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Is CT Cisternography in CSF Rhinorrhea Really Antiquated? Clinical Review of 14 Cases with Pictorial Depiction

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ABSTRACT

CSF rhinorrhea is a complex problem due to the intricacy of anatomy of the cranio-facial region and precision required in diagnosis and management. Detection of accurate site of leak is of paramount importance in this era of minimally invasive procedures. CT Cisternography provides us with both the site of leak and visualization of defect. CT Cisternography of late has lost its appeal with advent of Magnetic Resonance Cisternography, which is usually performed in combination with High Resolution Contrast Tomography (HRCT). In our series we pictorially depict various findings of CSF leak on CT Cisternography particularly emphasizing on its role in current scenario.

Keywords: Cerebrospinal fluid (CSF), Computed Tomography (CT), Computed Tomography Cisternography (CT C), High resolution Contrast Tomography (HRCT), Magnetic Resonance Cisterongraphy (MR C)

INTRODUCTION

CSF (Cerebrospinal Fluid) rhinorrhea was first reported as a physiological phenomenon by Galen in 18th century [1]. However, it was in mid-19th century that the complications of CSF leak like meningitis and abscess formation were identified, thus emphasizing the need for intervention [2-4].

An osseous and dural defect in the region of skull base resulting in communication between subarachnoid space and extracranial space usually the paranasal sinuses results in CSF rhinorrhea. CSF rhinorrhea is complex problem due to intricacy of regional anatomy and precision required in diagnosis and management. With advent of minimally invasive surgical technique exact localization is of paramount importance. Imaging modalities for detection of CSF rhinorrhea have evolved over time and include radionuclide study, CT Cisternography (CT C), completely noninvasive Magnetic resonance Cisternography (MR C), Contrast enhanced MR Cisternography and varying combination of these.

CT Cisternography is an imaging modality that involves injection of iodinated contrast into the spinal subarachnoid space and then imaging the cranial subarachnoid space using HRCT. It demonstrates the bony defect and also shows the leak. Of late it has fallen in disrepute due to the advent of MRI cisternography which is noninvasive and comprises of thin section heavily T2W contiguous images. As MRI is inadequate to demonstrate bony defect, it is usually combined with HRCT PNS to detect the bony defect.

We shall herein present a series of 14 cases of CSF Rhinorrhea in whom CT Cisternogram was performed, emphasizing on the current role of CT Cisternogram, along with review of clinical and imaging findings.

MATERIALS & METHODS

We retrospectively searched for cases of CSF rhinorrhea evaluated with CT Cisternography done over the past three years from September 2013 to September 2016 in the Hospital Information System-Picture Archiving and Communication System (HIS-PACS) system of our hospital. After excluding those with inadequate clinical and surgical records we found about 14 patients.

All the patients were evaluated by the otolaryngologist and neurologists of our hospital, the CSF leak was confirmed by clinical and biochemical investigations of nasal discharge.

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Imaging was performed during clinically active CSF leak, as reported by the patient. In all cases, high-resolution CT and contrast-enhanced CT Cisternography were performed on the same day.

CT scan was carried out on 128 slice Somatom definition edge, Siemens Health care, Erlangen., Germany. All the patients initially underwent non-contrast high-resolution CT of paranasal sinuses from the level of hard palate superiorly including the frontal sinuses in cranio-caudal direction using slice thickness of 1 mm. Sub-millimetric reconstructions (0.5 mm) in bone and soft tissue kernels were performed.

Prior to starting the procedures pledgets one in each nostril were introduced. After lumbar puncture, 8-10 mL of an iodinated nonionic low-osmolar contrast agent (iopropamide/iohexol, 300 mg/ml iodine) was instilled into thecal sac. The patients were then placed in knee elbow position with dependent flexed head to provoke CSF leak andcontrast enhanced CT scan was performed with collimation similar to non-contrast CT.

The images were evaluated by two radiologists (7- and 15-years' experience) and discrepancies if any were settled by consensus.

