Challenges in the Management of Symptomatic Fallopian Canal Meningoceles: A Multicenter Case Series and Literature Review

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Objective: To describe the presentations, the diagnosis, our treatment approaches, and the outcomes for 11 patients with fallopian canal meningocele (FCM).
Study Design: Multicenter: Retrospective case series.
Setting: Tertiary referral centers.
Patients: Patients (N = 11) with radiographically or intraoperatively identified, symptomatic FCM.
Interventions: Surgical repair of cerebrospinal fluid (CSF) leak and meningocele versus observation.
Main Outcome Measures: Presentation (including symptoms, radiographic imaging, and comorbidities), management (including surgical approach, technique for packing, use of lumbar drain), clinical outcomes (control of CSF leak, meningitis, facial nerve function), and revision surgery.
Results: Patients presented with spontaneous CSF leak (n = 7), conductive (N = 11) and sensorineural hearing loss (n = 3), nonpositional intermittent vertigo (n = 3), headaches (n = 4), and recurrent meningitis (n = 1). Risk factors in our series included obesity (n = 4), Chiari 1 malformation (n = 1), and head trauma (n = 2). Noncontrast computed tomography of the temporal bone and magnetic resonance imaging were positive for FCM in 10 patients. Eight patients were managed surgically via a transmastoid approach (n = 4), combined transmastoid and middle fossa (N = 3), or middle fossa alone (n = 1); three were managed conservatively with observation. Postoperative complications included worsened facial nerve palsy (n = 1), recurrent meningitis (n = 1), and persistent CSF leak that necessitated revision (n = 1).
Conclusions: Facial nerve meningoceles are rare with variable presentation, often including CSF otorrhea. Management can be challenging and guided by symptomatology and comorbidities. Risk factors for FCM include obesity and head trauma, and Chiari 1 malformation may present with nonspecific otologic symptoms, in some cases, meningitis and facial palsy. Layered surgical repair leads to high rates of success; however, this may be complicated by worsening facial palsy.
Key Words: Cerebrospinal fluid otorrhea—Facial nerve—Fallopian canal meningocele.

Otol Neurotol **45:**434–439, 2024.

INTRODUCTION

Identifying the cause of cerebrospinal fluid (CSF) otorrhea in a patient without a history of surgery, trauma, infection, or existing tumor can prove difficult. Trauma or infection can impact the integrity of the temporal bone and dura, permitting CSF to flood into the mastoid and the middle ear. A secondary development of a meningocele or encephalocele may develop from these processes and lead to the formation of a fistula. Congenital malformations of the middle ear cavity or mastoid may also provide a pathway for spontaneous CSF otorrhea.

A fallopian canal meningocele (FCM) is an exceedingly rare cause of CSF otorrhea with only 15 reported cases in the literature (1–10). This herniation of the meninges and the facial nerve through the fallopian canal can occur anywhere from the lateral aspect of the internal acoustic canal (IAC) to the stylomastoid foramen, most commonly at the tympanic segment and geniculate ganglion (11). A resulting CSF fistula is thought to form through a damaged or congenitally patent fallopian canal via a disruption of dural tissue or increased CSF pressure gradient that exceeds the tensile capacity of the dura (9). Previous reports suggest an association with increased intracranial pressure (ICP), idiopathic intracranial hypertension, empty sella syndrome, and Arnold Chiari malformation type I (1,9,10). CSF otorrhea or middle ear effusion resulting in unilateral conductive hearing loss is often the first presenting sign (1–8). CSF rhinorrhea may be present due to retrograde flow through the Eustachian tube (8). Less commonly, patients may present with sensorineural hearing loss, facial palsy, or recurrent meningitis (11–14).

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Conflicts of interest: No relevant conflicts of interest were reported by any author.

DOI: 10.1097/MAO.0000000000004155

Surgical treatment is often elected in the setting of recalcitrant CSF otorrhea or rhinorrhea, progressive facial palsy, and recurrent meningitis. Reported techniques include transmastoid or a combined middle cranial fossa and transmastoid approach.

We present a multiinstitutional case series of 11 patients with FCM, reviewing presentation and management for these and all prior 15 cases reported in the literature.

