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ORIGINAL ARTICLE

Spontaneous Cerebrospinal Fluid Leak of Posterior Surface of Temporal Bone: Review and Case Report

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ABSTRACT

Aim. To analyze the rarity of the clinical case report on spontaneous liquorrhea (SL) posterior cranial fossa (PCF) together with a review of the literature. To determine the features of PCF SL in contrast to anterior and middle cranial fossa (MCF) SL. To identify the optimal diagnostic and treatment tactics in patients with PCF SL.

Materials and methods: We present clinical observations of a PCF SL patient and a detailed review of reports on SL with a defect on the posterior border of the petrous temporal bone (PTB).

Results: Based on the literature review, 22 cases with a sufficient description were analyzed. PCF SL occurs more commonly in women than in men; middle-aged and elderly patients prevail. The average body mass index (BMI) was 33.3 ± 7, corresponding to a degree I obesity. The number of postoperative complications was quite large; each included recurrent liquorrhea.

Conclusion: The data from our experience and a literature review for PCF SL patients are comparable with those of similar studies of SL in the anterior and MCF. Middle-aged and elderly women with excessive BMI are the most vulnerable. In most patients, the defect is located in the lateral regions of the posterior border of the PTB, namely anteriorly, medially from the sigmoid sinus, and below the upper petrosal sinus. A fairly high percentage of postoperative complications is due to insufficient preoperative diagnosis or intraoperative examination of the posterior PTB in patients with combined MCF and PCF defects, causing revision surgery.

Clinical significance. If rhinorrhea, otorrhoea, and/or signs of intracranial space integrity loss (pneumocerephaly or latent liquorrhea with recurrent meningitis) are revealed, careful examination of bone structures of the anterior, middle, and also, be sure PCF is required, especially at the intersection with paranasal sinuses (frontal, ethmoidal, sphenoid) and mastoid air cells and PTB.

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INTRODUCTION

In adults, the most common cause of temporal bone liquorrhea is intraoperative iatrogenesis in the petrous temporal bone (PTB) or damage to the latter in case of traumatic brain injury. Only 4% of all patients include spontaneous liquorrhea (SL) arising from this anatomical region.[1,2]

In the vast majority of patients, when SL is observed in temporal bone, the defect is located on the anterior third of the PTB, that is, in the MCF, namely, in the tegmen tympani. This can be explained by the presence of a thinner bone layer in this region and the direct impact of the temporal lobe weight on the tegmen tympani.[3,4] Much less often, cases of SL are reported when the bone defect is located in the posterior cranial fossa (PCF), on the back surface of the PTB.[3,5,6]

We report a detailed literature review of all the cases of SL caused by PCF bone defect and a clinical case, and we summarize the literature review regarding the SL pathogenesis, exact defect locations, surgical methods, complications, and recurrences.

MATERIALS AND METHODS

We present the clinical observation of a PCF SL patient who underwent surgical treatment at Cerebral Neurosurgery Mechnikov Dnipropetrovsk Regional Clinical Hospital, and detailed literature review observations of SL with a defect located on the posterior PTB. The PubMed database was used to conduct the literature review and covered articles published between 1970 and 2018 inclusive, based on the following keywords: spontaneous cerebrospinal fluid leaks, posterior cranial fossa,

temporal bone, encephalocele, arachnoid granulations, and petrous bone, and abbreviations CSF, PCF, and AGs.

Table 1 presents a detailed description of all the PCF SL patients. Table 1 does not include (a) nonspontaneous (iatrogenic, after surgery or radiotherapy, post-traumatic) PCF liquorrhea;[7,8] (b) spontaneous PCF pneumocephalus due to bone defects that did not manifest in the form of SL;[9-11] (c) PCF bone defects or encephalocele (herniation) not accompanied by SL (or if this fact was not mentioned in the report);[12,13] (d) clivus defects manifesting in the form of SL, which, in turn, also can be attributed to PCF defects, but we believe that such pathology requires individual analysis;[14,15] (e) literature reviews without specifying own experience of PCF SL treatment;[16] and (f) reports where a PCF defect was mentioned for the general group of SL patients, but that had no required observation data (age, sex, symptoms, defect location, approach, complications, and so forth)[3,17,18]

CASE REPORT

A 46-year-old woman had transparent fluid leakage from the left nasal passage and had impaired left ear hearing. Against the background of general well-being, she had first noticed the impaired hearing of the left ear approximately six months ago; there was no head injury or surgical intervention. Two months later, she had left-sided nasal liquorrhea. The patient was examined by an ear, nose, and throat (ENT) specialist, who diagnosed serous otitis media; however, cerebrospinal fluid (CSF) was obtained during tympanostomy. The patient had no meningitis symptoms. BMI was 32. Audiometry revealed impaired function on the auditory analyzer (34 dB), left side conductive type. Computed tomography (CT) and magnetic resonance (MR) cisternography detected a defect (along with bone thinning) in the lateral posterior border, petrous part of the left temporal bone. The defect was adjacent to the posterior sections of the mastoid antrum, where contrast medium flowed. Encephalocele was found at the defect site (Figs 1 and 2).

