The Laryngoscope

Management of Meningoencephalic Herniation of the Temporal Bone: Personal Experience and Literature Review

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Objectives/Hypothesis: Temporal bone meningoencephalic herniation is a rare condition with potentially dangerous complications. The aim of this study is to analyze the pathogenesis, clinical presentation, surgical treatment, and postoperative outcome of 133 cases of surgically confirmed temporal bone meningoencephalic herniations. A review of the literature is also presented.

Study Design: Retrospective case series (quaternary referral otology and skull base center).

Methods: This study is based on the analysis of the collected data of 133 cases of temporal bone meningoencephalic herniations surgically treated from 1984 to 2006. The follow-up ranged from 12 to 204 months with a mean of 38.4 months.

Results: Meningoencephalic herniations were divided into four etiologic groups: spontaneous (24.8%), secondary to chronic otitis media (21.8%), iatrogenic (45.9%), and posttraumatic (7.5%). Different surgical techniques were used for treatment: transmastoid approach (27.8%), middle cranial fossa approach (27.8%), combined technique (transmastoid plus minicraniotomy, 3%), and middle ear obliteration with blind sac closure of the external auditory canal (41.4%).

Conclusions: Temporal bone meningoencephalic herniations are potentially life threatening, and surgery must take place expeditiously. The choice of the most appropriate surgical approach must be based on the localization and size of the herniated tissue, preoperative auditory function, the presence of active infection, intraoperative cerebrospinal fluid leak, and concomitant pathology.

Key Words: Meningoencephalic herniation, encephalocele, tegmen defect, temporal bone, cerebrospinal fluid leakage, meningitis, middle ear surgery, chronic otitis media, cholesteatoma.

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INTRODUCTION

Herniation of meningeal and brain tissue into the temporal bone through a bony defect is a rare and potentially life-threatening condition requiring surgery. The existence of a pathway from the middle ear to the subarachnoid space can lead to potential infectious sequelae including meningitis, encephalitis, and otogenic cerebral abscesses. Furthermore, the dysfunctional herniated cerebral tissue may trigger epilepsy. The introduction of antibiotics, together with the implementation of microsurgical procedures and imaging techniques, has greatly decreased the incidence of potentially dangerous complications.[1–3]

In the recent literature, some authors[4–7] have employed the term "encephalocele," although we consider the term "meningoencephalic herniation" (MEH) more appropriate because it describes the presence of meningeal and/or encephalic tissue prolapsed into temporal bone but excludes the term "cele" because a cystic area containing cerebrospinal fluid (CSF) is not a constant finding.[8]

While the existence of a tegmen defect is a consistent prerequisite for the development of MEH, the etiology of this defect varies and includes chronic otitis media (COM), with or without cholesteatoma; head trauma with temporal bone fracture; previous otologic surgery; and spontaneous brain herniation. With a defective middle fossa floor, increased intracranial pressure can lead to the herniation of meningeal and/or cerebral tissue into the middle ear or mastoid cavity. A histopathologic examination of the herniated tissue usually reveals normal glial tissue, disorganized neural tissue, or gliosis.[9]

This study analyzes the pathogenesis, clinical presentation, surgical treatment, and possible complications of temporal bone MEH. We describe a series of patients surgically treated at the Gruppo Otologico from 1984 to 2006 and review the literature.

MATERIALS AND METHODS

A retrospective chart review identified 122 patients (63 males and 59 females) presenting with temporal bone MEH. Of

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these, 11 had bilateral MEH, giving a total of 133 surgically treated ears. All cases were diagnosed and treated at the Gruppo Otologico from 1984 to 2006. In this study, we included patients with an intraoperative confirmation of MEH, excluding those with a tegmen defect associated with simple CSF leakage. We collected pre-, intra-, and postoperative data from all patients. A minimum follow-up of 12 months was required for inclusion in the study. All patients were reviewed every 6 months for the first year after surgery, then once a year, and studied with a magnetic resonance imaging (MRI) and/or computed tomography (CT) scan at 1, 3, and 5 years after surgery.