On plain study note was made of site of visible defects, fractures in case of trauma, presence of pneumoencephalous and sinus opacification /extracranial fluid in communication with CSF.

On contrast study an increase in 50% of HU value of the paranasal sinus fluid /extra cranial fluid as compared to non-contrast study [2], a column of contrast extending from subarachnoid space to the extra cranial space (paranasal sinus) or opacification of the nasal pledget was considered positive for leak. Pooling of contrast material at a fracture site with deformity of a local sulcus as described by Fagerlund and Liliequist [5] was considered positive. Additonally, pneumoencephlous in communication with para nasal sinus air was also considered positive for CSF leak.

The demographic data, clinical presentation, treatment details of surgical or conservative treatment were evaluated and tabulated.

RESULTS

We retrospectively evaluated 14 patients who underwent CT Cisternography from the year 2013 to 2016. The medical records, surgical details, investigations and CT Cisternography images were evaluated. Of the 14 patients 9 were females mean age was 45.5 with age ranging from 29 years to 56 years. The procedure was tolerated well in most patients with minor headache controlled by NSAIDS and head rising in most, while the headache was severe in one patient warranting admission for 2 days post procedure. The demographic distribution, imaging and surgical details are as depicted in **Tables 1 and 2**.

All patients presented with typical history of persistent clear nasal discharge, which could not be sniffed back, and which increased on forward bending. The duration of symptoms ranged from 1 day to one and a half year. 2 of 14 patients one with spontaneous leak and the other with post traumatic leak had signs and symptoms suggestive of meningitis. The cause was non traumatic in 11 of 14 patients, while in others the cause was trauma accidental (n=3) or iatrogenic (n=1). Over all the cribriform plate and sphenoid sinuses were equally involved (n=5).

Of the patients with non-traumatic leak, thecribriform plate (n=5/11) was most commonly involved, the next common site was sphenoid sinus (n=4/10). Left cribriform plate was more frequently involved than left (n=3/5). In all patients with leak fromcribriformplate the defect was clearly appreciated and a continuous column of contrast extending from the subarachnoid space to the extra cranial space (intofrontoethmoidal recess) was noted with opacification of nasal swab **(Figure 1)**. One patient had two sites of leak from both the Fovea Ethmoidalis and Cribriform plate **(Figure 2)**, while in the other two no leak or defect could be appreciated. Of the four cases of sphenoid defect, one patient had defect in the inferolateral wall also known as Sternsberg Canal **(Figure 3)** and other three in the posterior wall **(Figure 4)**. These leaks were seen as continuous extension of contrast from subarachnoid space to the sinus **(Figures 2 and 3)**. In one patient significant spontaneous penumoencephalus prevented opacification of subarachnoid space. The defect was easily identified on HRCT with subarachnoid air seen communicating with para nasal sinus air **(Figure 5)**. All the patients with cribriform leak or absence of obvious leak on CT Cisternogram were treated conservatively and it subsided spontaneously, while those involving sphenoid sinuses were treated surgically **(Table 1)**.

The post trauma group of the 3 patients comprised of one iatrogenic leak and two post traumatic leaks. One patient presented immediately post trauma and CT Cisterogram was done within a week of trauma. This patient had fracture of anterior and middle cranial fossa and pneumoencephaolus with extra dural extravasation of contrast reaching upto the fracture with deformity of an adjacent sulcus **(Figure 6)**. This patient was treated with fracture fixation and duroplasty. The other patient has past history of road accident and nasal bleed. He presented with one-and-a-half-year history of CSF rhinorrhea. CT Cisternogram showed multiple defects in frontal sinus and roof of sphenoid and ethmoid sinus. Leak was however seen from the roof of ethmoid and sphenoid sinus as increase in density of sinus fluid by

Table 1. Clinical, imaging and treatment details of all 14 patients.