METHODS

Eleven adult patients with FCM between 2015 and 2019 were retrospectively identified at three tertiary referral centers: two in Manhattan, NY, and the third in Virginia. Chart review was performed and demographic data as well as details regarding symptomatic presentation, present comorbidities, radiographic imaging abnormalities (noncontrast computed tomography [CT] of the temporal bone and magnetic resonance imaging [MRI] of the internal auditory canal with and without gadolinium), surgical management (including approach, technique for repair, and use of lumbar drain) and postoperative outcomes (including control of CSF leak, meningitis, and facial nerve function), and revision procedures were reported. Data regarding presentation, surgical management, and clinical outcomes were combined with the available cases reported in the literature. Because of a small sample size, descriptive statistics and case examples were used to present the collected data.

RESULTS

Patient data for our cohort (N = 11) is presented in Table 1. The mean age was 62 years old (range, 46–76 yrs) with a mean length of follow-up of 24 months (range, 8 to 42 mo). Of the patients, 90% had symptoms mimicking serous otitis media, conductive hearing loss, and aural fullness; other symptomatology was variable. Patients presented with spontaneous CSF leak (otorrhea, n = 6; rhinorrhea, n = 1), ipsilateral asymmetrical sensorineural hearing loss ranging from 80 to 95 dB (n = 3), intermittent nonpositional vertigo (n = 3), headaches (N = 4), and recurrent meningitis (n = 1). Risk factors in our series included obesity (n = 4), Chiari 1 malformation (n = 1), and head trauma (n = 2).

The diagnosis was made with a combination of clinical presentation, imaging, a beta-2-transferrin assay, and intraoperative findings. Noncontrast CT of the temporal bone and contrast-enhanced magnetic resonance of the IAC were positive for FCM in 10 of 11 patients; dehiscence was noted at the geniculate ganglion in nine patients, whereas two patients had both the geniculate and labyrinthine dehiscence. Patient 7 did not have a meningocele on CT or MRI, but one was discovered intraoperatively during a tympanomastoidectomy.

Seven of 11 patients had active CSF leaks confirmed with beta-2-transferrin and were managed surgically. One patient without CSF leak, symptomatic with headaches and conductive hearing loss, opted for definitive repair despite knowing the risk to the facial nerve due to patient preference (patient's concerns for enlargement of the encephalocele, future CSF

TABLE 1. *Summary of key findings of 11 patients with facial canal meningocele*

Case	Age/Sex	Presenting Symptoms	Risk Factors	Imaging Findings[a]	Site of Facial Dehiscence [b]	Surgical Approach	Intraoperative Dehiscence	Postoperative Complications
1	39/M	CSF, SNHL, CHL	None	Yes	Geniculate	TM	Geniculate	No
2	74/F	SNHL, CHL vertigo tinnitus	Chiari 1	Yes	Labyrinthine, geniculate	None	N/A	No
3	71/F	CSF, SNHL, CHL, vertigo, HA	None	Yes	Labyrinthine, geniculate	TM, MFC	Labyrinthine, Geniculate	No
4	50/F	CSF, HA	None	Yes	Geniculate	TM	Geniculate	No
5	58/F	CHL	None	Yes	Geniculate	None	N/A	No
6	76/F	CSF, CHL	Obesity, head trauma	Yes	Geniculate	TM	Geniculate	No
7	56/M	CSF, CHL, tinnitus	Obesity	No	None	TM, MFC	None	No
8	47/M	Vertigo	None	Yes	Geniculate	None	N/A	No
9	44/F	CSF, meningitis, facial paresis (HB 2)	Overweight, MVA	Yes	Geniculate	MFC	Geniculate	Yes[c]
10	49/F	CSF, CHL, HA	Obesity	Yes	Geniculate	TM, MFC	Geniculate	No
11	51/F	CHL, HA	Obesity	Yes	Geniculate	TM, MFC	Geniculate	No

F indicates female; CHL, conductive hearing loss; CSF, cerebrospinal fluid leak; HA, headache; HB, House–Brackmann; M, male; MFC, middle fossa craniotomy; MVA, motor vehicle accident with head trauma; SNHL, sensorineural hearing loss; TM, transmastoid; vertigo, nonpositional intermittent vertigo.

[a]Imaging findings: positive findings of FCM on noncontract CT of the temporal bone, MRI of the internal acoustic canal with contrast.