Through a left-sided retrosigmoid approach, a meningoencephalocele (MEC) was found in the posterior border of the temporal bone, anteriorly and medially from the sigmoid sinus and below the upper petrosal sinus; it was firmly adherent to dura mater. After coagulation, the MEC was dissected and separated. In its place, there was a 3 × 4 mm bone defect, which was plugged with temporalis muscle and Tachocomb (Takeda, Linz, Austria). In addition, the surface of the posterior border of the left temporal bone was inspected throughout its length (a caudal group of cranial nerves, acousticofacial nerve complex, and trigeminal nerve were visualized). No other bone defects were revealed (Fig. 3).

There were no postoperative complications. The CSF leakage from the left nasal passage stopped. Auditory analyzer function improved to 18 dB. The follow-up CT and MR cisternography images are shown (Fig. 4).

The follow-up period was for 3 years. The liquorrhea did not recur.

RESULTS

Based on a literature review (including our patient), 22 patients with a sufficient description were analyzed. PCF SL occurred more commonly in women than in men [16 (73%) vs. 6 (27%), respectively; ratio 1:2.7]. There was a prevalence of middle-aged and elderly patients (average age, 56.7±16.9). The defects were on the right and left sides in 14 (64%) and eight (36%) patients, respectively. The average BMI was 33.3±7, corresponding to degree I obesity.

The dominant clinical conditions included meningitis in 12 patients (55%), hearing loss in 10 (45%), and aural fullness in nine (41%). In addition, otorrhea was diagnosed in nine (41%) patients, but in five only (according to the authors) was it caused mechanically during diagnostic tympanostomy. Otorhinorrhea (otic and nasal liquorrhea) with CSF leakage through tube auditiva was reported in seven (32%) patients, while otitis media was diagnosed in six (27%). Nonspecific symptoms included otalgia in two (9%) patients, pulsatile tinnitus in two (9%), headache in one (5%), and spontaneous pneumocephalus in one (5%).

Among the diagnostic methods, CT/high-resolution CT [8 (36%) patients, and CT and MR imaging (MRI; 4 [18%] patients) were preferred. Also, in five (23%) patients, before CT and/or MRI, transferrin testing was performed (in two the test was false-negative). CT and/or MRI was supplemented by CT and/or MRI cisternography in two patients only (9%).

In the majority of patients, the bone defect was located in the lateral regions of the posterior PTB border, namely anteriorly and medially from the sigmoid sinus and below the upper petrosal sinus. In one patient, the defect had a noncharacteristic location, namely anterior to the porus acusticus internus. In four (18%) patients, combined MCF and PCF defects were detected.

In eight (36%) patients, the bone defect was accompanied by MEC. Most of the surgeons selected mastoidectomy and a transmastoid approach [12 (55%) patients], while a transtemporal approach (middle fossa craniotomy) was used in one (5%), three (14%) underwent a combination of the above approaches, one (5%) underwent postauricular transcanal middle ear exploration, and one (5%) underwent a retrosigmoid approach. A wide variety of autogenous tissues and materials were used

Table 1: Summary of spontaneous posterior cranial fossa (posterior surface of temporal bone) CSF leaks described in the literature to date including our patients

