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Mitsutoshi Nakada, MD Kanazawa University, Ishikawa, Japan



Original Article

# Spontaneous cerebrospinal fluid rhinorrhea as a primary presentation of idiopathic intracranial hypertension, management strategies, and clinical outcome

Ahmed Elshanawany<sup>1</sup>, Farrag Mohammad<sup>2</sup>

Department of Neurosurgery, Faculty of Medicine, Assiut University, Department of Neurosurgery, Assiut University, Assiut, Egypt.

E-mail: \*Ahmed Elshanawany - aelshanawany@aun.edu.eg; Farrag Mohammad - farragmohammad@aun.edu.eg



# \*Corresponding author: Ahmed Elshanawany, Department of Neurosurgery, Faculty of Medicine, Assiut University, Assiut, Egypt.

aelshanawany@aun.edu.eg

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#### **ABSTRACT**

Background: Causes of cerebrospinal fluid (CSF) rhinorrhea could be divided into primary (spontaneous) and secondary (head trauma and iatrogenic). Idiopathic intracranial hypertension (IIH) has emerged as a cause for spontaneous CSF rhinorrhea but is still underestimated, may be overlooked and needs special consideration in management. The objective of this study is to demonstrate spontaneous CSF rhinorrhea as the primary presentation of IIH and explore the algorithm of management.

Methods: All patients with spontaneous (primary) CSF rhinorrhea were included with complete clinical and radiological assessment. We performed lumbar puncture and CSF pressure measurements in the lateral decubitus position for all included patients to detect those with intracranial hypertension. A pressure of 20 cmH₂O in cases of CSF rhinorrhea is considered a cutoff for diagnosing raised intracranial pressure. When intracranial hypertension was diagnosed, patients were subjected immediately to lumboperitoneal shunt. If CSF leakage stopped after shunt insertion, we would not perform skull base repair, and the patient was sent for follow-up. However, if CSF leakage did not stop after shunt insertion despite normalization of intracranial tension or recurrence of CSF rhinorrhea despite shunt patency or there was intracranial pneumocephalus, skull base repair would be performed.

Results: During the period of the study, 293 cases of CSF rhinorrhea were seen. Only 42 (14.3%) patients were diagnosed with spontaneous CSF rhinorrhea, and the remaining were posttraumatic. Thirty-seven patients (88.1%) of 42 patients revealed high CSF pressure readings. All 37 patients received lumboperitoneal shunt followed by CSF rhinorrhea stoppage. Later, during follow-up, 7 patients developed recurrence of leakage; 3 of them revealed shunt obstruction, and rhinorrhea improved after shunt revision. The other 4 patients revealed patent shunt and needed skull base repair.

Conclusion: Spontaneous CSF rhinorrhea is considered secondary to IIH until proven otherwise. Initial placement of lumboperitoneal shunt may provide an effective alternative to skull base repair for the treatment of patients with IIH presenting with CSF rhinorrhea.

Keywords: Idiopathic intracranial hypertension, Lumboperitoneal shunt, Skull base repair, Spontaneous cerebrospinal fluid rhinorrhea

#### INTRODUCTION

Causes of cerebrospinal fluid (CSF) rhinorrhea could be divided into primary (spontaneous) and secondary (head trauma and iatrogenic). Head trauma (penetrating or closed) is the most

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common cause of CSF rhinorrhea. 80% of CSF rhinorrhea cases are traumatic, and 16% are iatrogenic in intracranial or rhinology procedures like sinus surgery.<sup>[16]</sup> Spontaneous CSF rhinorrhea accounts for only 4% of cases. Among the spontaneous (nontraumatic) causes, the majority are due to congenital dehiscence, skull base erosion as a sequelae to osteomyelitis tumor of the skull base, or focal brain atrophy (olfactory or sellar).[15] Idiopathic intracranial hypertension (IIH) has emerged as a cause for spontaneous CSF rhinorrhea but is still underestimated and may be overlooked and needs special consideration in management.[1,7,33,36]