No.	AGE	GENDER	PRESENTATION	DURATION	CAUSE	SITE OF DEFECT ON PLAIN	SITE OF LEAK ON CTC	PROCEDURAL COMPLICATION	TREATMENT
1	30	M	Nasal discharge	4mths	Non traumatic	None	None	Persistent headache required admission	Lost to follow-up
2	48	M	Nasal discharge	2mths	Non traumatic	None	None	None	Conservative
3	43	F	Retroviral positive, meningitis	15days	Non traumatic	Right Cribriform plate	Right Cribriform plate	None	Conservative
4	40	M	Hypotensive headache, discharge	1week	Traumatic	1) Multiple fractures of inner table of left frontal sinuses 2) Left Cribriform plate	Distorted sulcus sign near fracture of left frontal sinus inner table	None	Fixation of fracture with duroplasty and exteriorisation of frontal air cells.
5	29	M	Left nasal discharge	18mths	Delayed Traumatic	1) Left frontal sinus encephalocele 2) Roof of posterior ethmoid 3) Sphenoid air cells	Leak from ethmoid and sphenoid roof defect, increase in density of	None	Endoscopic endonasal CSF leak repair with fascia lata
6	56	F	Right nasal discharge	1 mth	Non traumatic	Posterior wall of sphenoid sinus	Leak into sphenoid sinus, sinus opacification	None	Endoscopic repair, meningocele cauterised, fat graft from thigh, middle turbinate
7	52	M	Nasal discharge	15days	Non -Traumatic	Posterior wall of sphenoid	Spontaneous pneumoencephalous in continuity with sphenoid sinus air through defect in posterior sphenoid wall, no opacification of	None	Fascialata graft, endoscopic repair

8	54	F	Meningitis , Nasal discharge	6days	Non traumatic	Right cribriform plate	Right Cribriform plate	None	Conservative	
9	54	F	Nasal discharge post-surgery	2days	Traumatic, Iatrogenic	1)Right lateral wall sphenoid sinus with adjacent sinus opacification 2) Right	Opacification of right mastoid air cells and Eustachian tube	None	Conservative	
10	38	F	Nasal discharge	6mths	Non traumatic	Right inferolateral sphenoid sinus defect	Leak into the sphenoid sinus via inferolateral wall defect	None	Fascialata graft +middle turbinate graft,	
11	50	F	Nasal discharge	15days	Non traumatic	Posterior wall sphenoid	Leak into the sphenoid sinus via posterior wall defect	None	Endoscopic transphenoidal surgery recommended/ patient not	
12	49	F	Nasal discharge	20days	Non traumatic	Left Cribriform plate	Left Cribriform plate	None	Conservative	
13	38	F	Nasal discharge	20days	Non traumatic	Left Cribriform plate	Left Cribriform plate	None	Conservative	
14	56	F	Nasal discharge	1mth	Non traumatic	1) Left cribriform plate 2) Left fovea ethmoidals	1) Left cribriform plate 2) Left fovea ethmoidali	None	Conservative	

M: Male, F: Female, mths : Months

more than 50% **(Figure 7)**. Frontal sinus encephalocele was also noted in this patient **(Figure 7)**. This leak was also surgically treated. We had one patient of iatrogenic cause of CSF rhinorrhea post microsurgery for trigeminal neuralgia. In this patient there was a right occipito-mastoid defect seen with extravasation of contrast into the defect and mastoid aircells, further extension into the middle ear and subsequently into the Eustachian tube and right nasopharynx were seen **(Figure 8)**. In this patient another defect was appreciated in the right lateral wall of sphenoid sinus with adjacent sinus opacification **(Figure 8)**.

DISCUSSION

CSF Rhinorrhea was classified by Oyammainto two groups traumatic (including iatrogenic) and non-traumatic [6]. He further subdivided the latter into nontraumatic with normal pressure and nontraumatic with CSF hypertension. The high-pressure leaks were those in whom the leak was secondary to congenital defects, infections or tumors resulting in raised intracranial tension [6,7]. While the other category that is low pressure leak is now identified as spontaneous leak and is thought to be a manifestation of benign intracranial hypertension considering its association with empty sella [7].

