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Original Research Paper

General Medicine

CSF RHINORRHEA

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ABSTRACT Cerebrospinal fluid (CSF) rhinorrhea refers to the loss of CSF through the nasal cavity. Its causes can be classified as either spontaneous or non-spontaneous. Spontaneous causes of CSF rhinorrhea include congenital anatomical defects and are extremely rare, accounting for less than 4% of reported cases. Following failure of conservative management, definitive treatment most commonly involves an endoscopic transsphenoidal repair of the defect. I present four cases, first case is of a 53 years old male with history of old head injury following which he had recurrent bacterial meningitis and second case is of a 42 years old female showing incidence of spontaneous csf rhinorrhea developing after acute ischemic stroke

KEYWORDS :

INTRODUCTION

Spontaneous cerebrospinal fluid (CSF) rhinorrhea is a rare disease; its exact cause is not yet fully understood, and is usually related to congenital temporal bone, skull base, and dural malformations and defects. The combination of a pre-existing weakening of the meninges and sudden violence can also cause CSF rhinorrhea. Intracranial pressure fluctuations cause gradual herniation of the dura mater into the bone fissure, which with time causes thinning of the dura. A weakened dural structure may easily lead to the formation of diverticula or expansions, and increase the possibility of dural tear formation, ultimately resulting in CSF leakage into the epidural space. In patients with no history of trauma, nasal leakage is often overlooked.

The causes of cerebrospinal fluid (CSF) rhinorrhea can be classified as either spontaneous or non-spontaneous. Spontaneous or non-traumatic causes include congenital anatomical defects related to the temporal bone, skull base, or dura mater [1]. Non-spontaneous or traumatic causes of CSF rhinorrhea are related to surgical and accidental trauma, tumors, or exposure to radiation therapy involving the base of the skull [1]. Early diagnosis and effective management are needed to prevent the life-threatening complications of CSF rhinorrhea, including bacterial meningitis and brain abscesses.

Bacterial meningitis is a severe, potentially life-threatening infection that is associated with high rates of morbidity and significant disability in survivors. In recent years, despite improvements in antimicrobial therapy and intensive care support, overall mortality rates related to bacterial meningitis of around 20% to 25% have been reported by major centers. Potential long-term neurological sequelae include cranial nerve palsies, hemiparesis, hydrocephalus and seizures as well as visual and hearing impairment, which can have a profound impact on the quality of life of the survivors.

"Recurrent meningitis" is defined as being two separate episodes of meningitis that are separated by a period of convalescence and full recovery. Therefore, true recurrence results from a reinfection with the same or a different bacterial organism. In contrast, recrudescence and relapse represent persistence of the "original" infection resulting from treatment failure.

Case 1

this case shows complication of treating csf rhinorrhea conservatively in a patient of traumatic cribriform plate fracture

Patient Mr. Bansi Jadhav 53 years old Male came to casualty on 4th October 2021 with complaints of frontal headache, acute in onset, intermittent in nature in the past 4 months associated with giddiness not related to exertion, position, vomiting, photophobia, phonophobia. He also complained of new onset moderate degree fever since last 2 days

He had no history of runny nose, any known drug allergy or any history of upper respiratory tract infection, chest pain, palpitat ions, syncopal attacks, breathlessness and cough.

He is a known case of seizure disorder on treatment

Medication history: Tab. METOPROLOL-XL 25mg, Tab. LEVETIRACETAM 500mg, Tab. ATORVASTATIN 40mg, Tab. BETAHISTINE 16mg, Tab. ISOSORBIDE MONONITRATE 5mg. On 2nd October 2021 he got tested for covid RTPCR which was reported negative.

His MRI Brain from outside hospital was suggestive of few linear irregular enhancing FLAIR hyperintense exudates within cortical sulci over bilateral cerebral hemispheres, basal cisterns over surface of brainstem – meningitis to be ruled out

He has past history of bacterial meningitis with CSF rhinorrhea in January 2019.

He has past history of motorcycle road traffic accident 15 years back after which he developed runny nose which was ignored as it was thought to be due to simple cold but later when it was not responding to treatment was investigated and found to have a CSF rhinorrhea for which he was operated but despite that he had intermittent CSF rhinorrhea

In 2019 he has history of IHD for which thrombolysis with alteplase was done.