Regardless of the etiology, CSF rhinorrhea occurs when there is a disruption in the arachnoid and dura mater coupled with an osseous defect and a CSF pressure gradient that is continuously or intermittently greater than the tensile strength of the disrupted tissue.<sup>[21]</sup> The mechanism by which IIH causes spontaneous CSF rhinorrhea is believed to be persistent or intermittent elevated intracranial pressure (ICP). Over time, the elevated CSF pressures and the hydrostatic pulsatile forces that are transmitted to the skull base eventually erode through thin areas of bone, most commonly the ethmoid roof, cribriform plate, and the lateral recess of the sphenoid sinus. This may mask the classic clinical presentation of IIH. In many instances, the classic clinical picture of IIH occurs after the repair of the skull base defect and stoppage of CSF leakage. [27,31,32,34]

The objective of this study is to demonstrate spontaneous CSF rhinorrhea as the primary presentation of IIH and explore the algorithm of management.

#### **MATERIALS AND METHODS**

This study was conducted in Assiut university hospitals in the period between 2015 and 2021. It is a retrospective descriptive hospital based study. Consent from patients to share their data in the study was obtained with an ethical committee approval number (04-2024-300356). All patients with spontaneous (primary) CSF rhinorrhea after exclusion of the presence of nasal and intracranial pathologies or history of head trauma were subjected to the following protocol.

First: Neurological, ENT, and ophthalmological clinical assessment of all patients, reporting laterality of the leakage and complete visual assessment (fundus examination and visual field). Determination of body mass index (BMI) for all patients included in this study.

Radiological studies, including computed Second: tomography (CT) brain (with contrast, thin cut CT skull base bone window and paranasal sinuses) were done for all patients to document the presence of skull base defect, its anatomical location, and any brain tissue herniating from the defect. Magnetic resonance imaging brain with gadolinium and magnetic resonance venography (MRV) were done to

detect the presence of any nasal pathology, brain pathology, brain herniation, and nasal meningocele, empty sella, assessment of patency of venous sinus.

Third: lumbar puncture and CSF pressure measurement in lateral decubitus position for all included patients to detect those with intracranial hypertension [Diagram 1]. Patients with raised intracranial tension were reported. The established cut off for diagnosing IIH is 25 cm H<sub>2</sub>O<sup>[38]</sup>, but we consider 20 cmH<sub>2</sub>O in cases of CSF rhinorrhea as a cutoff, which could be explained by the already release of pressure that occurred by CSF outflow. [29] When intracranial hypertension was diagnosed, patients were subjected immediately to a lumboperitoneal shunt [Diagram 1]. We used Medtronic lumboperitoneal system.

If CSF leakage stopped after shunt insertion, we would not perform skull base repair, and the patient was sent for followup. However, if CSF leakage did not stop after shunt insertion despite normalization of intracranial tension or there was intracranial pneumocephalus, skull base repair would be performed.

For those patients with CSF recurrence again later during follow-up, the shunt was assessed for its patency. If the shunt is patent, skull base repair will be performed. If the shunt were not patent (obstructed), shunt revision would be performed. Assessment of shunt obstruction was performed using a CT shuntogram (injecting intrathecal dye and following its spillage in the abdomen).

#### **RESULTS**

During the period of the study, 293 cases of CSF rhinorrhea were seen. We had 88 females and 205 males [Table 1]. Age ranged between 23 and 78 years old. Most of them are posttraumatic (history of trauma), and only 42 (14.3%) patients were diagnosed with spontaneous CSF rhinorrhea (no history of trauma) [Table 1]. Lumbar puncture and CSF pressure measurement were performed for all patients diagnosed with spontaneous CSF rhinorrhea.

After doing CSF pressure measurement, 37 patients (88.1%) of 42 patients revealed high readings. Their readings ranged between 24 cmH<sub>2</sub>O and 43 cmH<sub>2</sub>O. The other 5 patients revealed readings below 20 cmH<sub>2</sub>O and were sent immediately for skull base repair [Table 2].

