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CASE REPORT

Recurrent Cerebrospinal Fluid Rhinorrhea Secondary to a Seizure-Induced Skull-Base Defect: An Unusual Complication of Epilepsy

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Abstract: Cerebrospinal fluid (CSF) rhinorrhea is a rare but serious condition that most commonly follows skull-base trauma or surgery, and its manifestation as a complication of epilepsy is exceptionally rare. Epilepsy management focuses on seizure control and prevention of immediate complications, such as status epilepticus or fall-related injuries. We describe a 36-year-old male with focal-onset epilepsy who developed recurrent CSF rhinorrhea temporally associated with nocturnal convulsions and a seizure-related cribriform-plate defect confirmed by magnetic resonance imaging and β-2 transferrin testing. We hypothesize that repeated minor cranial trauma and transient intracranial pressure surges during these seizures precipitated the skull-base defect. All alternative etiologies were systematically excluded; although a definitive causal link cannot be proven, the strong temporal association supports this hypothesis. The CSF leak was successfully addressed through endoscopic surgical repair, and preventive measures, including alterations to sleeping arrangements, were implemented. This case underscores the importance of considering CSF leakage in individuals with epilepsy presenting with unexplained nasal discharge, highlighting the need for enhanced seizure management, prompt recognition, and multidisciplinary care to prevent complications.

Keywords: CSF rhinorrhea, epilepsy, minor trauma, skull base defect

Introduction

Cerebrospinal fluid (CSF) leakage through the nasal passage, ie, CSF rhinorrhea, is an uncommon, yet medically important, condition.[1] Although CSF rhinorrhea commonly results from severe trauma, surgical mishaps, and congenital defects,[1] leaks have also been linked to idiopathic intracranial hypertension (IIH), neoplastic or infectious erosion of the skull base, connective-tissue dural weakness, and meningocele formation.[1,2] Reports of CSF rhinorrhea in patients with epilepsy are exceptionally rare and, when described, have invariably occurred after cranial surgery or alongside pre-existing encephaloceles.[3,4] To my knowledge, no published cases document a CSF leak arising de novo in close temporal relation to a seizure, in the absence of clear antecedent factors. Epilepsy, a long-term neurologic disorder characterized by recurrent unprovoked seizures, is often managed with a focus on controlling seizures and preventing immediate complications, such as status epilepticus and fall-related injuries.[5]

In this report, we describe a patient with focal-onset epilepsy who developed recurrent CSF rhinorrhea in close temporal relation to nocturnal seizures, and we hypothesize that transient ictal surges in intracranial pressure acting on a skull base already weakened by prior subclinical impacts precipitated an osteodural defect of the cribriform plate through which CSF escaped. This case, therefore, illustrates how seemingly minor seizure-related forces, particularly when accompanied by brief pressure spikes, may culminate in structural bone and dural damage leading to CSF leakage, even though a definitive causal link cannot be confirmed from a single observation.

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Case Presentation

A 36-year-old Middle Eastern man (height, 169 cm; weight, 63 kg) with a history of focal-onset epilepsy presented with recurrent unilateral clear nasal discharge. His first witnessed convulsive seizure occurred in March 2021, and subsequent outpatient video-electroencephalographic (EEG) recording led to a formal diagnosis of epilepsy in May 2021; this event was ultimately classified as a focal seizure with secondary generalization. He reported that for approximately one year prior (since early 2020) he had experienced undiagnosed nocturnal episodes with unwitnessed convulsions, often waking up on the floor. His seizures occurred exclusively during sleep and were characterized by a sudden vocalization followed by generalized convulsion with tongue biting, upward eye deviation, and urinary incontinence. He was started on levetiracetam (500 mg) twice daily in May 2021; the dose was increased to 1000 mg twice daily by August 2021. On the lower dose he experienced seizures approximately every 2–3 months. After escalation, seizures recurred only when clear precipitants were present: a missed evening dose resulted in a breakthrough convulsion on March 12, 2022, and several nights of poor sleep preceded another convulsion on January 7, 2024. Apart from these two events, he reported excellent adherence and required no further dose adjustments. Approximately one month after the March 2022 seizure he developed a two-week episode of clear, watery nasal discharge that resolved spontaneously, without headache, pulsatile tinnitus, visual disturbance, fever, or other sinonasal symptoms. Nearly two years later, in January 2024, a similar unilateral nasal discharge reappeared 48–72 hours after the nocturnal convulsion and again resolved without intervention. Despite the convulsive nature of his seizures, he had no history of significant head trauma, although he occasionally awoke on the floor beside his bed after an event. He also had no history of meningitis, prior cranial surgery, or previous CSF leakage. His family history was unremarkable for epilepsy or other neurological disorders.