No.	*Author(s)*	*Year*	*Cases*	*Age (yr)/ sex*	*Side*	*BMI*	*Presentation*	*Diagnosis methods*	*Bone defect location*	*Encepha-locele*	*Approach*	*Repair*	*Complica-tions and recurrences*
1	Briant and Bird	1982	1	76/M	Right	–	Otalgia, hearing loss, meningitis	–	Anterior to sigmoid sinus	No	Mastoidectomy	Temporalis muscle	–
2	Schuknecht et al.[24]	1982	1	54/F	Left	–	Otitis, otorrhea, meningitis	–	Between superior petrosal sinus and lateral sinus	No	Mastoidectomy	Adipose tissue	–
3	Wetmore et al.	1987	1	53/F	Right	–	Hearing loss, tinnitus, rhinorrhea	–	Between sigmoid sinus and posterior semicircular canal	No	–	–	–
4	Gacek	1992	2	64/M	Right	–	Aural fullness, hearing loss, otorrhea	CT, MRI	Anterior to sigmoid sinus	No	Mastoidectomy	Adipose tissue	–
				53/F	Right	–	Serous otitis media	–	Anteromedial to sigmoid sinus	No	–	–	–
5	Langman et al.[23]	1993	1	71/F	Left	–	Otitis, meningitis	CT	Anterior to sigmoid sinus	No	Mastoidectomy	Adipose tissue	No
6	Welge-Luessen and Probst[21]	2004	2	64/M	Right	–	Otalgia, spontaneous pneumoce-phalus, meningitis	CT	Lateral part of posterior surface of temporal bone (Trautmann's area)	No	Mastoidectomy	Temporal fascia, muscular flap, bone patee, fibrin glue	No
				57/F	Left	–	Meningitis	CT	Trautmann's area	No	Mastoidectomy	Temporal fascia, muscular flap, fibrin glue	No
7	Rao et al.[5]	2005	1	75/F	Right	–	Serous otitis media, meningitis	High-resolution CT	Medial to sigmoid sinus	No	Transmastoid	Temporal fascia, fibrin, fat	No
8	Lee et al.[29]	2008	2	41/M	Left	–	Otorrhea, meningitis	CT	Lateral third of posterior wall of temporal bone	No	–	–	–
				88/F	Right	–	–	CT	–	No	–	–	–
9	Nadaraja et al.[20]	2012	3	63/F	Right	29	Rhinorrhea, aural fullness, otitis media, meningitis	CT, MRI	Superior to endolymphatic sac	Yes	Middle fossa craniotomy and mastoidectomy	Temporalis fascia and muscle	No
				37/F	Right	28	Meningitis	CT, MRI	Between sigmoid sinus and posterior semicircular canal	Yes	Mastoidectomy	Temporalis fascia and muscle	No
				68/M	Right	30	Rhinorrhea, aural fullness, otorrhea (after tympano-stomy), hearing loss	CT, MRI, β-2-transferrin positive testing	Tegmen defect and between sigmoid sinus and posterior semicircular canal	No	Middle fossa craniotomy and transmastoid	Hydrox-yapatite cement	Recurrent CSF leak Reoperation

Contd...