In literature few authors report traumatic CSF leak to be most common [8,9]. However, in our study we found the non-traumatic category to be common, which is consistent with research findings [10,11]. However, this could be because of the fact that plain HRCT is usually done for patients with significant head injury usually

Table 2. Etiology and site of leak.

	n
Females	9/14
Mean age (range)	45.5yrs (29-56yrs)
Site of leak Cribriform Sphenoid Fovea ethmoidalis Occpitomastoid Frontal	 6 5 1 1 1
Cause Traumatic Non traumatic	 3 11
Multiple defects	4/14
Multiple leaks	2/14
No leak no defect/sinus Opacification	2/14
No leak only defect present	1/14
Post procedure complication	1/14
Surgical treatment done/suggested	6/14

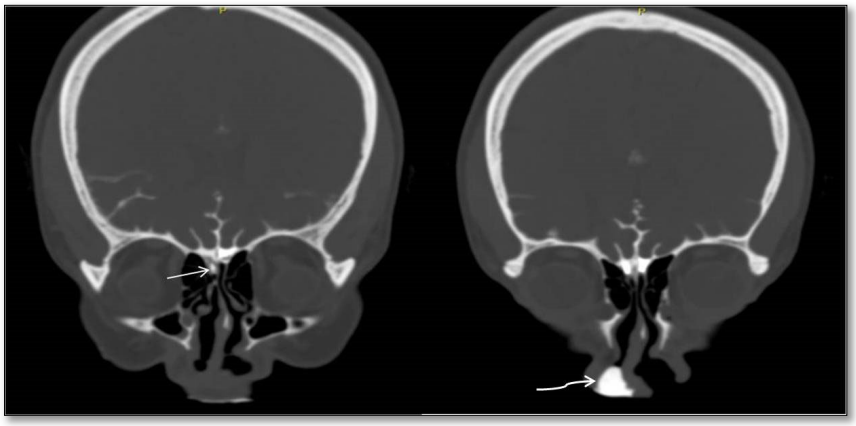

Figure 1. Linear column of contrast extending from the subarchnoid space through defect in right cribriform plate into the superior meatus (right frontoethmoidal recess) (white arrow).

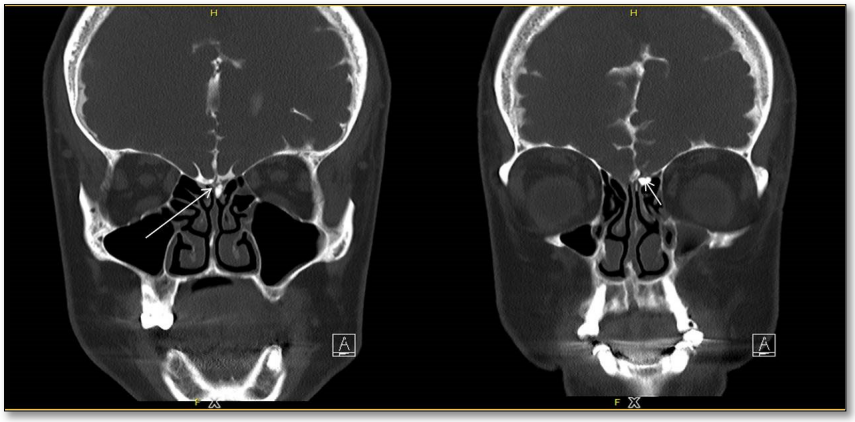

Figure 2. Two defects in Left cribriform plate (Long arrow) and left fovea ethmoidalis (Short arrow).