On evaluation during the 2024 episode, the patient was alert, oriented, and hemodynamically stable, with no focal neurological deficits. Examination of the nasal secretions revealed a positive “halo” sign on filter paper, raising suspicion of a CSF leak. Biochemical analysis of the nasal fluid confirmed the presence of beta-2 transferrin, establishing that the discharge was CSF. Routine laboratory test results were within normal limits. An interictal EEG demonstrated rare medium-to-high voltage epileptiform discharges over the left frontal region, time-locked with sleep K-complexes, consistent with focal seizures with secondary generalization (Figures 1 and 2). High-resolution magnetic resonance imaging (MRI) of the brain in February 2024 revealed a CSF collection tracking to a skull-base defect through the cribriform plate (Figure 3). Notably, a brain MRI performed in June 2021 (shortly after his epilepsy diagnosis) had been reportedly normal, though those images were obtained at an outside facility and were unavailable for review.

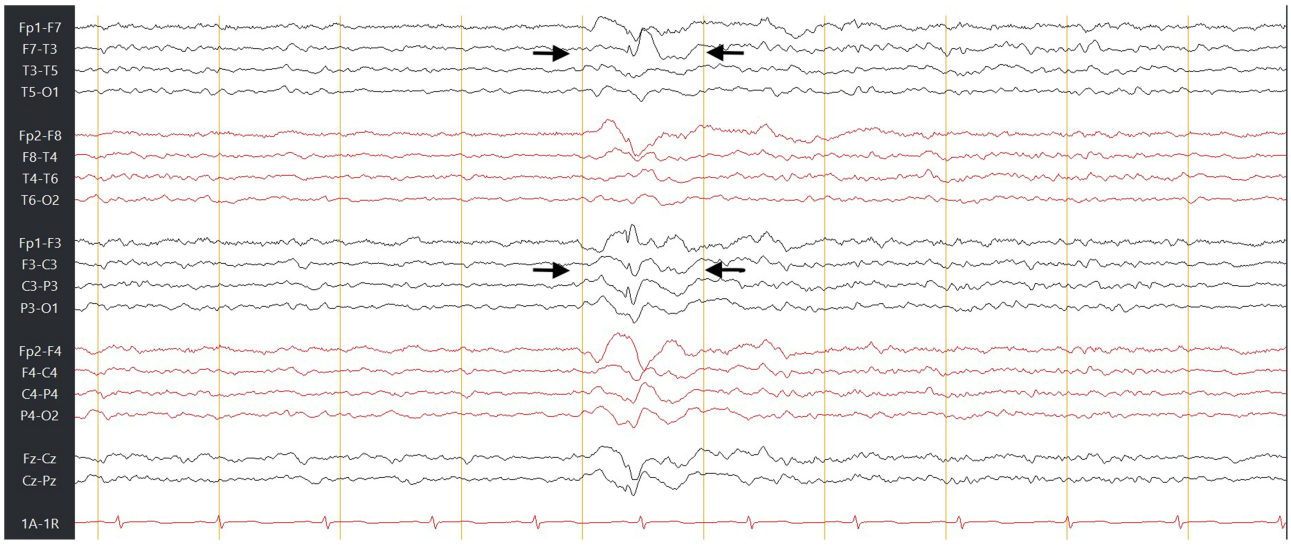

Figure 1 Electroencephalography (EEG) recording during sleep in a bipolar montage. EEG confirms a left frontocentral focus with a spike and wave complex. The epileptiform discharge shows phase reversal prominently localized at the F3-C3 electrodes, with a field also evident over the F7-T3 electrodes. This discharge is integrated within a concurrent K-complex. EEG settings: sensitivity, 7 μV/mm; high-pass filter, 1 hz; low-pass filter, 70 hz; notch filter, 60 hz; paper speed, 30 mm/s; and electrode placement, standard 10–20 system.