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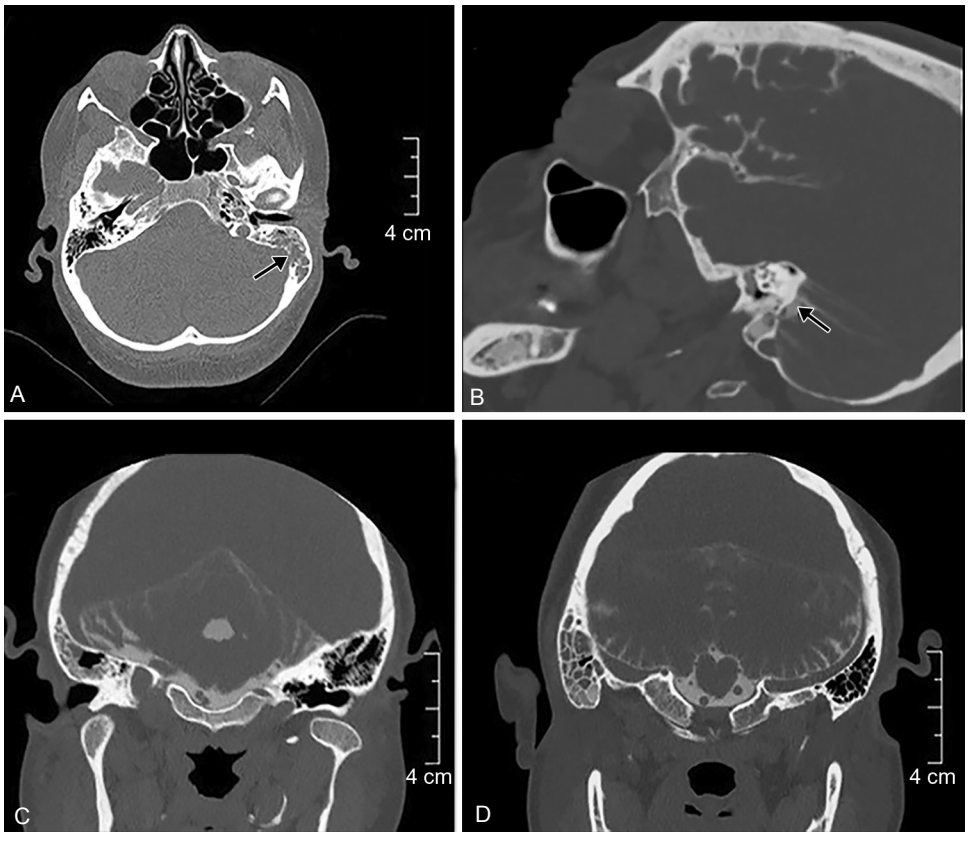
No.	Author(s)	Year	Cases	Age (yr)/ sex	Side	BMI	Presentation	Diagnosis methods	Bone defect location	Encepha-locele	Approach	Repair	Complica-tions and recurrences
10	Kim et al.[6]	2014	1	36/F	Right	42.8	Otorrhea, hearing loss	CT	Combination middle fossa and posterior fossa defects	Yes	Transmastoid	Autologous mastoid bone, temporalis fascia, tissue sealant	No
11	Rereddy and Mattox[12]	2016	1	31/F	Right	44.2	Otorrhea	CT	Medial to sigmoid sinus	Yes	Transtemporal approach	Periosteal graft and DuraSeal	No
12	Wick et al.[19]	2016	5	56/F	Left	32.8	Aural fullness, otorrhea (after tympanostomy), rhinorrhea, hearing loss, meningitis	CT, MRI, β-2-transferrin negative testing	Tegmen defect and anterior to the porus acousticus internus.	Yes	Middle fossa craniotomy	Abdominal fat, fibrin glue	Recurrent CSF leak Reoperation
				79/F	Left	23.8	Aural fullness, rhinorrhea, hearing loss, meningitis	CT, MRI cisternography, β-2-transferrin negative testing	Anterior to sigmoid sinus	No	Transmastoid	Temporalis fascia, fibrin glue	No
				61/M	Right	24.4	Aural fullness, otorrhea (after tympanostomy), hearing loss	CT, MRI, β-2-transferrin positive testing	Hypotympanum	Yes	Postauricular transcanal microscopic middle ear exploration	Bone wax, bone pate, temporalis fascia	No
				19/F	Right	40.4	Aural fullness, otorrhea (after tympanostomy), hearing loss, meningitis	CT, β-2-transferrin positive testing	Medial to sigmoid sinus and posterior to common crus of semicircular canals	No	Transmastoid	Bone wax, temporalis muscle, bone cement	Recurrent and purulence CSF leak Reoperation
				55/F	Left	41.4	Aural fullness, otorrhea (after tympanostomy), rhinorrhea, hearing loss, headaches, pulsatile tinnitus	CT, MRI, β-2-transferrin positive testing	Tegmen mastoideum, tegmen tympani, medial to the sigmoid sinus	Yes	Middle fossa craniotomy and mastoidectomy	Bone cement, temporalis fascia and muscle	Recurrent CSF leak Reoperation
13	Sirko et al. (present study)	2018	1	46/F	Left	32	Aural fullness, serous otitis media, rhinorrhea, hearing loss	High-resolution CT, MRI with and without gadolinium, CT and MRI cisternography	Anterior, medial to sigmoid sinus and inferior to the superior petrosal sinus	Yes	Retrosigmoid	Temporalis muscle and Tachocomb	No

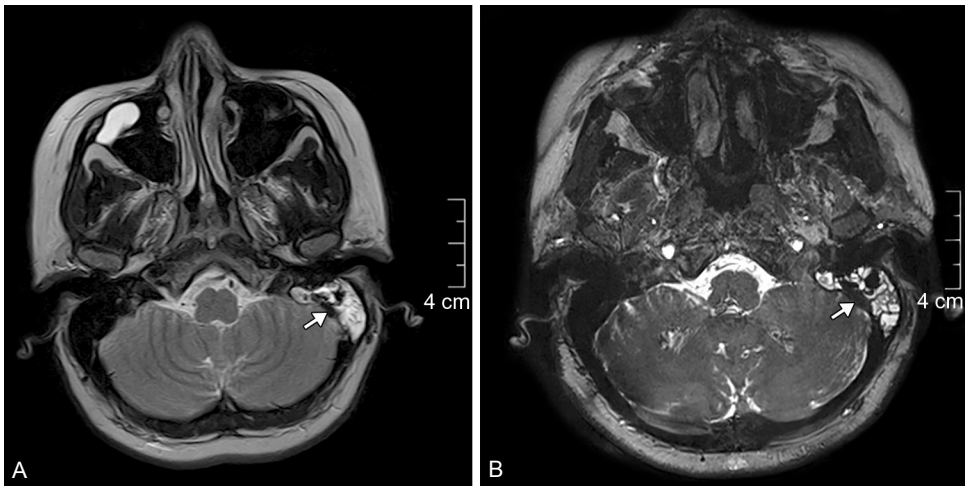
–: no data in source report

to plug the defect; however, in almost all the patients, temporalis fascia and/or muscle combined with various adhesive compositions were applied.

