

## CASE REPORT

# Enterococcus Faecalis Brain Abscess after Tympanoplasty for Middle Ear Cholesteatoma

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**ABSTRACT:** Despite the significant progress achieved in era of antibioticotherapy, the rate of suppurative complications of chronic otitis media (COM) remains alarming, and there is a growing evidence of unusual causative bacteria. We present a case of post-mastoidectomy wound infection caused by *Enterococcus Faecalis* that resulted in brain abscess and was resolved by means of a craniotomy with skull base reconstruction, revision mastoidectomy and long-term antibiotic therapy. We also discuss the possible pathogenesis of this rare complication.

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## Introduction

Suppurative complications of COM are traditionally divided into extracranial and intracranial. Meningitis, lateral sinus thrombosis and brain abscess are the most common among the latter, while mastoiditis with subperiosteal abscess is the most frequent extracranial one.<sup>[1]</sup> Complications of COM usually follow exacerbation of the disease and are more common when COM is associated with cholesteatoma.<sup>[2, 3]</sup> On one hand, the appropriate and widespread use of antibiotic therapy has decreased their incidence although it remains significant: 0.45% of all COM patients are prone to develop an intracranial complication, while 3.07% an extracranial one; these rates did not seem to have changed over the last 10 years.<sup>[4]</sup> On the other hand, some reports have appeared in the literature about the insurgence of complications related to unusual causative bacteria.<sup>[4-6]</sup> Possible reasons may be the development of drug resistance among the pathogens typical for COM or a real upraise of rare bacterial strains. We report the first case, to our knowledge, of post-mastoidectomy wound infection and brain abscess caused by *Enterococcus faecalis*.

## Case Report

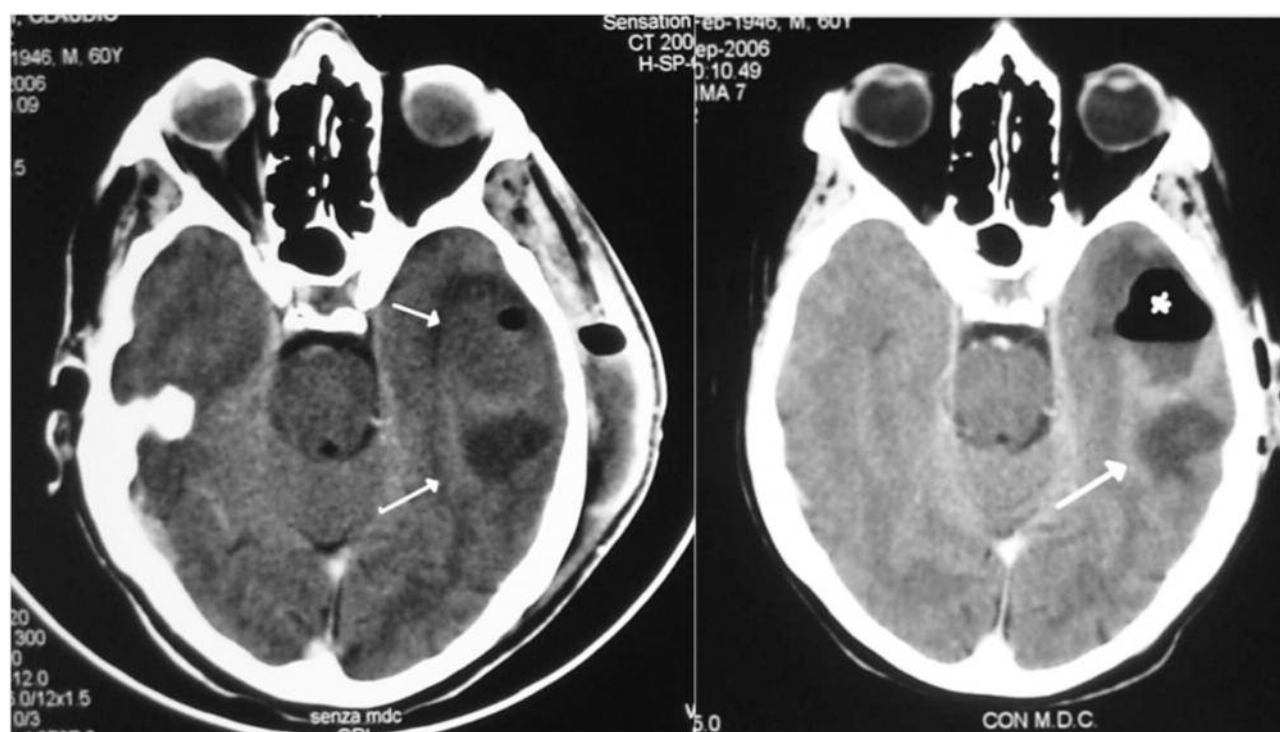
A 60-year-old male farmer was referred to our department with a history of recurrent purulent left otitis media, resistant to local and systemic antibiotic therapy. On presentation, a left epi-mesotympanic cholesteatoma with fowl smell otorrhea, without otalgia or vestibular complaints, was observed. Pure tone audiometry ascertained a mixed moderate-to-severe hearing loss. Physical examination of the vestibular function was normal. The patient underwent a canal wall-up mastoidectomy with wide atticotomy and posterior tympanotomy. An extensive meso-epitympanic and mastoid cholesteatoma was cleared, leaving an incomplete fistula of the lateral semicircular canal and a dehiscence of the Fallopian canal in its tympanic portion. The cholesteatoma removal was particularly challenging in the region of oval and round window niches, and from the sinus tympani area. The eardrum perforation was repaired by means of an autologous fascia graft in an underlay fashion; the totally absent ossicular chain was reconstructed by means of titanium total ossicular reconstruction prosthesis (Gyrus Acmi Inc., Southborough, MA). The patient was put on

