaks, or to the general population (incidence of 1-5% in various population studies). Given the known association between OSA and intracranial hypertension (ICH), it may be prudent to screen all patients with spontaneous CSF rhinorrhea for symptoms of OSA as well as for ICH, and vice versa.

Keywords

CSF Rhinorrhea; Intracranial Hypertension; Obstructive Sleep Apnea; Empty Sella; Obesity

Introduction

Cerebrospinal fluid (CSF) rhinorrhea results from an osseous defect along the skull base with an associated anatomic disruption in the arachnoid and dura mater ¹. Spontaneous CSF fistulae occur in the absence of a known inciting factor such as tumor, trauma, or iatrogenic injury. Previous studies have observed an increased incidence of elevated intracranial pressure (ICP) in patients with spontaneous CSF leaks ². Patients with spontaneous CSF leaks also exhibit clinical signs of elevated ICP, such as pressure-type headaches, vertigo, pulsatile tinnitus, and visual complaints. Furthermore, MRI findings of empty or partially-empty sella, which are frequently noted in patients with elevated ICP, have also been observed in patients with spontaneous CSF leaks (Figure 1A) ^{3,4}. Elevated ICP is thought to play a role in the development of spontaneous CSF fistulae and encephaloceles by exerting pulsatile forces at innately weakened anatomical sites within the skull base ⁵.

From a demographic perspective, spontaneous CSF leaks occur commonly in obese, female patients ¹, suggesting the possible role of elevated body mass index (BMI) in the pathogenesis of elevated ICP and subsequent development of spontaneous CSF fistulae. Large-scale retrospective studies have shown that CSF pressure and BMI have a positive, linear relationship, with a 0.24mm-Hg increase in CSF pressure for every unit of BMI ⁶. Elevated BMI is thought to lead to elevated ICP through the mechanisms outlined in Figure 2. Elevated ICP can result in CSF fistulae in two ways: (1) pulsatile forces at arachnoid villi cause formation of arachnoid pits and erosion of the skull base (Figure 1D); and (2) an elevated CSF pressure gradient can act on intrinsically weak sites within the skull base, such as the fragile cribriform plate (Figure 1B) or overly-pneumatized lateral sphenoid recesses (Figure 1C).

Independent of obesity, it has been conjectured that obstructive sleep apnea (OSA) may also have a role in the pathogenesis of spontaneous CSF leaks. Periodic apnea, a hallmark of OSA, is believed to cause an analogous episodic increase in ICP ⁷. While observational studies have linked spontaneous CSF otorrhea with OSA, the data for CSF rhinorrhea and OSA are sparse ⁸⁻¹⁰.

We hypothesize that there is a relationship between OSA and the development of spontaneous CSF rhinorrhea and skull base encephaloceles. The aim of our study is to assess for possible associations between anterior skull-base encephaloceles and CSF fistulae with OSA.

Materials and Methods

Patient Data

This was conducted as a single-institution retrospective review of 126 consecutive patients with surgically confirmed anterior skull base dehiscence leading to encephaloceles or CSF leaks over a 12-year period (2000-2012). Demographic, clinical, and radiographic information including age, sex, BMI, presence of OSA, co-morbidities, CT and MRI findings, operative approach and findings, and site of defect were obtained. Only patients with a clearly stated diagnosis of OSA in their medical history were classified as having the disease. All medical records included a symptom inventory, and presence of hallmark symptoms of OSA, namely loud snoring, daytime somnolence, apneic episodes during sleep and headaches, were obtained. All radiographic findings were individually confirmed by a board-certified radiologist with a certificate of added qualification in neuroradiology (B.H.)

Diagnosis and Pre-operative Management

Spontaneous CSF leak or encephalocele was defined by presence of Beta-II transferrin positive nasal secretion, and/or skull base dehiscence with herniation of brain tissue or meninges into paranasal sinuses on MRI/CT, in the absence of a clearly-defined etiology such as trauma, surgery, or tumor involving the base of skull. Other modalities of diagnosis included cisternograms for slow or intermittent leaks, cisternography, MRI with or without cisternograms, and intrathecal fluorescein.

Patients with CSF leaks that did not resolve with conservative management, lumbar drain, or acetazolamide therapy, were considered to be surgical candidates. A variety of approaches were used to repair the CSF leak or encephalocele, and decision on which technique to use was based on surgeon (C.E., A.Z., B.S.) preference, location, and size of defect. Patients suspected to have high-pressure leaks frequently received a lumbar drain prior to procedure, which generally remained in place for 48-72 hours post-operatively.