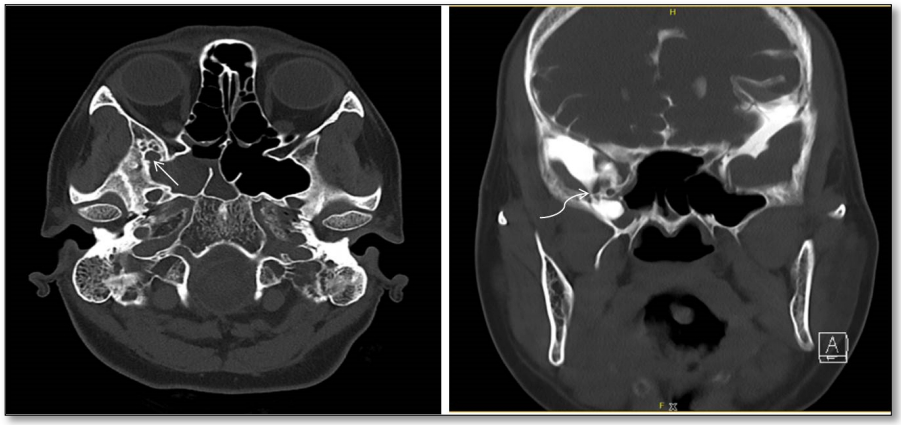

Figure 3. Defect measuring 3.5mm noted in the inferolateral wall of sphenoid sinus on the right side with minimal thinning of adjacent greater wing of sphenoid Sternbergs canal – persistent lateral cranio pharyngeal canal connecting intracranial space and sphenoid sinus due to congenital fusion defect between basisphenoid and greater wing of sphenoid .Contrast leak is seen through the defect from the base of right middle cranial fossa,pooling at the right sphenoid sinus (curved arrow).

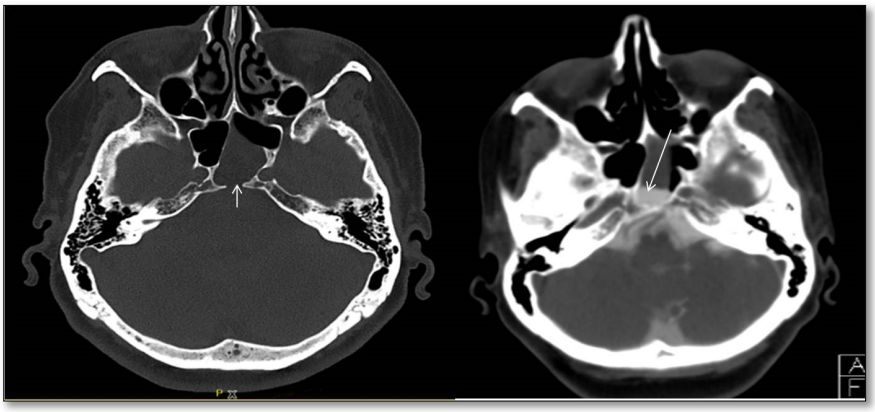

Figure 4. 5mm bony defect (short arrow) at that level in the posterior wall of left sphenoid sinus just above the clivus with CSF leak through defect. into sphenoid sinus (long arrow).

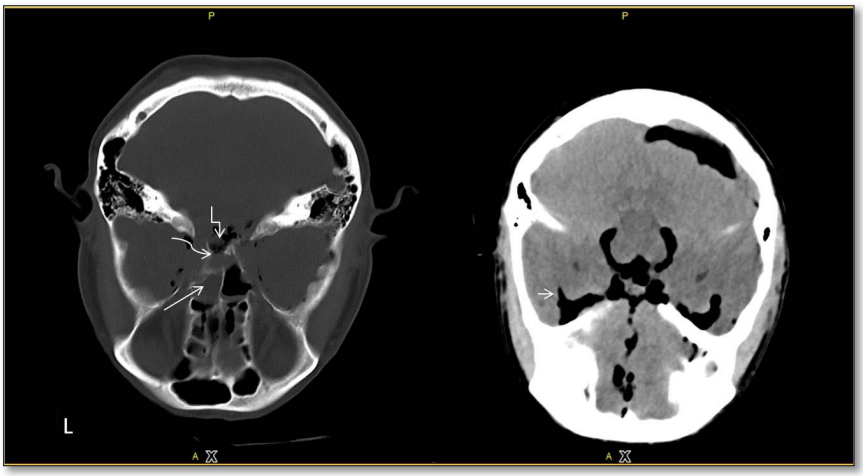

Figure 5. Extensive spontaneous pneumocephalus (short arrow) with fluid level in left sphenoid sinus (long arrow) with 6mm defect in the posterior wall of the sphenoid sinus above the clivus (curved arrow) with air pocket in the prepontine subarachnoid space communicating with the sphenoid sinus (elbow arrow) .No demonstrable flow of contrast from spinal to the brain subarachnoid space.