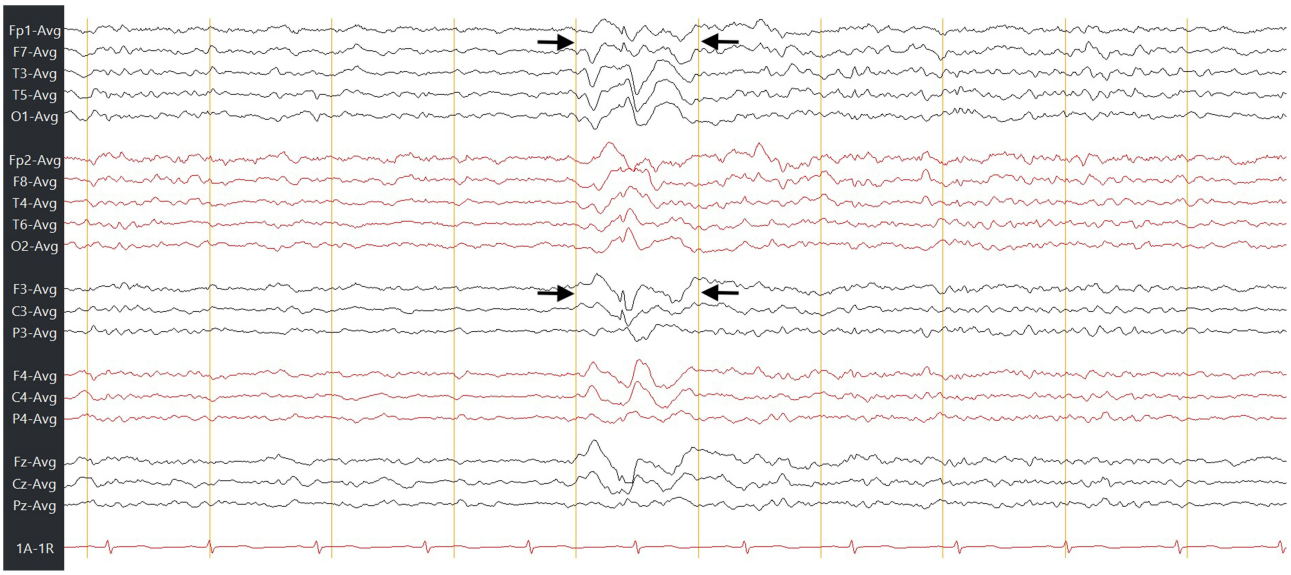

Figure 2 The electroencephalography (EEG) epoch shown in Figure 1 recorded in an average reference montage. This image shows the same discharge. Maximal field negativity remains consistent at the F3 electrode, with secondary involvement at the C3, F7 and Fp1 electrodes. EEG settings: sensitivity, 7 μV/mm; high-pass filter, 1 hz; low-pass filter, 70 hz; notch filter, 60 hz; paper speed, 30 mm/s; and electrode placement, standard 10–20 system.