The review process involved a literature search using the MEDLINE database to identify works in English addressing treatment of temporal bone MEH. The following key words were used: encephalocele, temporal bone, brain herniation, middle ear, meningoencephalic herniation. Among articles complying with the aims of the study, those reporting the largest number of patients were reviewed. The collected data from each article included in the review were analyzed for number of patients treated, etiology, surgical approaches, complications, recurrences, and length of follow-up.

The following are the basic steps of the four surgical approaches adopted in this series.

Transmastoid Approach (TM)

Making a classical retroauricular incision and a mastoidectomy, the herniated brain tissue is exposed, coagulated, and sectioned at the hernia waist. Simultaneously, middle ear pathology is treated, if present. Then the repair of the bony defect is effected "from below" by inserting a piece of cartilage through the defect. The reconstruction may be reinforced with bone dust and then covered with fascia.

Middle Cranial Fossa Approach (MCF)

A skin incision is made from the pretragal area, extending superiorly for 7 cm to 8 cm, curving first posteriorly and then anteriorly. An incision in the temporalis muscle exposes the bone of the temporalis squama. A 4 cm $\times$ 4 cm craniotomy is then performed, with the lower edge of the craniotomy at the level of the zygomatic process. The dura of the temporal lobe is carefully elevated until the neck of the hernia is identified, coagulated, and cut. The herniated tissue is left in the middle ear or mastoid. A piece of cartilage is placed between the bony defect and the dura. The defect is further reinforced by placing a piece of temporalis fascia between the dura and the cartilage.[10]

Combined Approach

This approach is the combination of the transmastoid approach with a minicraniotomy at the level of the temporal fossa. Through the cortical mastoidectomy, the brain tissue is treated as already described in the transmastoid approach. Next, a sufficiently large piece of cartilage is inserted extradurally through a small craniotomy.[11] The bony defect is further repaired by bone paté and covered by temporalis fascia from the mastoid cavity.

Middle Ear Obliteration (MEO)

After blind sac closure of the external auditory canal, a subtotal petrosectomy is performed, with complete removal of mucosa, skin, tympanic membrane, malleus, incus, and disease. The herniated tissue is reduced using bipolar coagulation and cut with a microscissor. Once the disease is eradicated, the middle ear opening of the eustachian tube is packed with soft tissue and bone wax. The cavity is then obliterated with abdominal fat.[12]

TABLE I.
Etiology.

	Number	%
Spontaneous	33	24.8
COM $\pm$ cholesteatoma	29	21.8
Iatrogenic	61	45.9
Posttraumatic	10	7.5

COM = chronic otitis media.

RESULTS

The patients ranged from 9 to 78 years of age, with a mean of 48.4 years. They were divided into four groups according to the etiology of the herniation (Table I): The first group included cases of spontaneous presentation; the second group included cases in which herniation was associated with COM, with (24/29, 82.8%) or without (5/29, 17.2%) cholesteatoma; the third group was composed of patients who had undergone a previous mastoidectomy (iatrogenic herniations); and the last group consisted of patients with a positive history of head trauma (posttraumatic herniations). In iatrogenic MEH, a cholesteatoma was found in 32 cases (52.5%). We included these cases in the iatrogenic group because it was not possible to demonstrate if MEH was due to previous surgery or caused by cholesteatoma.