[b]Site of facial dehiscence on imaging.

[c]Postoperative meningitis, worsening facial palsy to HB 2 to HB 4, and CSF rhinorrhea requiring surgical revision (refer to case 9 for details).

leak, or future meningitis). Of the 11 patients, 8 total were managed surgically via a transmastoid approach (n = 4), combined transmastoid and middle fossa craniotomy (n = 3), and middle fossa craniotomy (n = 1); 3 patients were managed conservatively with observation based on patient preference. The observed nonsurgical patients were otherwise complication free and at their baseline symptomatology for an average of 20 months. Types of material for repair include autologous fat graft (n = 3), temporalis muscle (n = 3), and dural regeneration matrix (N = 1). Postoperative complications were noted only in one patient who had worsened facial nerve palsy (House–Brackmann [HB] 2 to HB 4), recurrent meningitis, and recurrent postoperative CSF leak that necessitated revision. The remainder of operated patients (7/8) was free of postoperative CSF otorrhea and rhinorrhea. All operated patients had closure of their air–bone gaps. No other patients suffered from meningitis. Three of 11 patients had placement of a lumbar drain, which was removed before discharge. The three patients managed with close observation were free of any complications (including meningitis or CSF otorrhea and rhinorrhea).

Table 1 summarizes these key clinical characteristics, risk factors, presentations, diagnosis, imaging findings, surgical approaches, and outcomes. Table 2 combines our presentation, surgical management, and clinical outcomes data with the previously published FCM literature.

Case Presentation of Two Key Selected Patients

Case 7

A 56-year-old overweight male (body mass index, 28 kg/m^2) with hypertension presented with a chief concern of constant nonpulsatile tinnitus and right-sided hearing loss associated with aural fullness for 1 year. Apart from a right opacified tympanic membrane, the examination was unremarkable with a normal dilated eye. An audiogram showed a right conductive hearing loss and 30 dB air–bone gap. A myringotomy was performed, and excess clear fluid was positive for Beta 2 Transferrin. The computed tomography of the temporal bone and MRI IAC showed opacification of the right mastoid with a moth-eaten tegmen without an encephalocele (Fig. 1A). The remaining CT images of the geniculate ganglion (not seen in Fig. 1) were deemed normal by the neuroradiologist and attending surgeon.

The patient initially deferred surgical therapy, and his otorrhea resolved, but his tinnitus and hearing loss persisted—the decision was made to repair the defect. Intraoperatively, a fallopian meningomyelocele was encountered in the mastoid cavity and epitympanum extending to the geniculate ganglion where it originated. There were concurrently multiple tegmen dehiscences as well as an active CSF leak (Fig. 1B). A combined transmastoid approach with a temporal craniotomy was utilized to resect the encephalocele with preservation of the facial nerve; a lumbar drain was placed (no opening pressure was recorded at this time), and the defect was repaired with a temporalis muscle rotational flap and an abdominal fat graft. This patient was followed for 2 years postoperatively with audiograms. The conductive component of his conductive hearing loss has since resolved on the right side. The etiology of the FCM is unclear but is likely due to increased ICP from obesity or perhaps a congenital osseous defect.

TABLE 2. *Combined summary of key details of all previously published cases of symptomatic fallopian canal meningocele and this current case series*

Preoperative Clinical Characteristics	Number (N)	Percentage (%)
Age		
Median ± SD (yrs)	34.45 ± 23.88	
Range	2–76	
Gender		
Female	14	52%
Male	12	48%
Preoperative CSF leak	22	88%
Preoperative meningitis	9	36%
Preoperative facial palsy	1	4%
Site of dehiscence		
Geniculate	19	76%
Labyrinthine and geniculate	2	8%
Tympanic	4	16%
Postoperative facial palsy	4	16%
Postoperative CSF leak	5	20%
Repair		
Transmastoid	11	44%
Transmastoid and middle cranial fossa	7	28%
Middle cranial fossa	4	16%
Secondary repairs	5	20%

SD indicates standard deviation.