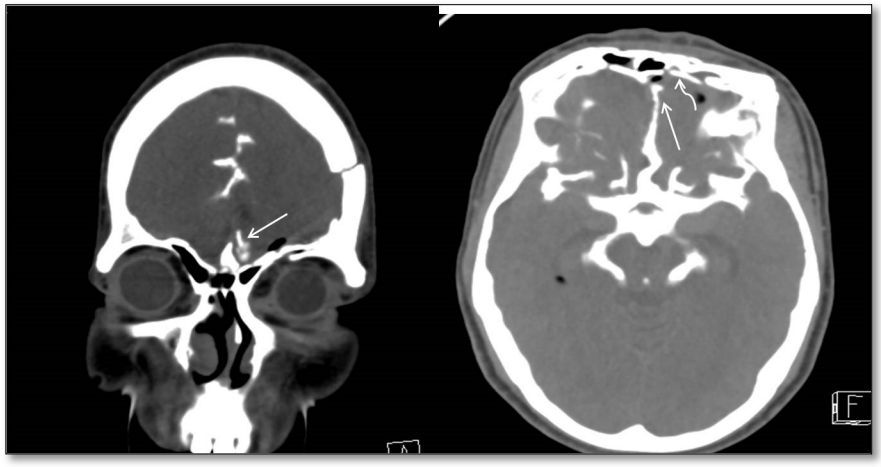

Figure 6. Contrast seen extending upto the fractures (curved arrow) in the inner table of left frontal sinus (straight arrow), distorted sulcus (straight arrow) with small pneumocephalus (curved arrow).

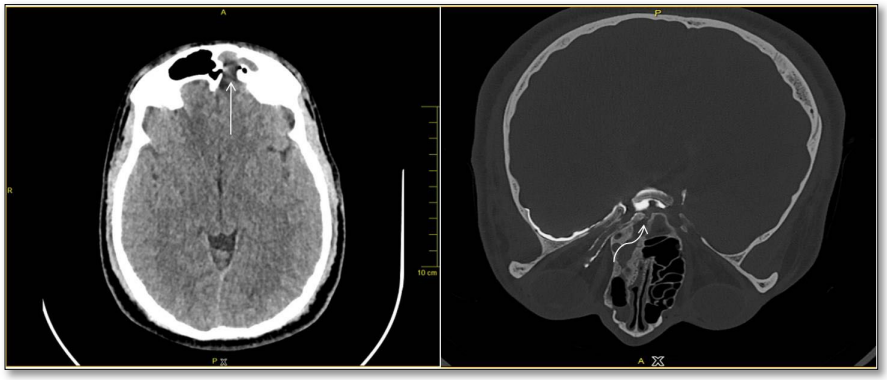

Figure 7. Defect in the inner table of left frontal sinus with herniation of brain tissue (straight arrow) but cisternogram showing CSF leak in the defect in the roof of the left sphenoid sinus and left posterior ethmoid sinus (curved arrow).

Figure 8. Post-surgery Right occipito-mastoid defect (long arrow), opacification of mastoid air cells and Eustachain tube (short arrow,defect in right lateral wall of sphenoid (curved arrow).

immediately after trauma, it usually suffices to detail the site of fractures in such cases and CT Cisternography is not feasible in acute settings.

The commonest site of leak was cribriform plate followed by sphenoid bone which was similar to other findings [8]. This is probably due to the fact that cribriform plate is thinnest and weakest portion of anterior cranial fossa [8,12]. All patients had typical findings of persistent nasal discharge which could not be sniffed back and increased on forward bending. Only two patients presented with meningitis, while hypotensive headache was present in all. Meningitis in the two cases was probably the reason of CSF rhinorrhea. Raised intracranial tension due to meningitis

was probably the cause for CSF leak in these patients rather than meningitis being a complication CSF leak.