Presenting symptoms are summarized in Table II. The most frequent was conductive or mixed hearing loss, present in 87.2% (116/133) of the total number of cases. In the majority of the cases, presenting symptoms were related to the concomitant disease (for example, otorrhoea in COM), while more specific symptoms of MEH (CSF leakage, meningitis, and epilepsy) occurred mainly in the spontaneous group. CSF leakage was present at the time of diagnosis in 10.5% of patients (14/133), particularly in those with posttraumatic (3/10, 30%) or spontaneous (5/33, 15.1%) herniations. Meningitis was the first manifestation of MEH in 14 cases (10.5%), especially in those patients with a spontaneous herniation (9/33, 27.3%). Three of these patients also had an episode of cerebral abscess in childhood. An additional two patients had a cerebral abscess before diagnosis without any sign of meningitis. In three patients with spontaneous herniation and in one case of iatrogenic herniation, there was a history of seizure disorders (epilepsy) at the time of diagnosis. Other symptoms present at the time of diagnosis were pulsatile tinnitus, facial nerve palsy (in one case of iatrogenic MEH), trigeminal neuralgia, and headache.

Previous surgery in patients with iatrogenic MEH (61 cases) was a Canal-Wall Down (CWD) tympanoplasty in 28 cases, while 17 cases had a Canal-Wall Up (CWU) tympanoplasty; the remaining 16 cases had a radical cavity. Twenty-three of these patients had undergone

TABLE II.
Presenting Symptoms According to Etiology.

Etiology	CHL	COM Related	Vertigo	CSF Leak	Meningitis	Epilepsy	Others
Spontaneous	25		8	5	9	3	4
COM ± cholesteatoma	27	21	9		1		6
Iatrogenic	55	31	13	6	3	1	5
Traumatic	9			3	1		1
Total	116	52	30	14	14	4	16

CHL = conductive hearing loss; COM = chronic otitis media; CSF = cerebrospinal fluid.

more than one surgery in the same ear, seven of them more than three.

In 11 patients (8.3%), we found bilateral MEH. Six of these had spontaneous herniation, three had bilateral COM with cholesteatoma, and two had previous bilateral surgery.

The different surgical approaches used for MEH repair, according to the etiology of the herniation, are summarized in Table III. We performed a total of 37 TM approaches, 4 combined approaches, 37 MCF approaches, and 55 MEO.

Intraoperative Findings

The right ear was involved in 69 cases (51.9%) and the left ear in 64 (48.1%). The bony defect was located in the tegmen mastoideum in 93 cases (69.9%), tegmen antri in 27 cases (20.3%), and tegmen tympani in 18 cases (13.5%). Multiple defects were found in 19 cases (14.3%), notably in spontaneous or iatrogenic cases. Bony defect involving the external auditory canal was found in 2 cases (1.5%). An MEH of the posterior cranial fossa was found in 12 cases (9%). The materials used for defect repair were temporalis fascia, muscle, cartilage, and bone. Active CSF leakage was an intraoperative finding in 54 cases (40.6%). A cholesteatoma was present in 56 cases for which we simultaneously treated the MEH and cholesteatoma, performing 35 middle ear obliterations, 19 TM approaches, and 1 combined approach. In 1 case, with a large mastoid and tegmen antri herniation in the presence of an infected cholesteatoma, we elected to treat the MEH first by performing an MCF approach, delaying the cholesteatoma removal for a second procedure (4 months later) by a TM approach.

Postoperative Follow-up

The mean follow-up time was 38.4 months (range, 12–204 months). Three recurrences were observed during the follow-up period as listed in Table IV. All of them were revised surgically. Two main complications occurred during the postoperative period. One patient, treated by a combined approach technique for posttraumatic MEH, developed meningitis 4 days after surgery. This was successfully treated medically. Another patient, treated by an MCF approach for a spontaneous MEH, developed an extradural subtemporal hematoma 1 day after surgery that receded with conservative treatment.

Literature Review

A literature review on surgical treatment of MEH of the temporal bone is summarized in Table V. This is to date the largest case series of this pathology reported in the literature.