Case 9

A 44-year-old overweight (body mass index, 28.8 kg/m^2) female with a history significant for recurrent meningitis (four episodes over a 26-year period) presented with progressive left facial nerve paresis, headaches, left-sided watery otorrhea, rhinorrhea, fluctuating conductive hearing loss, and intermittent nonpositional vertigo. Symptoms reportedly started after a motor vehicle accident 26 years prior. The facial paralysis was intermittent or slowly progressive for 2 years, which coincided with an event of meningitis (also 2 yrs before surgical repair). Physical examination showed left facial paresis, HB 2/6, and no middle ear effusion bilaterally. A small clear fluid-filled sac herniating into the left external acoustic canal draining clear fluid was noted. She had a normal dilated eye exam, and previous lumbar punctures showed no sign of elevated pressure.

CTTB and MRI IAC demonstrated a bony defect along the floor of the left middle fossa, including the bone over the temporomandibular joint. There was enlargement of the temporomandibular joint, erosion of the condyle and the ramus of the mandible, dilation of the labyrinthine facial canal and geniculate fossa, as well as demineralization of the clivus and sphenoid wing, all on the left side. There was a small defect of the anterior bony external auditory canal that corresponded to the area of herniation visible on examination. The labyrinthine and geniculate dehiscences are radiographically shown in Fig. 2, A and B. The leak was B2T positive. Videonystagmography for workup of the vertigo was inconclusive.

For personal reasons, the patient opted for surgical repair at this juncture. She underwent a left middle fossa craniotomy with partial decompression of the internal auditory canal and decompression of the seventh nerve medial to the

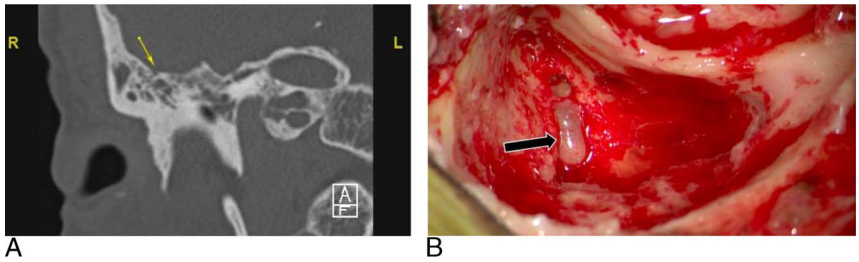

FIG. 1. *A*, Coronal noncontrast CT of the temporal bone; a moth eaten tegmen is seen (arrow), but no meningocele is noted. *B*, Intraoperative view of the right ear, transmastoid approach. The facial canal meningocele (arrow) is herniating into the right mastoid.

geniculate ganglion. On unroofing the geniculate fossa, pulsing fluid was found and thin nerve fibers were noted to be floating in the fluid. The IAC and geniculate fossa were compressed with a dura regeneration matrix; a lumbar drain was placed. Postoperatively, her examination was HB 4/6 and rhinorrhea resolved. She did well for 9 months until she developed a sense of recurring rhinorrhea and had another event of meningitis. At that time, a CT cisternogram was suspicious for CSF leak through the clivus into the sphenoid sinus, and rhinorrhea was positive for B2 transferrin. She then underwent an endoscopic transsphenoidal repair with no further complications.

The etiology of the skull base degeneration and the multiple defects are a long-term result of skull base trauma (the patient did have a motor vehicle accident, although the patient did not have a radiographically evident skull base fracture), congenital bony deformity, obesity, or a progressive process such as Gorham Stout. (15)

DISCUSSION

FCMs are a rare entity, and this cohort of 11 patients adds significantly to the 15 previously reported cases in the literature. Our case series supports prior data suggesting a defect in the fallopian canal associated with increased ICP, idiopathic intracranial hypertension, empty sella syndrome, and/or Arnold Chiari malformation type I (1–10). Patients typically present with conductive hearing loss and CSF otorrhea or rhinorrhea, but they may also demonstrate sensorineural hearing loss, vertigo, headache, facial palsy, or recurrent meningitis, which merits further investigation and surgical intervention, whereas mild symptomatology can be closely observed (11). We found that these clinical characteristics are largely consistent with our series of 11 patients with a few key differences.