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prophylactic treatment with ciprofloxacin, 250 mg twice a day. The initial post-operative course showed the clinical picture of left (acute) vestibular dysfunction that was treated with antiemetics. On the 6th postoperative day there appeared a high grade remitting fever, retroauricular wound swelling and purulent otorrhea; few hours later the patient complained of otalgia and manifested vomiting, aphasia and state of confusion without meningeal or additional focal neurological signs. An urgent computed tomography (CT) showed a left temporal lobe abscess in its initial stage (Figure 1). Bacterial cultures obtained from the operative wound and by an ear swab identified an Group D *Enterococcus Faecalis*. After an Infectious Diseases Department consultation, a prompt treatment with vancomycin 500 mg 4 times per day and meropenem 2 g t.i.d. was started according to the results of the antibiogram. Since no clinical response was achieved during the following 8 days, and the size of brain abscess did not change according to the repeated CT scans, the patient underwent a revision canal wall down mastoidectomy combined with a craniotomy for the drainage of

temporal lobe abscess. Granulation tissue mixed with necrotic material and areas of osteitis were found in the mastoidectomy well. Periphlebitis of the sigmoid sinus and a dehiscence of the middle cranial fossa cortical plate were observed, with exposure of the dura mater and granulation tissue (Figure 2). After its cleansing and evacuation of the abscess, the skull base was repaired with a temporal musculofascial rotation flap as an intracranial layer. The sinodural-angle was filled with bone patè mixed with fibrin glue over a second (extracranial) layer of autologous fascia. A wide 2-flaps meatoplasty completed the procedure. The antibiotic therapy was changed to ampicillin 2 g, six times per day and levofloxacin 500 mg b.i.d. for 3 further weeks. The patient gradually improved, becoming afebrile in a few days; he recovered from vertigo, and the purulent auricular secretions resolved. The aphasia remarkably improved over the next 3 weeks, and the patient is alive and well 2 years after the operation. On the most recent CT scan (1 year after treatment), complete resorption of the brain abscess was observed. The neotympanum is preserved and the posterior cavity is dry and well epithelialized.