DISCUSSION

A bony defect is necessary for the development of MEH, allowing meningeal and/or brain tissue to protrude into the temporal bone.[13] This condition can be due to temporal bone trauma,[14] iatrogenic injury,[15] or chronic infection of the middle ear.[16] In addition, spontaneous herniations have been reported, although the etiology is unclear because autopsy findings of temporal bone defects are higher than those of MEH,[14] suggesting that MEH is not an inevitable sequela of tegmen dehiscence. Some theories have been proposed to explain the pathogenesis of congenital herniations, including normal variations of intracranial pressure, decades of CSF pulsation, low-grade inflammation, or aberrant arachnoid granulations.[1,5,14,17] It appears that in the presence of a natural defect in the tegmen, the dura is progressively thinned by CSF pulsation, resulting in brain herniation. This theory is supported by the fact that the mean age of presentation of spontaneous herniation in our series is 57. Probably, in the presence of a small dehiscence, a congenital or acquired dural defect is also present, while in the case of a large tegmen defect, the weight of the temporal lobe together with its pulsation are sufficient to cause brain prolapse into the middle ear or mastoid cavity.

Generally MEH occurs through the middle cranial fossa, at the level of the tegmen mastoideum or tympani and rarely through the posterior fossa. The higher frequency of middle cranial fossa MEH is correlated with

TABLE III.
Surgical Approach According to Etiology.

Etiology	TM	Combined	MCF	MEO
Spontaneous	3	1	21	8
COM ± cholesteatoma	15	1	1	12
Iatrogenic	17	1	9	34
Traumatic	2	1	6	1
Total	37	4	37	55

TM = transmastoid; MCF = middle cranial fossa; MEO = middle ear oblitaration; COM = chronic otitis media.

TABLE IV.
Recurrences.

	First Surgery (Approach)	Revision Surgery	Distance of Recurrence (Months)	Etiology	Position of MEH	Note
1	Combined	MEO	114	Traumatic	M + TA	Recurrence of CSF leak
2	MCF	MEO	112	Iatrogenic	M + TA + TT	Recurrence of MEH + cholest
3	TM	TM	53	Iatrogenic	M	Recurrence of MEH + residual cholest

MEH = meningoencephalic herniation; MCF = middle cranial fossa; MEO = middle ear obliteration; TM = transmastoid; M = mastoid; TA = tegmen antri; TT = tegmen tympani; CSF = cerebrospinal fluid.

the presence of a thinner bone layer at this location[5,18] and with the direct impact of the weight of the temporal lobe over the tegmen.

In cases where MEH is secondary to another disease process (particularly COM), clinical presentation is often masked by symptoms related to the primary pathology.[19] In this situation, the diagnosis of MEH may be an intraoperative finding. More specific presentations (CSF leak, epilepsy, meningitis, brain, or epidural abscess) tend to be found in cases of spontaneous MEH, a finding confirmed by other reported series.[5,7,14,20] A typical presentation is with a discharging ventilation tube, often sited to treat a presumed otitis media with effusion. CSF otorrhea or otorhinorrhea can be present in the case of MEH, but it does not inevitably imply herniation of meningeal or brain tissue.[21]

The typical otoscopic findings in MEH are a pulsatile mass or a middle ear effusion, but occasionally, especially in operated ears, the clinical diagnosis can be challenging due to the nonspecificity of the findings.

Audiometric tests often demonstrate a conductive hearing loss, due to CSF in the middle ear or to herniated brain tissue. Of course, these findings are often indicative of coexisting disease.

Once MEH is suspected, a thorough radiologic assessment should be performed.[22] High-resolution computer tomography scan (HRCT) with axial and coronal sections is useful to define the presence, location, number, and size of the bony defects. While coronal sections give a better view of middle cranial fossa MEH, the rare posterior cranial fossa herniations are well defined with axial sections. In the diagnosis of brain herniation, HRCT is nonspecific, demonstrating a nonenhancing soft tissue in continuity with a tegmen defect (Fig. 1) that is indistinguishable from cholesteatoma, granulation tissue, cholesterol granuloma, or other soft-tissue masses inside the middle ear cavity.[23] MRI is the ideal method to differentiate these conditions: Herniated meningoencephalic tissue is seen as a nonenhancing contiguous mass, isointense to brain in all sequences. Contrarily, a cholesteatoma appears hyperintense in T2-weighted images, and a cholesterol granuloma appears hyperintense both in T1- and in T2-weighted images. Contrast administration enhances only granulation tissue.[24] Usually T2-weighted coronal sections are the most useful to identify middle cranial fossa brain herniations (Fig. 2). Recently,[25] non-echo-planar imaging-based diffusion-weighted MRI has been proposed for the detection of cholesteatoma. The same technique, when in doubt, may be adopted to differentiate a cholesteatoma from herniated tissue.