Our series confirmed previously noted risk factors of intracranial hypertension, obesity, and Chiari malformation type I as well as presentations of unilateral hearing loss and CSF otorrhea or rhinorrhea (1–8). Table 2 combines all previously published cases with our series as the most up-to-date collection of 26 patients. As previously published, meningitis and facial nerve palsy were also noted in our patients (1–10). The combined male to female ratio is approximately 1:1, supporting a varied etiology (rather than a preponderance of idiopathic intracranial hypertension). The combined findings further strengthen the case that dehiscences may be more common at the geniculate ganglion, but these can be at the labyrinthine and tympanic segments. A successful repair can be performed with transmastoid approaches, but a combined approach may be utilized. Unique to our study is vertigo as the only presenting symptom (Table 1, case 8), which is difficult to rationalize and has not been previously reported. This intermittent but persistent nonpositional vertigo was explored with routine clinical

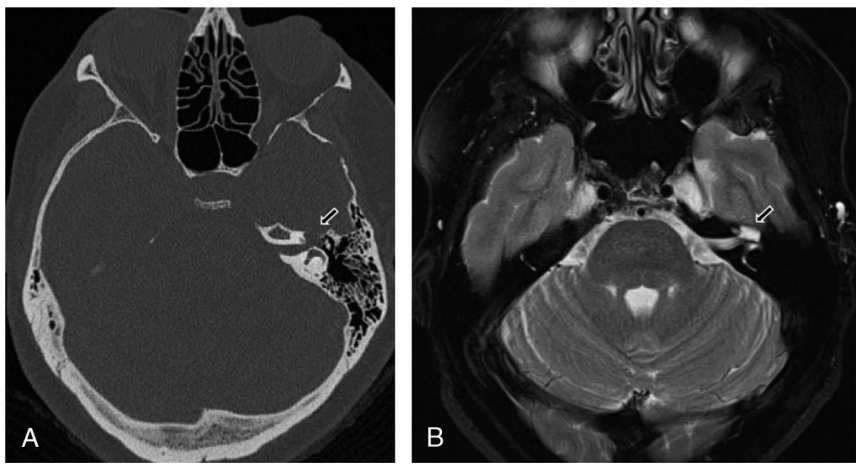

FIG. 2. *A*, Axial noncontrast CT of the temporal bone; the left intralabyrinthine facial nerve and geniculate ganglion (arrow) are enlarged, a finding consistent with FCM. *B*, Axial T2-weighted MRI of the internal acoustic meatus with contrast; a prominence with fluid in the geniculate ganglion on view consistent with fallopian meningocele (arrow).

examination with a videonystagmography—testing was inconclusive. Presumably, the vertigo could have been due to high ICPs rather than an FCM, but without further testing, such as ICP measurements, we cannot draw further conclusions. It is also possible that this symptom was an idiopathic vestibulopathy, which led to the incidental discovery of an FCM. This patient was sent to vestibular rehabilitation but continued to have intermittent symptoms over the course of 12 months of follow-up. A referral to neurology for further testing was placed but deferred by the patient. Another finding unique to our series is negative imaging and an unexpected intraoperative discovery of FCM during a mastoidectomy, a situation that may place the facial nerve at considerable risk.

Understanding the pathophysiology of CSF leaks in FCM is important in explaining symptomatology. A bony defect in the facial canal must form or be present. Ectopic arachnoid granulations in the setting of increased ICPs have been described; these enlarged arachnoid villi may exert pressure and cause chronic erosive trauma to the cranial base leading to its breakdown. Other reasons for this defect are a congenitally patent fallopian canal, temporal bone trauma, as well as increased ICP. As herniated dura stretches into the middle ear or mastoid, progressive dural thinning may lead to a defect, fistulization, and extravasation of CSF. This may present as an effusion or rhinorrhea, leading to symptoms of otitis media. Small defects or low volume leaks may be self-limiting or may escape imaging. Notably, although one might expect increased ICP measurements with lumbar drains or lumbar punctures, patients presenting with CSF leaks may have falsely low or normal pressures.