All those with high CSF pressure were females; their ages ranged between 25 years old and 63 years old (mean 44). Seven of them are known to have hypertension controlled by medical treatment. One of them had a history of open heart surgery. Obesity was documented in 29 patients as we had 8 patients with a BMI between (25 and 30), 28 patients with a BMI between (30 and 35), and one patient with a BMI between 35 and 40. Twenty-one patients proved to

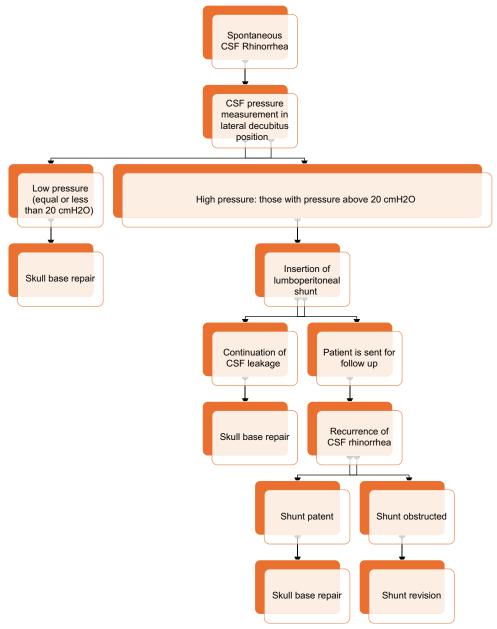


Diagram 1: Algorithm of spontaneous cerebrospinal fluid rhinorrhea management. CSF: Cerebrospinal fluid.

**Table 1:** Summary of patients characteristics.

The pathology	Number (%)	Gender
Posttraumatic	236 (80.5)	Females: 43 (18.2) Males: 193 (81.8)
History of skull base surgery	15 (2.8)	Females: 8 (53.3) Males: 7 (46.7)
Spontaneous CSF rhinorrhea	42 (14.3)	Females: 37 (88.1) Males: 5 (11.9)
CSF pressure above 20 mmHg	37 (12.6)	All are females

**Table 2:** Summary of different CSF pressure measurements.

Pressure measurement readings	Number of patients
Below 20 mmH <sub>2</sub> O	5
Between 20 and 30 mmH <sub>2</sub> O	34
Above 30 mmH <sub>2</sub> O	3
CSF: Cerebrospinal fluid	

have gynecological problems and were receiving hormonal treatment. Three patients had sleep apnea [Table 3].

Visual assessment revealed papilledema in 3 patients; 2 were grade I papilledema, and 1 was grade II, while the remaining patients had no visual impairment. Those 3 patients with visual impairment were with ICP above 35 mmH<sub>2</sub>O, BMI between 30 and 35, and all of them had sleep apnea.

Cribriform plate was the common site for skull base defects in 27 patients [Figure 1], ethmoidal defects were seen in 5 patients [Figure 2] while the last 5 patients had sphenoidal defects, 23 patients with CSF coming from the left nostril and 14 from right nostril [Table 4]. Nineteen patients had meningoencephalocele, 35 patients had an empty sella as a radiological finding, 31 patients revealed attenuated unilateral transverse venous sinus [Figures 2 and 3] had bilateral attenuated transverse sinus (no actual venous sinus thrombosis detected) [Table 5].

Table 3: Summary of comorbidities.		
Hypertension	7	
Cardiac	1	
Obesity	BMI between (30 and 35): 28	
	BMI between (35 and 40): 1	
Sleep apnea	3	
Gynecological problems and	21	
receiving hormonal therapy		
BMI: Body mass index		

All 37 patients stopped rhinorrhea after shunt insertion. During the period of follow-up, 7 patients showed recurrence of CSF rhinorrhea. In 3 of them, their shunts were obstructed, and symptoms improved after shunt revision. The other 4 patients had patent lumboperitoneal shunt, so skull base repair was performed either through an open skull base approach (2 patients), endoscopic transnasally (1 patient), or combined (1 patient) [Diagram 2].

# **DISCUSSION**

Most causes of spontaneous (nontraumatic or primary) CSF rhinorrhea are now thought actually to be secondary to elevations in ICP that might be seen in patients with IIH.[1,7,15,36] In 1994, Clark et al. were the first to propose that IIH was one of the causes of CSF rhinorrhea. [7] However, increased ICP is not always present in cases of spontaneous CSF rhinorrhea.<sup>[16,41]</sup> In our study, we had 88.1% of cases admitted with spontaneous CSF rhinorrhea were secondary to IIH. Hence, performing CSF pressure measuring on the diagnosis of spontaneous rhinorrhea and before applying any treatment option is mandatory to establish the diagnosis and determine the direction of treatment.