O/E: He had fever of 101-degree F, Pulse rate of 100bpm, Blood Pressure of 130/80mmHg,

CONCIOUS ORIENTED, NO FOCAL NEUROLOGICAL DEFICIT, NECK RIGIDITY+On Fundoscopy there was no evidence of papilledema in both of the eyes

On complete blood count analysis, he had leukocytosis, his fever profile and HIV, HbsAg, HCV markers were nonreactive

Lumbar Puncture was done for cerebrospinal fluid (CSF) studies suggestive of bacterial (pyogenic) meningitis

He was started on intravenous antibiotics: INJ. CEFTRIAXONE 2gm, INJ. VANCOMYCIN 1gm both for 1-month courses

On nasal endoscopy it was observed to have bilateral congested nasal cavity with 4 to 5 drops of clear fluid seen coming from left nasal cavity on bending forward

CT CISTERNOGRAPHY was done suggestive of no bony structural abnormality or any other positive findings.

He gradually improved after antibiotics course and symptoms resolved gradually.

His repeat Lumbar Puncture was done after completion of treatment and compared with admission sample

CSF ANALYSIS	5TH OCTOBER 2021	23RD OCTOBER 2021
TOTAL COUNTS	657 cells/cumm	67 cells/cumm
POLYMORPHS	45%	80%
LYMPHOCYTES	55%	20%
CSF PROTIEN	305.43 mg/dl	50 mg/dl
CSF GLUCOSE	48.9 mg/dl	76 mg/dl
CSF ADA	2.0 U/L	1.0 U/L

He was later discharged on course of antibiotics with advice of getting the procedure of ethmoidal plate repair done

Case 2

this case shows incidence of spontaneous csf rhinorrhea secondary to raised intracranial tension with underlying cribriform plate defect

Patient Savitri Rajule 42 years female came to MGM ER on 24/6/22 at midnight with complaints of sudden onset left sided upper and lower limb weakness with slurring of speech since 1 day back, weakness was sudden in onset associated with deviation of angle of mouth to right no associat ed with involuntary movements, fever, nausea, vomitting, trauma to head.

she is a known case of hypertension since 3 years She has past history of right parietal iscemic CVA with Cerebral venous sinus thrombosis in march 2021 and history of papiloedema with vision loss in June 2021.

She also gave history of endoscopic left cribriform plate repair for CSF leak with VP shunt on arrival her PR= 80bpm, BP= 130/80mmHg concious, pupils reactive to light equal with deviation of angle of mouth to right with power of 3 /5 of left upper limb and lower limb her ECG was suggestive of low voltage complex with T wave inversions

2Decho suggestive of hypokinesia of anterior and anteroseptal wall involving the distal IVS and apex

MRI suggestive of right ischemic MCA territory stroke she was started on antiplatelet, anticoagulation for initial 5 days and physiotherapy she was discharged later with advise to continue antiplatelet and physiotherapy

Case 3

22 years old male patient came to MGM on 5th november 2022 at 1 pm with complaints of sudden onset headache, behavioural changes associated with vomitting and fever spike within last 6 hours and was admitted in SICU.

There was history of RTA with trauma to head, face and neck 1 month back for which he was admitted in MGM , NCCT BRAIN showed frontal pneumocephalus and patient was admitted in SICU and managed conservatively with high flow o2 and started on phenytoin and later discharged, he later started having continuos runny nose for which after 1 month on follow up was suspected to be due to CSF rhinorrhea and a MRI BRAIN CISTERNOGRAM done suggestive of communicating hydrocephalus with pneumocephalus causing mass effect and midline shift of 5.6 mm to right with sphenoid sinus fracture and associated hemosinus with no obvious csf leak near cribriform plate.

he was vitally stable and conciuos oriented with normal bilaterally equal pupillary reactions he was started on tab diamox, inj eptoin, inj pantoprazole, inj emset, inj paracetomol and a painkiller on 18/11/22 he was taken up for endoscopic sinus repair for CSF rhinorrhea his lumbar drain and nasal pack was removed on 22 /11/22 and patient was given discharge and is aymptomatic since then.