The number of liquorrhea recurrences [4 (18%) patients] was rather large. One patient had a recurrent CSF leak with purulent otitis media, mastoiditis, and purulent otorrhea (gram-negative anaerobic flora common to the oral cavity—Eikenella corrodens and Prevotella) two weeks postoperatively. Sanative reoperation and antibacterial therapy were used to eliminate the complication. [19] In the remaining three patients, the complications were because of insufficient preoperative diagnosis and intraoperative examination of the posterior PTB border. The common feature in these patients was combined MCF and PCF defects; the MCF defect was closed but the PCF defect was omitted for some reason, which caused recurrent



Figs 1A to D: (A) Preoperative axial CT. In the lateral part of the posterior border of the petrous left temporal bone, bone defect (arrow) and liquid (CSF) filling the mastoid antrum are seen; (B) Preoperative sagittal CT cisternography. MEC is clearly visualized (arrow); (C, D) Preoperative coronary CT cisternography. Mastoid antrum cells are massively filled with contrast medium

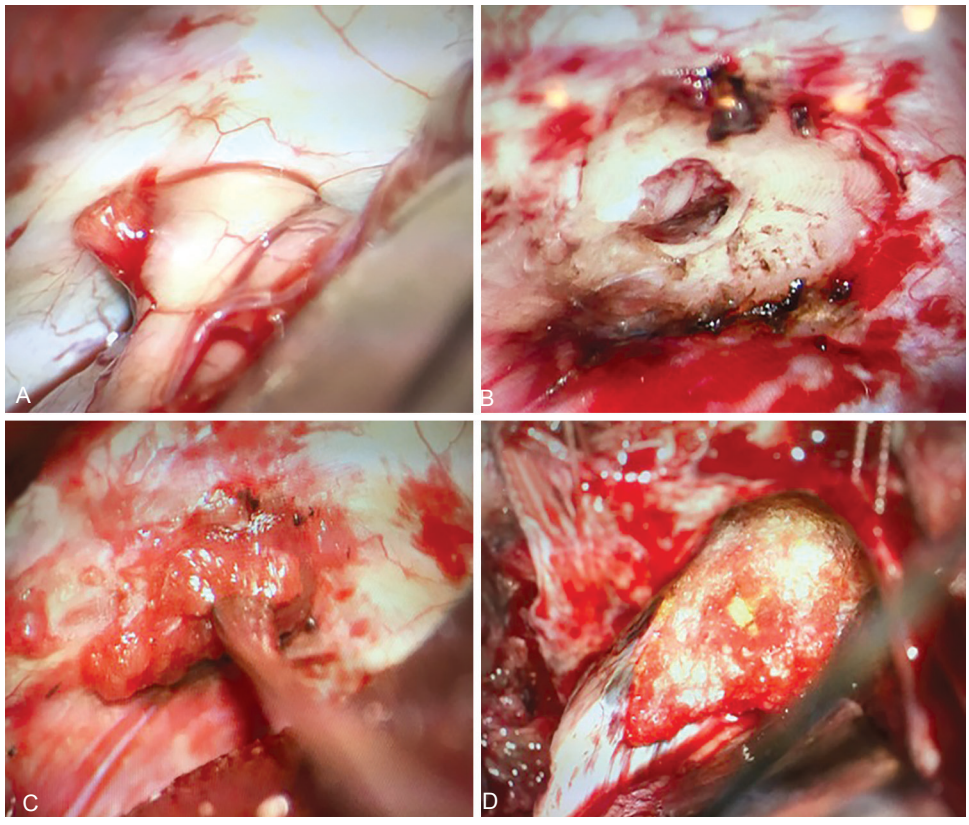


Figs 2A and B: (A) Preoperative T2 mode MRI. Hyperintense content (CSF) in the mastoid antrum; (B) Preoperative MRI cisternography. MEC is clearly visualized (arrow)

liquorrhea. All the three patients underwent reoperation and the recurrent liquorrhea ceased successfully. Thus, liquorrhea recurred in three (75%) of four patients with combined MCF and PCF defects.