IIH occurs mainly in women, and the pathogenesis is not fully understood. The combination of raised intracranial tension without mass lesion, normal CSF composition, and

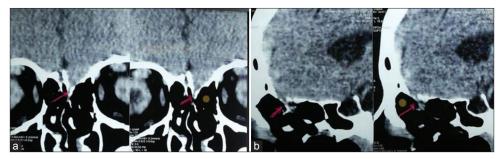


Figure 1: A 23-year-old female presented with spontaneous cerebrospinal fluid rhinorrhea; her fundus is papilledemic, (a) coronal, and (b) sagittal. Computed tomography brain revealed a skull base defect at the cribriform plate with herniated brain tissue and meninges. Arrows refer to cribriform plate defect with herniated brain matter.

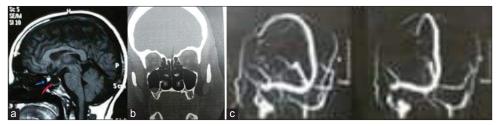


Figure 2: 55-year-old female presented with spontaneous cerebrospinal fluid rhinorrhea, fundus revealed papilledema, body mass index 30 with history of open heart surgery (a) sagittal magnetic resonance imaging revealed empty sella (red arrow) and ethmoidal meningocele (blue arrow), (b) computed tomography bone window revealed ethmoidal skull base defect (black arrow), (c) magnetic resonance venography revealed attenuated left sigmoid sinus.

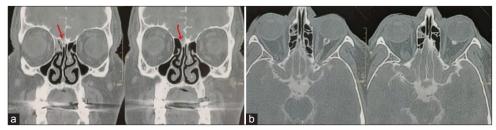


Figure 3: A 46-year-old female presented with spontaneous cerebrospinal fluid rhinorrhea. (a) Coronal and (b) axial computed tomography cisternography revealed a defect in the cribriform plate. Red arrows refer to skull base defect at ethmoidal sinus.

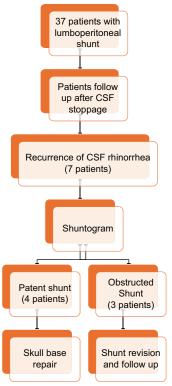


Diagram 2: Summary of patient management results. Cerebrospinal fluid

no underlying etiology are accepted criteria to diagnose IIH.[11,25] Patients are usually clinically presented with a headache that is nonspecific and resembles other primary headaches [13] and visual affection in the form of papilledema, blurred vision, diplopia (6th cranial nerve palsy), and transient visual obscurations (darkening of vision). Furthermore, there are neck pain, back pain, and pulsatile tinnitus.[22] Recently, spontaneous CSF rhinorrhea is increasingly considered a manifestation of IIH.[7,15,28]

Patients do not present with the classic clinical picture of IIH when developing spontaneous CSF rhinorrhea, as CSF release from the nose relieves intracranial tension and most of the pressure symptoms.[2] On this occasion, we consider the pressure of 20 mmH<sub>2</sub>O to be diagnostic for intracranial hypertension.<sup>[29]</sup> Many reports revealed

Table 4: Side of CSF leakage.	
Side of CSF leakage	Left nostril: 23 (62.2%) Right nostril: 14 (37.8%)
CSF: Cerebrospinal fluid	

Table 5: Summary of radiological findings.		
Radiological finding	Number of patients (%)	
Site of skull base defect	Cribriform plate: 27 (73) Ethmoidal: 5 (13.5) Sphenoidal: 5 (13.5)	
Meningoencephalocele	19 (51.4)	
Empty sella	35 (94.5)	
Transverse sinus attenuation	<ul><li>Unilateral 31 (83.8)</li><li>Bilateral: 3 (8.1)</li></ul>	

the appearance of classic manifestations of idiopathic increase intracranial tension after the repair of the skull base defect and stoppage of CSF leak without management of raised intracranial tension.[2] The ophthalmological assessment revealed 3 (8.1%) patients with papilledema; their papilledema varies between grade I to II discovered during the routine clinical examination, and those patients had no classic visual affection of cases of IIH, which could be explained by CSF leakage.