Making the correct diagnosis of FCM requires a high degree of clinical understanding and suspicion as well as radiographic evidence. Previous studies have noted that high-resolution CT of the temporal bone has shown an enlarged geniculate fossa and widened labyrinthine facial canal segment, but negative imaging has not been described. Key imaging studies include high-resolution CT, MRI of the IAC, and in some cases radionuclide cisternography or CT cisternography. High-resolution CTTB has been reported as more sensitive for detection of tegmen defects than CT cisternography or radionuclide cisternography (16). However, no data on sensitivity and specificity for imaging in the setting of FCM exist at this time. MRI is most helpful in defining larger and multiple cranial base defects and meningoencephalocele with herniation of white and gray matter. Magnetic resonance cisternography may also delineate defective sites, in the absence of active CSF leak (17). For one patient in our cohort, neither CT nor MRI supported a diagnosis of FCM; their pathology was discovered and confirmed intraoperatively. This finding indicates that imaging must be interpreted cautiously with an experienced neuroradiologist; in the case of intraoperative repair of the CSF fistula, the facial nerve may be dehiscent and exposed and prolapsing into the mastoid cavity in an unexpected location. This presents a considerable risk to the facial nerve.

Treatment approaches to FCM require careful considerations of the clinical scenario as well as patient preferences. Although the majority of CSF fistulae seal with minimal intervention, some patients require surgical intervention. Some cases of FCM are asymptomatic and may be an incidental finding on imaging (12–14). Those with minor or no symptoms can be observed. Judicious, close observation is an option in the setting of minimal symptoms as FCM can be an incidental, asymptomatic finding on MRI and CT imaging. Because of anatomic barriers that prevent the flow of CSF and the spread of infection in many cases, these patients may never encounter problems such as CSF leak or meningitis (12–14). Conversely, persistent CSF rhinorrhea, CSF otorrhea, progressive facial palsy, and recurrent meningitis often necessitate repair. Approaches require tympanomastoidectomy with or without middle fossa craniotomy. A successful long-term repair requires a multilayer closure with autologous material—often fat or temporalis—in combination with artificial materials and requires long-term follow-up (1–8,18). Overlay of the defect is often done with temporalis fascia, bone pate, abdominal fat graft, fibrin glue, or a combination of these materials. Secondary subtotal petrosectomy, with obliteration of the middle ear and Eustachian tube and blind sac closure of the external acoustic canal, has been described as well (11).

Other considerations include the breach of natural barriers allowing retrograde of pathogens into the CNS; although the use of antimicrobials does not prevent meningitis and their use is controversial, this retrograde pathway may lead to encephalitis or meningitis in up to 10% of all patients with CSF otorrhea or rhinorrhea (18). In particular, pediatric patients with congenital fistula and spontaneous CSF leak are three times more likely to develop meningitis (18). In our series, one adult patient had recurrent meningitis, likely due to this mechanism; she did not have any neurologic sequelae other than a preexisting facial palsy. It is difficult to make recommendations regarding the role of antibiotics and pneumococcal vaccinations given the rarity of this pathology (26 total cases now with the addition of this case series). It is reasonable to apply the existing CSF leak and encephalocele data to this scenario; as such age-appropriate preoperative pneumococcal vaccination and perioperative antibiotics may be applicable at the discretion of the otologic surgeon, and prophylactic antibiotics remain controversial (18,19).

The location of dehiscence of the fallopian canal where the distorted facial nerve anatomy lies has previously been reported as a site of intraoperative injury (15). Careful packing and instrumentation in the proximity of the geniculate fossa is necessary to prevent injury to the facial nerve, as the risk of postoperative facial nerve palsy is not negligible as noted by previous case reports (1–8). Case 9 demonstrates a complex defect with recurrent meningitis as well as a preoperative HB 2 facial palsy; this case was complicated by worsening postoperative facial palsy (HB 4) and subsequent recurrence of meningitis and CSF rhinorrhea. From this, one may deduce that larger, more complex defects are more susceptible to infection and facial nerve complications.

CONCLUSIONS

Meningocele through the fallopian canal is an exceedingly rare finding and presents a diagnostic challenge for

the otologic surgeon. In addition to CSF otorrhea or rhinorrhea as well as conductive hearing loss, FCM may present with nonspecific otologic symptoms including vertigo, as well as headache; less commonly, cases may present with meningitis and facial palsy. Risk factors noted in our series include obesity, head trauma, and Chiari 1 malformation. Diagnosis requires a high degree of clinical suspicion and careful review of imaging. Surgical management varies but includes transmastoid, middle cranial fossa or combined approaches. In patients with a low burden of symptoms, careful observation may be elected.

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