The choice of surgical approach is directed by the etiology, the position and size of the bony defect, preoperative audiometry, the presence of chronic infection in the middle ear, and/or intraoperative active CSF leakage.

In our experience, the TM approach is generally useful for single, small dehiscences localized at the level of the tegmen mastoideum or antri, if the ossicular chain is not involved. When the chain is removed for reasons related to the MEH and/or to concomitant COM, the approach may also be used for more anteriorly positioned defects. The approach is also used for the treatment of rare cases of MEH of the posterior cranial fossa. In these cases, the herniated tissue is generally small, even when the dura is extensively uncovered and it is sufficient to simply coagulate the herniated tissue and cover the bony defect with bone dust and fascia.

The MCF approach allows total control of the floor of the middle cranial fossa. The advantage of this approach is the opportunity to reach bony defects located anteriorly without any manipulation of the ossicular chain. In fact, this approach was principally used in our series for cases of spontaneous MEH, in which there was no middle ear pathology and the herniated cerebral tissue could be sectioned "from above" and left in the middle ear with limited risks for hearing, affected preoperatively only by the presence of the MEH. In addition, in cases with active infection, it may be safer to cut the herniated tissue from the sterile environment of the intracranium and leave it in place so that the MEH forms a barrier against the spreading of infection. The herniated tissue left in the middle ear and/or mastoid cavity shrinks progressively with time.[8] If concomitant disease requires middle ear surgery, this can be delayed for some months, allowing the tegmen reconstruction to heal. Some authors[1,19] propose an intradural repair of the dural defect. In our opinion, this procedure is often difficult to perform and gives no advantages compared with an extradural repair. Furthermore, it can increase the risk of neurological complications.

The combined approach is rarely used in our center because it offers no advantage over the TM approach in the case of a small hernia, and it is preferably substituted with an MCF approach in the case of a large bony defect. Furthermore, the insertion of the cartilage layer

TABLE V.
Literature Review on Surgical Treatment of MEH of the Temporal Bone.

Source	Patients/ No. of Cases Treated	Etiology	Surgical Approach	Complications	Recurrences	Months of Follow-up (Mean)	Note
Lundy et al. (1996)	17/19	11 Sp 4 COM 2 Iatr 2 PT	16 Comb 2 MCF 1 TM			31	Fascia-bone-fascia graft (extradurally)
Gubbels et al. (2007)	15/16	Sp	MCF	1 patient developed CSF leak + brain abscess, necessitating a frontal craniotomy		13	Hernia amputated in 11 cases, reduced in 5
Jackson et al. (1997)	35/35	27 Iatr 4 Sp 3 COM 1 AOM	4 MCF 15 TM 16 Comb	3 seizures 2 CSF leaks 2 ischemic events/ strokes 1 sepsis 4 SNHL	3(2 Iatr, 1 Sp) repaired with a combined approach	48.7	23 extradural repairs; 15 intradural repairs; 4 obliteration procedures;
Wootten et al. (2005)	12/12	Iatr	7 TM 5 Comb	3 tympanic membrane perforations 2 recurrent cholesteatomas 2 recurrent CSF	2 recurrences of CSF leaks (from other sites in the temporal bone)	48	Brain herniation and/ or CSF leak; intradural repair
Scurry et al. (2007)	8/7	Sp	5 Comb 2 TM		2 (required multiple operations)		Obese patients
Mosnier et al. (2000)	15/15	9 COM 5 Chol 1 Iatr	11 Comb 4 MCF			24	Extradural repair with fascia + bone
Nahas et al. (2008)	15/15	Sp	Comb		1 (doubt)		Mastoidectomy + 3 cm × 4 cm craniotomy; repair of both dural and bone defects; hydroxyapatite paste with bone grafting
Souliere et al. (1998)	6/6	2 PT 2 COM 2 Iatr	5 Comb 1 TM	1 temporary expressive aphasia		3–48	Extradural repair with fascia and bone
Feenstra et al. (1985)	35/35	26 Iatr 4 COM 4 PT 1 Sp	29 TM 6 Comb		1	27.8	Hernia reduced in 31 cases, amputated in 4
Present Series	122/133	61 Iatr 33 Sp 29 COM 10 PT	55 MEO 37 TM 37 MCF 4 Comb	1 meningitis (Comb) 1 extradural hematoma (MCF)	4 (2 Iatr, 1 PT, 1 Sp) Repair: 2 MEO, 2 TM	38.4	