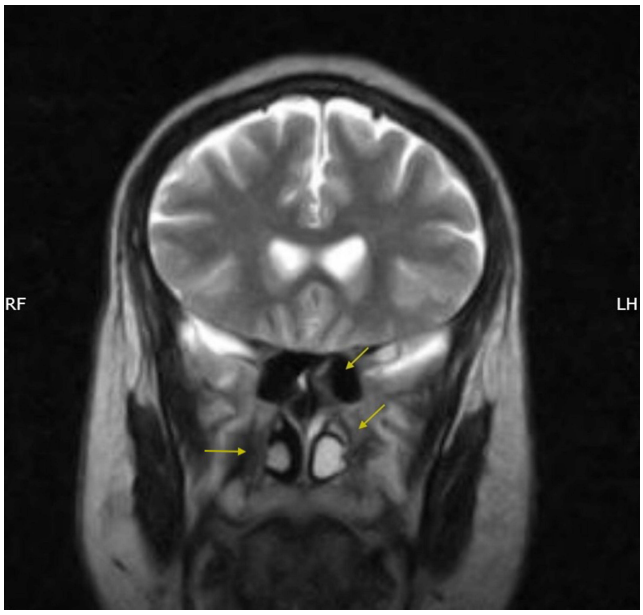

Figure 3 Coronal T2-weighted magnetic resonance imaging (MRI). MRI showing cerebrospinal fluid (CSF) pooling (arrows) along the anterior nasal cavity, extending through the cribriform plate bilaterally, but predominantly on the left side. This image highlights the defect at the cribriform plate and the associated fluid collection indicative of CSF rhinorrhea.

The patient was referred to otolaryngology and subsequently underwent endoscopic endonasal repair of the skull-base defect in late March 2024. Preventive measures were reinforced, including counseling on proper sleep hygiene and transitioning to a lower, wider bed to minimize the risk of trauma from any future seizures. At a one-year follow-up in March 2025, he had experienced no recurrence of CSF rhinorrhea and remained seizure-free on continued levetiracetam therapy.

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Discussion

The present case study examined the symptoms, diagnostic difficulties, and treatment approaches for seizure-related CSF rhinorrhea. It expands the range of potential complications of epilepsy, including structural cranial damage and CSF leakage. The patient's clinical progression demonstrates how inadequately controlled seizures, even when infrequent, may result in cumulative trauma and rare complications that may be overlooked in routine epilepsy management. Even when overall seizure frequency is low, nocturnal convulsions can deliver repetitive, subclinical head impacts that accumulate over time; these microtraumas may silently weaken the cribriform plate until cerebrospinal fluid rhinorrhea becomes clinically apparent.[6] This case also underscores the need to consider this uncommon complication in individuals with epilepsy who present with unexplained nasal discharge. By highlighting this unusual cause, we aimed to improve clinical recognition of this complication and promote collaboration to enhance patient outcomes. Additionally, this report raises awareness among healthcare professionals about this complication and its implications for epilepsy management.

In evaluating the present patient, all other common causes of CSF rhinorrhea were systematically ruled out. He had no history of head trauma or prior sinus or skull-base surgery, eliminating traumatic or iatrogenic etiologies. High-resolution imaging revealed a focal anterior skull-base defect without a tumor, clinical evidence of a chronic infection, congenital malformation, meningoencephalocele, or diffuse thinning. Ophthalmologic examination showed no papilledema, brain MRI showed no empty sella, and lumbar puncture results indicated a normal opening pressure, excluding idiopathic intracranial hypertension. No phenotypic features of a connective-tissue disorder were present. Thus, traumatic, post-surgical, IIH-related, congenital, neoplastic, inflammatory, and connective-tissue associated causes were excluded. In the absence of any other explanation, the tight temporal relationship between recurrent nocturnal seizures and CSF leakage suggests that repeated minor cranial trauma combined with transient seizure-related intracranial pressure surges most plausibly precipitated the defect.

Although trauma-related cases are well documented, to my best knowledge, no previous reports have described CSF leakage as an indirect complication of epilepsy due to a seizure-induced skull-base trauma without a pre-existing abnormality. This case presents an uncommon cause where paroxysmal, mild trauma during nocturnal seizures caused a skull-base defect, leading to CSF leakage. The anterior cranial fossa, especially the cribriform plate, is susceptible to fractures under continuous stress because of its delicate structure.[7] The patient's condition involved recurrent minor injuries during nocturnal seizures. Tonic–clonic convulsions transmit repetitive acceleration-deceleration that cause skull-base impacts and concentrate stress on thin regions, such as the cribriform plate. Under cyclic sub-fracture loading, cortical bone accumulates microcracks and loses stiffness; if remodeling cannot keep pace, these microdefects coalesce into full-thickness breaches.[6,8] In delicate, poorly vascularized areas like the cribriform plate, such focal bone discontinuities have been implicated in spontaneous CSF rhinorrhoea.[9] Therefore, low-amplitude but repetitive impacts can generate a clinically meaningful skull-base defect over time, explaining the present patient's presentation even in the absence of major trauma. Additionally, this case illustrates that inadequately managed epilepsy can result in complications beyond immediate concerns, such as status epilepticus and fall-related injuries. While seizure-related injuries usually involve soft-tissue trauma or long-bone fractures, a narrative review of more than 12,000 seizure-related head injuries identified only one skull fracture without mention of CSF rhinorrhea, underscoring the rarity of this outcome.[10] Recent omics-based studies in epilepsy have further shown that repeated seizure activity alters expression of matrix metalloproteinases, pro-inflammatory cytokines, and angiogenic factors within meningeal and bone tissues. These molecular changes can promote dural fragility and bone remodeling. For instance, transcriptomic profiling[11] and targeted hub gene analysis[12] have identified seizure-associated upregulation of pathways linked to extracellular matrix degradation, blood–brain barrier dysfunction, and chronic inflammation. These findings support a multifactorial mechanism wherein mechanical strain and a seizure-induced pro-degradative tissue environment converge to weaken skull-base integrity and predispose the patient to CSF leak.