In the first case of a 68-year-old patient with spontaneous rhinorrhea and aural fullness, the CT demonstrated a number of tegmen tympani defects. The craniotomy tegmen defect was eliminated through the middle fossa; however, postoperatively the patient suffered partial complex seizures and hydrocephalus. Despite the installation of a ventriculoperitoneal stent, the rhinorrhea and aural fullness persisted. After 3 months, reoperation through a transmastoid approach confirmed the defect between the sigmoid sinus and posterior semicircular canal, which was removed.[20]

In the second case, an MRI in a 56-year-old woman with rhinorrhea and left-sided otorrhea following tympanostomy demonstrated a CSF deposit in the left



Figs 3A to D: Intraoperative photo. (A) MEC of the posterior border, petrous temporal bone adjacent to cerebellar hemisphere surface. (B) Bone defect after encephalocele removal. (C) Defect thoroughly plugged by temporal muscle. (D) Defect additionally closed by Tachocomb

petrous apex while the left mastoid process also was filled with CSF; via middle fossa craniotomy, the tegmen defect was detected and was removed. Postoperatively, the patient had a headache and left-sided facial numbness. Her otorrhea and rhinorrhea resolved. Five years later, the woman was rehospitalized with a left ear infection and the third episode of meningitis. Repeated MRI showed a CSF deposit in the left petrous apex with a respective bone defect detected by CT. Middle fossa craniotomy was repeated and the anterior petrous apex was opened with a high-speed otologic drill. The defect was anterior to the porus acousticus internus. There were no further episodes of liquorrhea following its removal.[19]

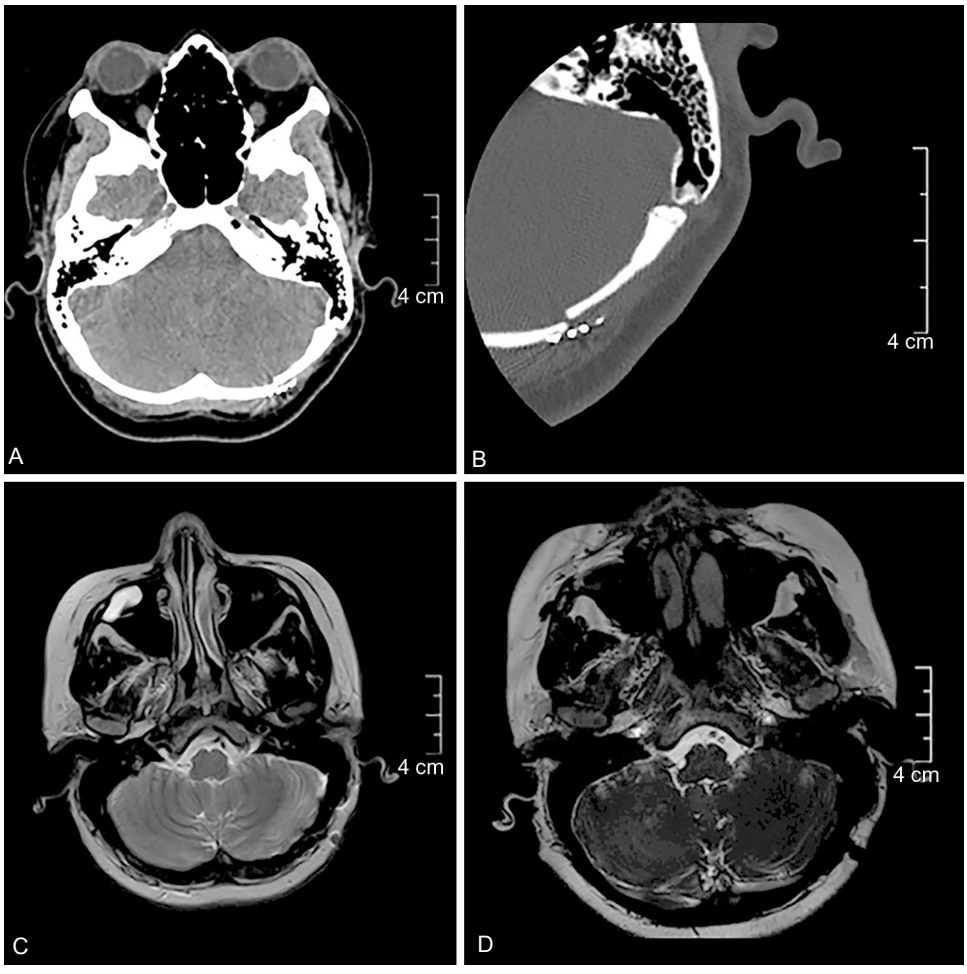
In the third case, a 55-year-old woman had spontaneous rhinorrhea, aural fullness, and left-sided otorrhea following tympanostomy. CT demonstrated several tegmen mastoideum and tegmen tympani bone defects, which were removed following combined middle fossa craniotomy and-mastoidectomy. However, wide otorrhea persisted. At reoperation using a transmastoid approach, the examination of the posterior PTB border revealed another bone defect with encephalocele medial to the sigmoid sinus. The defect was removed, and no further liquorrhea was reported.[20] There were no lethal complications.