Post-operative Follow-up

When indicated, a lumbar drain was placed by the neurosurgery team and intrathecal fluorescein was injected prior to the start of the repair procedure. Post-operatively, the drain was kept in place for 48-72 hours and CSF was drained at 10ml/hour to ensure that the graft remained in place. All patients were placed on strict activity restrictions, with no nose-blowing or CPAP for six-weeks post-op to ensure adequate healing at the site of repair.

Statistical Analysis

Statistical analysis was carried out between spontaneous CSF and non-spontaneous CSF groups using Fisher's one-tailed test with respect to OSA and gender, and using a two-sample *t*-test with respect to BMI and age. P-values <0.05 were determined a priori to be statistically significant.

Results

Results have been summarized in Tables 1 and 2. One hundred and twenty-six patients who underwent repair of encephalocele or CSF rhinorrhea were identified. Of these, 70 (55.5%) were found to have a spontaneous etiology, while 56 (44.4%) had a non-spontaneous cause.

Patients with spontaneous CSF rhinorrhea were more likely than their non-spontaneous counter-parts to have a diagnosis of OSA (30.0% versus 14.3%, $p=0.0294$) and on average had a significantly greater BMI (35.64 vs. 29.99, $p=0.0031$). On their symptom inventory, seven patients from the spontaneous group and two patients from the non-spontaneous group complained of loud snoring, but did not have a documented diagnosis of OSA or an available PSG, and were thus not included in the OSA-cohort.

Patients with spontaneous CSF rhinorrhea were also significantly older (mean age, 51.7 versus 42 years, $p=0.0005$). Overall, there was a higher prevalence of females in the spontaneous group compared to the non-spontaneous group (84.3% versus 41.1% female, $p=0.0001$).

MRI was available for 96 of the 126 subjects, and evidence of either empty or partially empty sella, defined as a sella turcica containing no discernible pituitary gland or in which the pituitary gland is displaced inferiorly and flattened along the sellar floor, was seen on MRI of 55.4% of spontaneous CSF leak patients, and 24.3% non-spontaneous CSF leak patients ($p = 0.0027$). When non-spontaneous CSF leaks were stratified by etiology, 16.7% of tumor, 18.8% of trauma patients, and 37.5% of iatrogenic patients were found to have evidence of an empty sella.

The most common region of dehiscence was found to be the ethmoid roof, inclusive of the fovea and cribriform (50.8%), followed by the lateral sphenoid (26.2%), central sphenoid (12.7%), and frontal sinus (10.3%).

Discussion

While the etiology of spontaneous CSF leaks is only partially understood at present, there is observational evidence suggesting the role of elevated intracranial pressure in their development and progression. Several previous studies have noted that there is considerable commonality between the clinical, radiographic, as well as demographic appearances of patients with idiopathic intracranial hypertension and spontaneous CSF leaks.

The human body produces CSF at the rate of 20 ml/hour, which roughly equals 350-500 ml per day. The majority of CSF is produced in the choroid plexus of the lateral, third, and fourth ventricles, while a smaller quantity is formed from a combination of capillary ultrafiltration and water metabolism¹¹. CSF is constantly absorbed via the arachnoid villi, which empty into the connecting dural sinuses. These arachnoid villi are one-way valves that promote anterograde flow of CSF into the low-pressure vascular dural sinuses. A hydrostatic pressure gradient of 1.5 to 7 cm H₂O is required to keep these valves open and to prevent retrograde CSF flow back into the villi. Under physiologic conditions, the pressure in the venous sinuses is always lower than that of CSF. Both obesity and OSA increase the

venous pressure within the brain's dural sinuses, and as a result, CSF is unable to effectively drain out of these arachnoid granulations. This creates a build-up of CSF within the cisterns, elevating intracranial pressure.¹²

Demographically, spontaneous CSF leaks are found more commonly in obese, female patients. In agreement with previously published literature, our study found that patients with spontaneous leaks had an average BMI of 35.64, which was significantly higher than their non-spontaneous counterparts (29.99, $p=0.0031$)^{8,9}. Elevated BMI and associated centripetal fat accumulation, results in elevated intra-abdominal and intra-pleural pressures. These high visceral pressures cause cardiac filling-pressures to rise, causing a systemic increase in venous pressure and decreasing venous return from the brain.

It is theorized that systemic venous pressure increases and has an effect on intracranial pressure in two independent ways. *First*, the dilation of veins in the brain causes a mass-like effect and increases intracranial pressures; and *second*, it prevents CSF circulation by blocking drainage of CSF from arachnoid villi into the dural sinuses⁷. Over time, pulsatile forces at arachnoid villi cause formation of arachnoid pits and erosion of the skull base. Once the CSF pressure in the brain exceeds the tensile strength of inherently weak sites within the skull base, such as the cribriform plate and pneumatized lateral sphenoid recesses, leaks follow. Furthermore, large-scale clinical studies assessing lumbar puncture opening pressures have confirmed that CSF pressure increases with every unit increase in BMI⁶.