Patients with CSF rhinorrhea pose a diagnostic challenge for both the otolaryngologists and radiologists. In this era of minimally invasive procedures where small skull base defects are routinely repaired using nasal endoscopes having limited field of view for, accurate localization of the leak is mandatory [3,8]. Also, symptoms in many patients with CSF leak resolve spontaneously or with conservative management [8]. Hence, we see that decision regarding management depends on the location and etiology. Plethora of imaging modalities have been described and discarded in an attempt to find the gold standard. The studies that are available for evaluation of CSF rhinorrhea are radionuclide study, HRCT alone, HRCT with CT cisternography, MRI cisternography with HRCT PNS and recently described CEMR cisternography. Of these the radionuclide study owing to its extremely low specificity is considered obsolete and is reserved only in cases where the diagnosis itself is in question [2,13]. There are few studies which say that HRCT alone suffices [14], but more than one site of leak and more than one defect are not rare. CT Cisternography has overtime undergone a drastic revolution due to the thin slice capabilities of current scanners and use of water-soluble contrast. It combines best of both worlds, that is it provides excellent localization of osseous defect and also shows the actual extra-cranial pooling of contrast. Of late it has lost its appeal with advent of MRI as it is slightly invasive in terms of requirement of lumbar puncture, patient discomfort in the form of post procedural headache, theoretical possibility of opening up of the defects, introducing infection, lumbar CSF leak and its low sensitivity in detecting the site in inactive leaks [3,15-17]. At present the most recommended modality is MRI Cisternography, HRCT PNS or combination of both which are non-invasive and quick [3,17]. In most of our patients we were able to isolate the site of leak with only mild patient discomfort. Post procedure complication rate was low and only one patient developed significant headache requiring admission.

We found that in patients who presented with recent trauma had multiple fractures on HRCT and pneumocephalus, which could interfere with visualization of leak on MRI. While with Cisternography though we could not see leakage into sinus of leak we could infer the possible site of leak using the distorted sulcal sign. Similarly, in a post-traumatic leak presenting late we found that the site of leak was different from the site of obvious defect or site of sinus opacification. In our only case of iatrogenic CSF leak post otomastoidectomy we additionally found a small defect in lateral wall of sphenoid sinus with adjacent sphenoid sinus opacification. However, on cisternography we found the leak to be into the mastoid air cells and subsequently into the naspharynx via Eustachian tube.

In non-traumatic category we found congenital defects in few patients. Sternberg's *Canal* which represents persistent lateral cranio-pharyngeal canal connecting intracranial space and sphenoid sinus due to congenital fusion defect between basi-sphenoid and greater wing of sphenoid was seen in one patient [18]. Additionally, defects in posterior wall were seen in three patients which requires surgical intervention as the sizes of defect were significant. In one patient placement of patient in knee elbow position probably induced spontaneous pneumocephalous, which prevented opacification of intracranial subarachnoid space. However, we could infer the site of leak as the subarachnoid air was seen in communication with sinus air through defect in posterior wall of sphenoid sinus. Other patients in non-traumatic group had leak from cribriform plate (seen as a column of contrast extending from subarachnoid space to extracranial space) and were treated conservatively. Multiple sites of leak from cribriform plate and fovea ethmoidalis were noted in one patient. In two patients no defects or leak could be appreciated probably due to inactivity of the leak during procedure.

Thus, in patients in both traumatic and non-traumatic categories wherein multiple defects, multiple sites of leak are anticipated CT Cisternogram provides concrete evidence regarding site of leak.

The major limitation of our study was lack of MRI for all patients due to which comparative analysis was not possible. Most of the post trauma patients rarely undergo CT Cisterography in acute settings and hence were not included in the study.

CONCLUSION

In conclusion, despite many limitations of CT Cisternography, it still holds importance as a problem-solving tool for detection of exact site of leak in patients where multiple sites of leak or multiple defects are suspected. Additionally, we have demonstrated spectrum of possible imaging findings of CSF leak on CT Cisternography.

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