DISCUSSION

Primarily, spontaneous otic liquorrhea and otic and nasal liquorrhea occurs in children due to various congenital pathologies where CSF penetrates into the middle ear through bone defects (Mondini anomaly, patent cochlear aqueduct, patent petromastoid canal, and so forth).[21,22]

However, sometimes spontaneous otorrhea occurs in adults also. Currently, the most common SL development theory indicates that arachnoid granulations (AGs), which are protrusions of arachnoid tissue through dura mater, are the causes.[21-23] They are located mostly near venous sinuses and are involved in CSF resorption; however, part of them may be inside the dura mater, which lies on the skull base. Such a location is nonpersistent and variable. Thus, the incidence of AGs in the posterior PTB border is 2.4–9.5%.[22,24] In the MCF (anterior PTB border), the incidence is higher at 12.7–22%.[25,26] Remenschneider et al.[27] reported that, upon examining 52 temporal bones, 30 (58%) cases of AGs were found (though SL was not always detected), of which in 16 (53%) had AGs on the posterior PTB border.

CSF pressure is much higher than venous pressure; therefore, CSF flows to the intracranial venous system through the AGs cells. The AGs located on the skull base can gradually destroy bone tissue in the long term.[22,28]



Figs 4A to D: (A) Postoperative CT, axial projection. Normal pneumatization of mastoid antrum on both sides. Bone flap after retrosigmoid craniotomy, fixed with a titanium implant; (B) Postoperative CT, spot imaging; petrous temporal bone; (C) Postoperative T2 mode MRI. The hypointense signal from mastoid antra on both sides (air); (D) MRI cisternography. There is no CSF in the mastoid antrum

Bone erosion is not clinically significant unless it is located near pneumatized parts of the skull. The risk of CSF leakage is lower when the AGs are in the anterior cranial fossa (sphenoid sinus, sella turcica, cribriform bone), MCF (tegmen tympani), or PCF (mastoid labyrinth cells), where the bone is much thinner.

It is evidenced that, with age, the AGs increase in volume. In the study of Gacek,[22] the average age was 74.6 (52–92) versus 55 (27–88) reported by Lee[29] and 56.7 (19–88) in our SL review. There is a prevalence of female patients. Gacek[22] reported a women-to-men ratio of 11:5 and Lee[29] reported a 17:13 ratio, while in our review the ratio was 16:6.

There is a correlation between elevated BMI and SL, indicating possible additional etiologic factors, namely, metabolic and bone mineralization disorders in overweight people.[12,14,20,30]

Several studies indicate a correlation between intracranial hypertension (ICH) and SL.[14,30,31] However, these studies only include cases when a defect is located in the anterior and MCF. The correlation with the PCF location was not assessed. On the contrary, in our clinical study, there was CSF hypotension. Following the lumbar puncture, the CSF pressure was 80 mm Hg, most likely because of continuous CSF leak from the subarachnoid space. Gadolinium contrast MRI demonstrated intensive contrast accumulation in the dura mater, a differential MR sign of CSF hypotension (Fig. 5).

At the same time, it should be noted that we did not know the true ICP until liquorrhea developed. We believed that the correlation between ICH and SL, at least in the presence of PCF defects, requires further study.

Regarding the SL diagnosis, it should be mentioned that each diagnosis method has pros and cons and certain application restrictions. According to our knowledge and the literature review, the optimal diagnosis option for spontaneous basal PCF liquorrhea is combined high-resolution CT and MRI supplemented with CT and/or MRI cisternography. In one (5%) patient, when CT was used for preoperative diagnosis, liquorrhea recurred postoperatively. Using a combination of CT and MRI, three (13%) patients had recurrent liquorrhea recurrence.