Case 4

24 Years old male, asif shaikh, unmarried, autorickshaw driver, cigarette smoker, no comorbidities history to RTA with injury to head in 2014 after which he developed runny nose a year later he was operated for CSF rhinorrhea but still had runny nose in 2018 he presented to casualty with runny nose associated with recurrent episodes of headache and fever and vomitting and had one episode involuntary movement with loss of conciousness for which he was admitted in ICU and started on phenytoin and later endoscopic repair was done at MGM in 2018.

After sinus repair he didnt have any repeated episodes of similar episodes but was still having CSF rhinorrhea and seizure episodes and had a episode of loss of conciousness was hospitalised in 2021 and had developed phenytoin toxicity for which he was switched to tab Leveracetam.

CT BRAIN suggestive of sever pneumocephalus in fontal region showing air fluid level with operative defect of 6.4 by 1.4 cm and fracture of lamina papyracea and horizontal plate of frontal bone.

MRI BRAIN CISTERNOGRAPHY suggestive of focal defect noted in the roof of left frontal sinus with surrounding CSF leak from intracerebral compartment associated with meningoencephalocele and was re-operated for endoscopic sinus repair surgery after which the rhinorrhea stopped but he continued to have right sided head turning with tonic clonic limb positioning lasting for 2 mins 3 to 4 episodes every year for which he was started on tab valproate and tab lacosamide and is seizure free since then

DISCUSSION

Catastrophic consequences of an untreated CSF rhinorrhea are described historically. These include meningitis, intracranial hypotension, pneumocephalus, and intracranial abscess formation. Meningitis, the most commonly described complication, is seen to develop in about 6.75% of cases of a persistent CSF leak, and in turn may lead to potentially debilitating problems and a long-term consequences. Almost 80% of pneumocephalus cases are caused by trauma. Patients with idiopathic intracranial hypertension and spontaneous CSF leaks are highly prone to develop encephaloceles. Mortality rates secondary to traumatic brain injury with CSF rhinorrhea are known to be significantly higher than those of traumatic brain injury without a CSF leak. CSF rhinorrhea occurs due to an abnormal communication

between the subarachnoid space and a defect in the skull base, leading to the loss of CSF through the nasal cavity [1]. Spontaneous or non-traumatic CSF rhinorrhea is extremely rare and accounts for only 4% of all reported cases of CSF rhinorrhea [2]. Spontaneous CSF rhinorrhea has been associated with elevated BMI and intracranial hypertension (ICH) [3,4]. The pathogenesis of CSF rhinorrhea is unclear, but previous studies have hypothesized that prolonged ICH may lead to defects in the skull base over time. These defects coupled with ICH can cause herniation of the dura mater into the bony defects, weakening the dura mater and making it more prone to dural tears and thus leading to a dural-mucosal fistula [5]. Similarly, obesity causes increased intra-abdominal pressure, leading to the elevation of the diaphragm and thereby leading to increased pleural and cardiac pressures, thus decreasing venous return from the brain to the heart and causing ICH [3,6]. The patient in the present case had an elevated BMI and complained of headaches that were worse on bending over, pointing to possible ICH, although the patient had no signs of ICH on examination.

The gold standard for detecting the presence of CSF is by testing for beta-2 transferrin or beta-2 trace protein [7,8]. Beta-2 transferrin is exclusively found in CSF, perilymphatic fluid, and the vitreous humor of the eye, with a reported sensitivity of 100% and a specificity of 95% [7]. In the case of our patient, we were unable to assess beta-2 transferrin testing and resorted to using comparative blood glucose concentrations of the draining fluid to the blood. The presence of glucose in secretions indicates the presence of CSF. However, detecting the presence of glucose in secretions is not recommended as a confirmatory test due to low diagnostic specificity and sensitivity and due to false-negative results in the case of bacterial contamination or false-positive results in diabetic patients [7]. Therefore, detection of glucose in CSF rhinorrhea cannot be used on its own to diagnose a CSF leak and requires concurrent clinical and radiographic evidence [9].