In a mechanism independent to that of elevated BMI, OSA has also been associated with elevated ICP. OSA is characterized by spells of apnea leading to episodic nocturnal hypoxia and hypercarbia. Jennum and Borgensen discovered that during sleep, patients with OSA developed significant ICP elevations that were synchronously associated with their episodes of apnea⁷. They noted that during episodes of apnea, three phases of ICP elevation could be distinguished: an initial increase in ICP associated with the onset of apnea, followed by a slow increase mediated by hypercapnia, hypoxia, and cerebral vasodilation; and finally, a steep increase in ICP mediated by increased systemic arterial and central venous pressures. Furthermore, they found that even during awake states, the values of ICP were pathologically elevated in patients with severe OSA. In agreement with the previously suggested association between OSA and elevated ICP, our study reports a statistically significant ($p=0.0294$) association between spontaneous CSF leaks and OSA. Furthermore, we believe that the true incidence of OSA may actually be even higher than reported in this study, since nine loud-snorers were included in the non-OSA cohort, as they did not have a definitive diagnosis of OSA or an available polysomnogram. It remains to be substantiated, however, whether OSA is causative or is simply a comorbidity among patients with elevated CSF pressures.

While there is no conclusive evidence that suggests treating OSA reduces CSF pressures, observational studies have reported that nocturnal oxygenation improves the signs and symptoms of idiopathic intracranial hypertension in men¹³. The reduction in nocturnal hypercarbia and subsequent reduction in vasodilation resulting from OSA treatment may, thus, reduce CSF pressures over time.

Female gender also seems to play a role in the pathogenesis of CSF leaks, although the etiology of this relationship still remains unclear. In our study, patients with CSF leaks were significantly more likely to be female, and this association was further magnified in the spontaneous CSF leak group compared to the non-spontaneous group (84.3% versus 41.1% female, $p=0.0001$). Related conditions such as idiopathic intracranial hypertension, which also has a high predilection for women, suggests role of hormonal influences in the development of elevated CSF pressures, but again, the pathophysiology of this phenomenon remains unknown¹⁴.

Several previous studies have documented the association between a radiographic empty sella and elevated intracranial pressure^{3,4,15}. There is also anecdotal evidence that in patients with elevated ICP, the CSF leak actually functions as a pressure-release valve of sorts. The 'empty' appearing sella results from elevated intracranial pressures causing dural and CSF herniation through the sella diaphragma into the sella turcica. This subsequently flattens the pituitary gland along the walls of the sella turcica, giving the appearance of a vacant, CSF-filled space. Our study found evidence of either empty or partially-empty sella in 55.4% of the MRI scans of patients with spontaneous CSF leaks and 24.3% of patients with a non-spontaneous etiology of their leaks ($p = 0.0027$). Interestingly, we also noted a high incidence of empty sella in patients with iatrogenic causes of CSF leaks (37.5%), possibly suggesting that elevated ICP may somehow be related to iatrogenic injury to the dura. However, this relationship is merely conjecture, and further studies would be required to elucidate this interesting observation.

Innately weakened areas in the skull base, such as the ethmoid roof, cribriform plate, and lateral recess of the sphenoid sinus and its embryologic cleavage plane, are prone to dehiscence, especially under the influence of chronically elevated intracranial pressures as seen in patients with spontaneous CSF leaks. It is an imbalance between the tensile strength of the tissue and the hydrostatic pressure of the CSF that is thought to contribute to this dehiscence. In our study, the most common defect was at the ethmoid roof (50.8%), followed by the lateral sphenoid (26.2%), central sphenoid (12.6%), and frontal sinus (10.3%). Extensive pneumatization of the lateral recesses of the sphenoid across the embryologic cleavage plane of the pre and post sphenoid, or congenitally weak areas such as the foramen cecum or cribriform plate, also increase the propensity of patients with elevated ICP to develop CSF leaks. Focal atrophy of olfactory bulbs at the cribriform plate can also create potential spaces that fill with arachnoid granulations, further implicating the anterior cranial fossa as a common location for CSF leaks¹⁶.