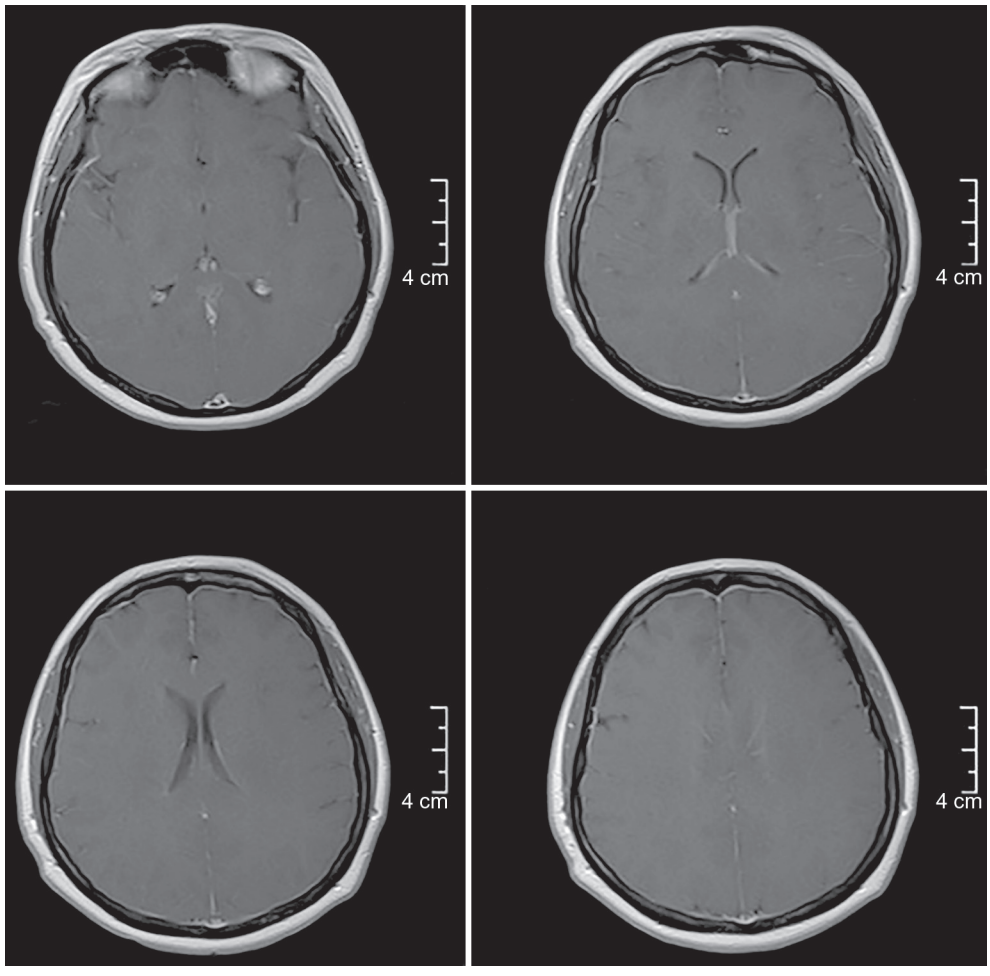


Fig. 5: Preoperative gadolinium contrast MRI. Contrast accumulated in dura mater (a sign of CSF hypotension) can be seen.

In none of the four patients with postoperative PCF SL recurrence, was CT or MRI cisternography used for the preoperative diagnosis. The sensitivity of CT cisternography for basal liquorrhea diagnosis was 48% to 96% according to the literature.[32,33] The sensitivity of MRI cisternography was 87–100%.[16]

CONCLUSION

In general, the data from own experience and a review of the literature on PCF SL patients were comparable with those of similar studies of SL of the anterior and MCF. Middle-aged and elderly women with excessive BMI are the most vulnerable.

SL with a bone defect located in the PCF (that is, the posterior PTB surface) is quite rare. In the vast majority of patients, the defect is located in the lateral posterior PTB border, namely anteriorly and medially from the sigmoid sinus and below the upper petrosal sinus.

A fairly high percentage of liquorrhea recurrence is because of insufficient preoperative diagnosis or intraoperative examination of the posterior PTB border in patients with combined MCF and PCF defects, which causes revision surgery.

CLINICAL SIGNIFICANCE

If the clinical signs of liquorrhea are found based on medical history, patient complaints, or rhinoscopy, we considered the following algorithm to be the most optimal for SL diagnosis. It is necessary to conduct the test for the presence of ß2-transferrin and beta-trace protein in the nasal cavity.[16] In case of positive results (keeping in mind potential false-negative response), high-resolution CT should be performed. If CT detects the exact location of the CSF fistula, the surgical closure should be performed. If the fistula location cannot be determined on CT, MRI cisternography should be performed, followed by surgery if positive results are obtained. If MRI cisternography is insufficient, other invasive diagnosis methods (CT cisternography, fluorescein test, and so forth) should be applied.

It is reasonable to suspect a paradoxical (otic and nasal) liquorrhea if rhinorrhea develops together with otologic symptoms (otitis medium, aural fullness, otorrhea, hearing loss, pulsatile tinnitus, otalgia). At the same time, in cases of isolated rhinorrhea (no ear damage symptoms), neurosurgeons and ENT specialists usually look for defects in the anterior cranial fossa base. In addition, the presence

of symptoms such as meningitis and spontaneous pneumocephalus, constitutes grounds to look for anterior and MCF defects. Thus, if rhinorrhea, otorrhoea, and/or signs of intracranial space integrity loss (pneumocerephaly or latent liquorrhea with recurrent meningitis) are revealed, careful examination of bone structures of the anterior, middle, and also, be sure PCF is required, especially at the intersection with paranasal sinuses (frontal, ethmoidal, sphenoid) and mastoid air cells and PTB.

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