Radiological diagnostic techniques include the use of high-resolution CT and MRI scans. High-resolution CT and MRI scans are the most reliable means of differentiation between spontaneous and nonspontaneous CSF rhinorrhea. CT and MRI can assist in localization of leaks, especially when associated with fractures of surrounding bone or tumors, but do not demonstrate the leakage itself [2,7]. CT/MR cisternography is the gold standard for the detection of CSF leaks as it can identify the size, location, and quantity of the leak but is an invasive procedure and thus considered unnecessary if the diagnosis is supported by both the clinical presentation and imaging findings on CT and MRI [7,10]. In the case of our patient, CT scan was found to be non-diagnostic. MRI with gadolinium enhancement was required to identify the leak and the diagnosis of CSF rhinorrhea was confirmed through MRI findings and the presence of glucose in the CSF.

Treatment of CSF rhinorrhea usually involves an initial conservative approach, which, if failed, is followed by a surgical approach. Conservative treatment of CSF leaks consists of the use of acetazolamide along with prolonged bed rest with elevation of the head, which can decrease intracranial pressure [11]. Surgical intervention can involve either an endoscopic/extracranial approach or an intracranial approach. Intracranial approach carries increased morbidity and failure rates of 20-40%, whereas an endoscopic approach has less associated morbidity and a success rate of 90-100% [12-14]. A study that looked at 193 cases of treated CSF leaks over a period of 21 years concluded that the overall success rate of endoscopic repair was 98%, which, combined with an associated low morbidity, reinforced endoscopic repair as the standard of care for repair of CSF leaks [15]. However, it should be noted that an intracranial approach comes with its own advantages, including wide visualization of the leak site, and allows for direct repair of the leak. The current recommendations suggest initial treatment of CSF leaks using endoscopic repair, with extracranial repair reserved if indicated or following failure of endoscopic repair [16]. Adjunctive techniques to surgery that are commonly used to decrease morbidity and increase success rates include the use of antibiotics, diuretics, lumbar drains, and prolonged bed rest with elevation of the head [11,17]. In the present case, we initially performed an endoscopic repair using a transnasal transsphenoidal approach with the placement of an autologous graft over the opticocarotid triangle. This was followed by an open surgical approach when the patient represented a week later with bilateral CSF rhinorrhea, where the defect in the sphenoid bone was repaired using a fascia lata graft. We were unable to identify the cause of the recurrence of the CSF leak following the initial endoscopic repair.

Early diagnosis and prompt treatment of CSF rhinorrhea are important to prevent complications such as meningitis, intracranial sepsis, and abscesses, which are associated with high mortality rates. The overall rate of ascending meningitis associated with CSF leaks is 19% [18]. Currently, there is no evidence for the use of prophylactic antibiotics in the prevention of meningitis [19]. With successful surgical repair of CSF leak and an uneventful postoperative period, patients usually have a favorable prognosis. It is important to note that patients with CSF leaks are required to have 13-valent pneumococcal conjugate vaccine (PCV-13) and 23-valent pneumococcal polysaccharide vaccine (PPSV-23) eights months apart especially in the case of cranial CSF leaks due to the presence of a communication between the brain and surrounding structures and the oropharynx and nasopharynx [20].

Recurrent bacterial meningitis is a rare phenomenon and generally poses a considerable diagnostic challenge to the clinician. Ultimately, a structured approach and early diagnosis of any underlying pathology are crucial to prevent further episodes and improve the overall outcome for the affected individual. On reviewing the existing literature on this topic over the last two decades, encompassing 363 cases of recurrent bacterial meningitis described in 144 publications. Of these cases, 214 (59%) were related to anatomical problems, 132 (36%) were related to immunodeficiencies, and 17 (5%) were related to Para meningeal infections.

CONCLUSION

Spontaneous CSF rhinorrhea is a rare condition usually associated with an elevated BMI and ICH. The gold standard for the detection of CSF in secretions is beta-2 transferrin testing; however, CT/MR cisternography is the imaging test of choice as it can accurately identify the CSF leak. Prompt diagnosis and treatment of CSF leaks are necessary to prevent complications including ascending meningitis, which is associated with high mortality rates.

Recurrent meningitis occurs mainly in patients with ear or sinus infections and cerebrospinal fluid leakage. Predominant causative pathogens are S. pneumoniae and H. influenzae. The disease course is less severe, resulting in lower case fatality compared with nonrecurrent meningitis patients.

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