**Figure 1.** Plain CT on axial plane showing a left temporal lobe abscess with central fluid collection and a peripheral enhanced rim (fine arrows); air is recognizable in its anterior part (asterisk)



**Figure 2.** Intraoperative picture showing elevator in the dehiscence of the middle cranial fossa floor through a subtemporal craniotomy, exposure of the dura mater and granulation tissue. Necrotic skin and subdermal tissue is visible at the incision site (arrow) over the mastoidectomy well.

## Discussion

In the antibiotic era the incidence of intracranial suppurative complications of COM has dropped from 2-6%<sup>[3]</sup> to 0.02-0.45%<sup>[4,6]</sup>. Nevertheless, they are still dreaded, due to the high mortality rate, especially in populations with lower socio-economic conditions ( $\approx 20\%$ ).<sup>[1]</sup> Some reports have appeared in the literature about a possible increasing incidence of intracranial otogenic complications also in industrialized countries.<sup>[7,8]</sup> There might be a few explanations for this tendency. Firstly, antibiotic therapy may have a masking effect on symptoms of the complications, causing delay in diagnosis. Inappropriate use of antibiotics (as in cases of viral infections, insufficient duration of treatment, use of inappropriate agents) may lead to the development of drug resistance of the typical causative pathogens and to the arousal of the unusual ones, though to date there is no clear evidence supporting it. Finally, the advances of antineoplastic chemotherapy lead to a significant growth of immune deficient population prone to infectious complications.

The causative pathogens typical for COM are the Gram negative *Proteus mirabilis*, *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, and anaerobic *Bacteroides* spp.<sup>[4,9]</sup> In some instances, Gram positive such as *Strep. pneumonia* and *Staph. Aureus* are involved<sup>[10]</sup>. Up to 39% of the COM cases the

middle ear cultures are negative, probably due to preceding antibiotic treatment or inadequate diagnostic technique precluding discovery of anaerobic agents.<sup>[11,12]</sup>

Post-operative intracranial suppurative complication in COM patients is rare.<sup>[16]</sup> The postmastoidectomy course can be complicated by wound problems (7.7–42.1%)<sup>[13]</sup>, meningitis, brain abscess, subdural empyema, sigmoid sinus thrombosis and epidural collection.<sup>[14]</sup> The infection can spread from the middle ear or mastoid to the intracranial compartment by direct extension through bone erosion, via thrombophlebitis of the cortical veins or of the sigmoid sinus, via preformed pathways or via hematogenous dissemination. The preformed pathway can be a normal opening in the bony wall, such as the oval or round window, or the developmental dehiscence of the floor of the hypotympanum, or it could be the result of a skull fracture or the previous ear surgery.

In our patient, the most likely pathogenetic mechanism is the inflammatory erosion of the tegmen tympani as can be presumed by the contiguity location of the temporal lobe abscess. The evidence of a labyrinthine fistula makes the trans-labyrinthine pathway of infection spread also possible, although in that case posterior cranial fossa involvement through the internal auditory canal would be expected. The third possible route of infection spread is a retrograde haematogenous one, via the sigmoid sinus, which was involved by the perisinus abscess, although no intraluminal thrombus could be identified. Virulence of the unique bacterial strain responsible for this complication can account for the rapid development of a dreadful intraparenchymal abscess which, still nowadays, has a potentially fatal outcome. This virulence can also explain the appearance of the brain parenchyma abscess without the preceding epidural abscess or subdural empyema. The presence of such a rare causative agent as *Enterococcus Faecalis* might be explained with professional exposure of the patient to domestic animals excretions, while faecal self-contamination cannot be excluded.

## Conclusion

Analysis of this case and review of the pertinent literature should alert the Otolaryngologists about the post-operative occurrence of severe intracranial suppurative disease possibly related with microbial agents that are unusual for COM.

Surgery may create circumstances favouring the spread of the infection. Combined with the high virulence of *Enterococcus Faecalis*, it may lead to an unexpected course of the disease, with a temporal lobe intraparenchymal abscess appearing early in the post-operative period and without warning clinical signs

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