The lack of substantial head trauma history in the present case highlights how mild head impacts during nocturnal seizures can accumulate, resulting in considerable cranial damage. This underscores the importance of considering subtle head injuries in patients with poorly managed seizures and implementing protective measures, eg cushioned bed rails or low beds.

Untreated CSF leaks pose substantial risks, including bacterial meningitis and intracranial infections, due to direct communication between the sterile subarachnoid space and external environment.[13,14] Persistent leaks can alter intracranial pressure dynamics, potentially exacerbating seizures in patients with epilepsy. These complications threaten

neurological stability and impose psychosocial burdens through recurrent hospitalizations and stigma associated with nasal leakage. Treating seizure-induced CSF rhinorrhea requires addressing structural issues and underlying seizures. Endoscopic endonasal procedures offer minimally invasive repair of skull-base defects, sealing of dural tears, and halting leakage. Prompt surgical intervention alleviated symptoms in the present patient, with no recurrence at the one-year follow-up.[15] Optimizing seizure management involves re-evaluating anti-seizure medications and considering alternative treatments if needed.[16,] Risk mitigation for nocturnal convulsions can be enhanced through lifestyle adjustments, such as using lower beds or protective padding.

A recent systematic review on the management of recurrent CSF rhinorrhea caused by skull-base defects highlights the importance of a comprehensive diagnostic approach.[15] One study emphasized reassessing imaging studies and performing a structured diagnostic workup to identify sequential CSF leaks occurring independently of the primary lesion.[15] This approach is recommended to improve the detection and management of this rare but complex condition.

This report highlights crucial points for medical professionals treating patients with epilepsy. Medical professionals should remain highly vigilant about unexplained nasal discharge in patients with epilepsy and implement protective measures to reduce trauma during seizure episodes.

Conclusion

This case highlights the necessity of identifying seizure-induced CSF rhinorrhea as an uncommon yet possible complication in patients with epilepsy. By documenting a leak arising de novo without evidence of secondary etiologies, and in temporal proximity to seizures, the present report broadens the recognized spectrum of epilepsy-related morbidity and alerts clinicians to a structural consequence that may be overlooked. As leaks that form during periods of poor seizure control may only become apparent once control improves, clinicians should maintain a high index of suspicion for CSF rhinorrhea even when seizures appear well managed. Recurrent nocturnal seizures may cause progressive cranial damage, potentially resulting in skull-base defects and CSF leakage. Additional research is required to investigate risk factors and preventive strategies for seizure-related head injuries, determine the frequency of CSF leakage, and identify predisposing factors that may increase susceptibility.

Abbreviations

CSF, cerebrospinal fluid; IIH, idiopathic intracranial hypertension; EEG, electroencephalography; MRI, magnetic resonance imaging.

Ethics Approval and Informed Consent

The study was approved by the Institutional Review Board (IRB) of Imam Abdulrahman Bin Faisal University on March 09, 2025 (IRB-2025-01-0190), and we confirm that institutional approval was required for publication of the case details and its associated figures.

Consent for Publication

The patient provided written informed consent for the publication of this case report and its associated figures.

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Disclosure

The author reports no conflicts of interest related to this work.

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