While there is only limited anecdotal evidence suggesting that positive pressure ventilation causes pneumocephalus,^{17,18} our team routinely recommends cessation of CPAP during an active CSF leak, as well as for 6-weeks after repair, as it poses significant risk for breakdown of the repair site. Alternate means of reducing post-operative hypoxemia may be used in these patients, and have been well described by Jensen et al. These include use of mandibular prostheses, nasal trumpets, supplemental oxygen delivered via nasal cannula, as well as minimization of opioids, early ambulation, and aggressive pulmonary toilet.¹⁹

Although this is the one of the largest cohorts of CSF leaks that has been reported in literature, several limitations exist, mainly related to the retrospective nature of data acquisition. A major limitation of this study is the absence of CSF opening pressures, which were unfortunately not recorded during lumbar puncture. In the absence of polysomnography (PSG) results for every patient, it was impossible to ascertain whether patients without a documented diagnosis of OSA were actually free of disease. However, since all patients were screened for symptoms of OSA, in the absence of any symptoms, it is less likely that a patient suffered from undiagnosed OSA. Conversely, the symptom inventory did reveal several patients with symptoms suspicious for OSA, but in the absence of a documented PSG or OSA diagnosis, they were not included in the OSA cohort. For this reason, we believe that the incidence of OSA in CSF leaks, both spontaneous and non-spontaneous, may actually be higher than what we report in this study. Finally, compliance of CPAP usage was unknown or inconsistently documented in the medical record, and the role of OSA treatment on progression of CSF leaks remains to be established.

Conclusion

In one of the largest cohorts of CSF leaks reported in the literature, we show that patients with spontaneous rhinorrhea or encephaloceles are significantly more likely to have a diagnosis of OSA compared to those without spontaneous causes of CSF leaks. In addition, these patients were also more likely to show radiographic manifestations of elevated ICP, such as an empty sella, and demographically, were more likely to be obese, older, and female.

OSA may contribute to elevated ICP, and consequently, spontaneous CSF leaks, by chronic hypercapnia-mediated cerebral vasodilation and decreased CSF outflow during apneic episodes. Given the known association between OSA and elevated ICP, we recommend that all patients with spontaneous CSF rhinorrhea be screened for intracranial hypertension: radiographically for signs of empty sella using MRI and ophthalmologically to rule out papilledema. We also suggest that these patients be evaluated for OSA with a focused clinical questionnaire and routine polysomnogram, when indicated.

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Figure 1.

Common radiographic findings seen in spontaneous CSF encephaloceles and fistulae: Empty Sella (A), Cribriform plate dehiscence (B), Excessively pneumatized lateral sphenoid recesses with greater sphenoid wing dehiscence (C), Arachnoid pits with erosion of skull base (D).

Figure 2.
Theoretical mechanism for role of obesity and OSA in development of skull base encephaloceles and CSF fistulae.

Table 1

Demographic and Radiographic differences between Spontaneous and Non-spontaneous CSF leaks.

	Spontaneous	Non-Spontaneous	
Age	N = 70pts Mean = 51.7 years	N = 56pts Mean = 42.0 years	P = 0.0005
BMI	N = 44 Mean = 35.64	N = 42 Mean = 29.99	P = 0.0031
OSA	21/70 (30.0%)	8/56 (14.3%)	P = 0.0294
Gender	Male = 11/70 (15.7%) Female = 59/70 (84.3%)	Male = 33/56 (58.9%) Female = 23/56 (41.1%)	P = 0.0001
Empty Sella	N = 56 (MRI available) 31/56 (55.4%)	N = 37 (MRI available) 9/32 (24.3%)	P = 0.0027

Table 2

Incidence of Obesity and OSA in CSF Leaks stratified by etiology and location.

Spontaneous	<i>n</i>	<i>BMI</i>	<i>OSA</i>
Ethmoid	36	36.1	30.6%
Frontal	5	36.2	40.0%
Lateral Sphenoid	25	34.8	20.0%
Central Sphenoid	4	34.4	75.0%
<i>All-locations</i>	<i>70</i>	<i>35.64</i>	<i>30.0%</i>
Non-spontaneous	<i>n</i>	<i>BMI</i>	<i>OSA</i>
Ethmoid	28	29.8	14.2%
Frontal	8	32.0	12.5%
Lateral Sphenoid	8	29.3	12.5%
Central Sphenoid	12	29.3	18.2%
<i>All-locations</i>	<i>56</i>	<i>29.99</i>	<i>14.3%</i>
All CSF Leaks	<i>n</i>	<i>BMI</i>	<i>OSA</i>
Ethmoid	64	33.0	23.4%
Frontal	13	34.0	23.1%
Lateral Sphenoid	33	33.0	18.2%
Central Sphenoid	16	30.9	33.3%
<i>All-locations</i>	<i>126</i>	<i>32.80</i>	<i>23.0%</i>