MEH = meningoencephalic herniation; Sp = spontaneous; COM = chronic otitis media; PT = posttraumatic; Iatr = iatrogenic; AOM = acute otitis media; Chol = cholesteatoma; MCF = middle cranial fossa; TM = transmastoid; Comb = combined (transmastoid plus minicraniotomy/MCF); MEO = middle ear obliteration; CSF = cerebrospinal fluid; SNHL = sensorineural hearing loss.

through the minicraniotomy without direct control increases the risk of dural lesions and/or bleeding. Contrary to our opinion, this technique is used by the majority of authors to repair the tegmen defect from above.[1,2,5,6,14,23]

MEO, in our opinion, represents the safest and most definitive treatment for MEH. Performing a blind sac closure of the external auditory canal, obliterating the eustachian tube and filling the surgical cavity with fat, the middle ear and mastoid cavities are completely isolated from the external environment, minimizing the risks of recurrence and other complications such as CSF leakage. However, due to the resulting conductive hearing loss (generally around 60 dB), it should be reserved for cases with poor auditory reserve or extensive middle ear destruction with a limited possibility for

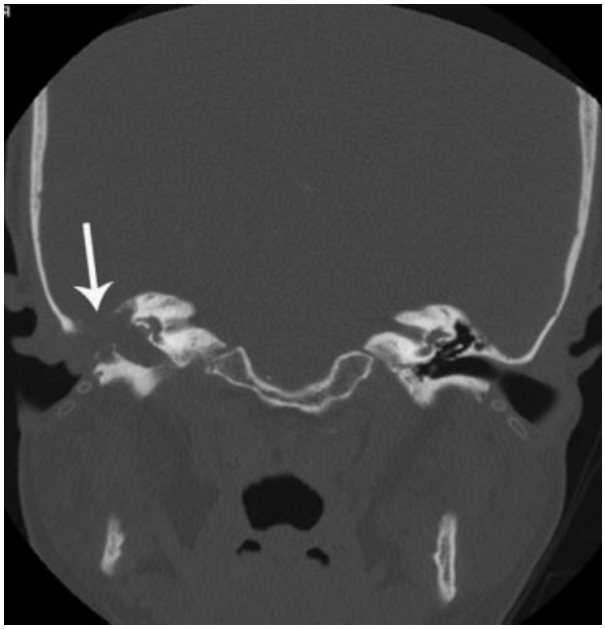

Fig. 1. High-resolution computer tomography (HRCT) scan, coronal view, showing a large bony defect of the floor of the middle cranial fossa (arrow).

reconstruction.[12] Because of the closure of the external auditory canal, in MEO postoperative radiologic follow-up is mandatory (Fig. 3) to reveal presence of residual cholesteatoma. In the literature, MEO is not described by other authors for the treatment of MEH, with Jackson alone reporting four cases of obliteration of the mastoid cavity.