Female gender in childbearing period, obesity, and obstructive sleep apnea are risk factors in developing IIH and spontaneous CSF rhinorrhea. [3,6,35,40] Lobo et al., reported that about 72% of patients with spontaneous CSF leaks were female, and about 45% had obstructive sleep apnea.[19] Our demographic characteristics revealed that 100% of cases are of female gender, with a BMI above 30 (means obese patient) in 29 patients (78.4%), and 3 cases had sleep apnea.

Rapid proper management of these cases is crucial, as the risk of developing meningitis in untreated CSF rhinorrhea has been reported between 10% and 36%.[8,21,41] In the literature, conservative treatment in cases of spontaneous CSF rhinorrhea, especially those cases following raised intracranial tension, is not successful as well as that

following trauma. After the diagnostic lumbar puncture, CSF rhinorrhea often temporarily improves and recures again unless definite treatment of raised tension is applied. [9,37]

Radiological findings in cases of spontaneous CSF rhinorrhea secondary to IIH are the same as classic IIH as empty sella and venous sinus occlusion but it differs in the picture of skull base erosion and meningencephaloceles.<sup>[2]</sup> The most common sites of the skull base that are affected and eroded with increased pressure are the lateral lamella of the cribriform plate and posterior ethmoid as it slopes to form the rostrum of the sphenoid. [30] This is consistent with our finding, as the cribriform plate was the most common site of defect in our cases [Table 5]. The dura of the anterior cranial base is the most liable skull base dura to be affected by raised ICP, as it is the thinnest dural thickness among skull base dura. [10] Furthermore, it is subjected to wide variations and changes in CSF pressure because of several factors, including normal arterial and respiratory fluctuations, in addition to Valsalva-like maneuvers during nose blowing or straining, which could lead to dural tears in areas of abnormalities of the bony floor. [28] Despite we did not encounter this in those patients who had skull base repair, the investigators suggested that intracranial hypertension may put patients at risk for developing multiple skull base defects.[20,39] Study by Lieberman et al. found evidence of a significant incidence of multiple simultaneous skull base defects in cases of spontaneous CSF rhinorrhea, reporting the existence of such defects in eight out of 44 patients (18.2%) in the study.[17]

In MRV studies, we did not encounter actual venous sinus thrombosis but attenuated transverse sinus (venous attenuation sign). Many reports consider venous attenuation signs as a sign of IIH. There are theories about the role of this stenosis in the etiology of IIH. Jugular vein stent angioplasty was reported as controlling intracranial hypertension in some patients.[24]

The optimum management strategy for spontaneous CSF rhinorrhea with IIH is not well established. CSF diversion by shunt then patient observation was reported by many authors in the literature. [18,23,26,36] Higher failure rates of skull base repair in the presence of elevated intracranial tension were reported in many series. [4,5] We recommend the insertion of a lumboperitoneal shunt and sending the patient for observation in cases of CSF rhinorrhea with elevated pressure. We did not encounter infection or intracranial pneumocephalus during follow-up. Skull base repair is kept for recurrence of CSF rhinorrhea despite the patency (not obstructed) of the shunt. Many authors advocate skull base repair first followed by lumboperitoneal shunt if pressure is high or failure of the repair. [14] We believe that skull base repair in the presence of elevated CSF pressure may lead to fulminant raise of

intracranial tension or failure of skull base repair. This is consistent with reports from various studies revealing a high recurrence rate if skull base repair is performed or the appearance of signs of IIH before controlling intracranial tension.[12,36]

#### CONCLUSION

Spontaneous CSF rhinorrhea is considered secondary to IIH until proven otherwise. Treatment of the raised ICP by CSF shunting without skull base repair can solve CSF rhinorrhea and control and, on many occasions, may eliminate the need for skull base repair.

# Ethical approval

The Institutional Review Board approved the research/study at the Faculty of Medicine, Assiut University, Egypt, number 04-2024-300436, dated June 04, 2024.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

# Financial support and sponsorship

Nil.

#### Conflicts of interest

There are no conflicts of interest.

# Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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