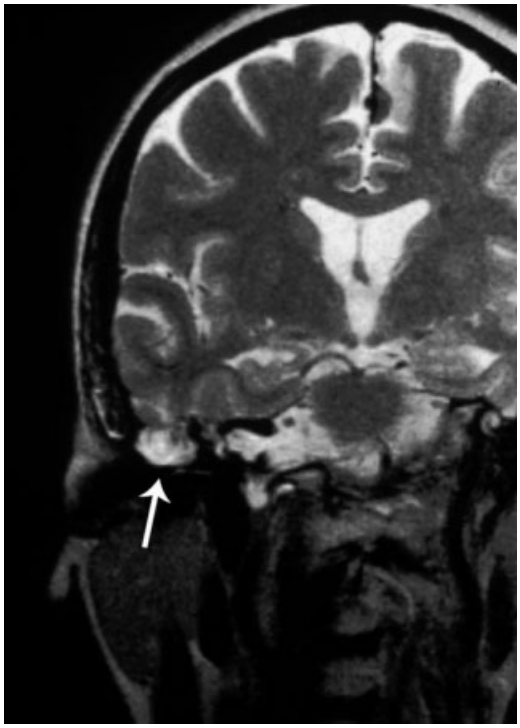

Fig. 2. Magnetic resonance imaging (MRI), coronal view. The mass occupying the middle ear is clearly in continuity with the cerebral tissue (arrow).

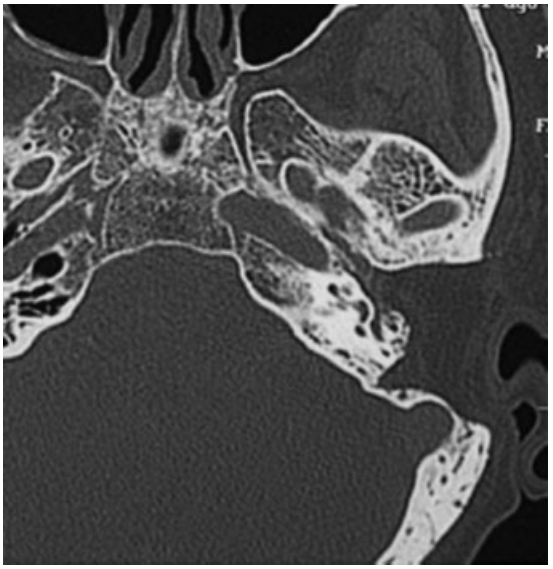

Fig. 3. High-resolution computer tomography (HRCT) axial view. Postoperative image after a middle ear obliteration.

Different materials can be used to repair the bone and dural layer. Simple repair with only a single layer of temporalis fascia is associated with a high rate of recurrence. Better results are described with the use of temporalis fascia in association with muscle, cartilage, or bone in a multilayer repair.[21] Generally, authors advocate the amputation of the herniated cerebral tissue, considered functionless. Others[1,7,14] suggest the reduction of herniated tissue intracranially when it seems to be viable. Gubbels and Nahas describe the use of a hydroxiapatite cement to reconstruct the bony defect.

In our series, we reported three cases of recurrences, corresponding to 2.3% of the 133 patients treated and comparing favorably to other reported rates (range, 8.6%–28.6%).

CONCLUSIONS

The presence of an MEH of the temporal bone is a rare and potentially life-threatening pathology mandating surgical intervention. Different causes can lead to this condition, such as COM, with or without cholesteatoma, head trauma, or previous surgical procedures involving the temporal bone. Moreover, it can occur spontaneously due to a congenital tegmen defect. Different surgical treatments are proposed in this study, with the most appropriate choice being based on the localization and size of the herniated tissue, the preoperative auditory function, and the presence of active infection or other coexisting pathology. When there is the possibility of hearing preservation or its rehabilitation, a TM or MCF approach should be considered. MEO with blind sac closure of the external auditory canal can be the safest and most definitive treatment modality and should

be offered in cases of active CSF leak or extensive herniation if the auditory reserve is poor.

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