

Temporal Epidural Abscess Complicating Acute Coalescent Mastoiditis in an Immunocompetent Young Adult: A Rare Case Report and Literature Review

Rsaissi C, El Mourabit F*, Lahjaouj M, Loudghiri M, Bijou W, Oukessou Y, Rouadi S, Abada R, Roubal M and Mahtar M

Department of Otolaryngology Head and Neck Surgery, University hospital Ibn Rochd, Casablanca, Morocco

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***Corresponding author:** Fadoua El Mourabit, Department of Otolaryngology Head and Neck Surgery, University hospital Ibn Rochd, Casablanca, Morocco

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A B S T R A C T

Background: Acute otitis media (AOM) and acute mastoiditis are common otologic infections; however, intracranial complications have become exceptionally rare in the antibiotic era. Among these, temporal epidural abscess represents an uncommon but potentially life-threatening condition requiring prompt diagnosis and multidisciplinary management. Delayed recognition may result in severe neurological sequelae despite initially subtle clinical manifestations.

Case presentation: An 18-year-old immunocompetent male presented with a two-week history of right-sided otalgia, persistent temporal headache, fever and progressive hearing loss. Despite empirical medical treatment, his symptoms worsened with the onset of vertigo. Clinical examination revealed purulent otitis without neurological deficits or facial nerve palsy. Laboratory investigations demonstrated an inflammatory syndrome. Contrast-enhanced computed tomography showed acute coalescent mastoiditis associated with a right temporal epidural abscess measuring 22.5×10 mm without evidence of cerebral abscess or dural venous sinus thrombosis. The patient underwent urgent cortical mastoidectomy combined with intravenous broad-spectrum antibiotic therapy for 10 days. The postoperative course was uneventful, with complete resolution of symptoms and no recurrence during follow-up.

Conclusions: Temporal epidural abscess remains a rare but serious intracranial complication of acute mastoiditis. Persistent headache disproportionate to otologic findings should raise suspicion for intracranial extension, even in the absence of neurological deficits. Early contrast-enhanced imaging, timely surgical intervention and prolonged intravenous antibiotic therapy are essential to achieve favorable outcomes.

Keywords: Acute otitis media; Acute coalescent mastoiditis; Temporal epidural abscess; Intracranial complication; Mastoidectomy; Case report

Introduction

Acute otitis media (AOM) remains one of the most common infectious diseases encountered in clinical practice and continues to represent a significant cause of morbidity worldwide despite the widespread availability of antibiotics. Although predominantly affecting children, AOM also occurs in adolescents and adults, in whom delayed diagnosis may predispose to suppurative complications. Since the introduction of antimicrobial therapy and pneumococcal vaccination, the incidence of both extracranial and intracranial otogenic complications has declined dramatically, making these entities relatively uncommon in modern otologic practice^{1,2}. Nevertheless, when present, they remain associated with considerable morbidity and potentially life-threatening consequences if diagnosis and treatment are delayed^{2,3}.

Acute mastoiditis is the most frequent suppurative complication of AOM and results from the extension of infection into the mastoid air-cell system. Progressive osteitis may lead to destruction of the intercellular septa, resulting in coalescent mastoiditis, which facilitates the spread of infection beyond the temporal bone. Depending on the route of dissemination, the infection may extend extracranially, producing subperiosteal, temporal, Bezold or zygomatic abscesses or intracranially through bony erosion or thrombophlebitis of diploic veins. Intracranial complications include epidural abscess, subdural empyema, meningitis, brain abscess and dural venous sinus thrombosis³⁻⁵.

Among intracranial complications, epidural abscess represents one of the most frequent localized suppurative collections of otogenic origin. It develops following direct extension of infection through erosion of the tegmen mastoideum or adjacent temporal bone, allowing pus to accumulate between the dura mater and the inner table of the skull. Because the dura initially limits the spread of infection, patients may present with persistent headache, fever and otologic symptoms while remaining neurologically intact. Consequently, diagnosis may be delayed despite the presence of a potentially life-threatening intracranial infection^{4,6}.

Contrast-enhanced computed tomography (CT) remains the first-line imaging modality for the evaluation of suspected complicated mastoiditis because it accurately identifies mastoid opacification, cortical bone erosion and intracranial extension. Magnetic resonance imaging (MRI) provides superior assessment of epidural collections, cerebritis, venous sinus thrombosis and associated intracranial complications and should be considered whenever neurological involvement is suspected or CT findings are equivocal^{3,5,7}.

Current management relies on prompt intravenous broad-spectrum antibiotic therapy combined with surgical eradication of the infectious focus, most commonly by mastoidectomy. Neurosurgical drainage is reserved for selected patients with large epidural abscesses, mass effect or associated intracranial complications^{1,4,8}.

Despite advances in antimicrobial therapy, imaging techniques and otologic surgery, temporal epidural abscess secondary to acute coalescent mastoiditis remains an exceptionally rare entity in immunocompetent young adults.

We report the case of an 18-year-old immunocompetent patient presenting with a temporal epidural abscess complicating

acute coalescent mastoiditis, successfully treated with urgent mastoidectomy and intravenous antibiotic therapy and provide a review of the current literature focusing on diagnostic challenges and therapeutic management.

Case Presentation

An 18-year-old previously healthy male presented to the Department of Otorhinolaryngology with a two-week history of acute right-sided otalgia associated with persistent right temporal headache that was poorly responsive to analgesics. The patient denied otorrhea or tinnitus but reported fever throughout the course of the illness. One week after symptom onset, his condition progressively worsened with increasing headache intensity, the onset of rotational vertigo and right-sided hearing loss. He denied facial weakness, visual disturbances, altered consciousness, seizures or other neurological symptoms. Prior to referral, he had received empirical injectable antibiotic therapy associated with local ear treatment at another healthcare facility without clinical improvement.

His past medical history was unremarkable except for allergic rhinitis. There was no history of chronic otitis media, previous otologic surgery, diabetes mellitus, immunodeficiency, head trauma or other significant comorbidities.

On admission, the patient was conscious, alert and hemodynamically stable but febrile, with a body temperature of 39°C. Otoloscopic examination of the right ear demonstrated marked inflammatory edema of the external auditory canal filled with abundant purulent secretions, preventing adequate visualization of the tympanic membrane (**Figure 1**). Examination of the left ear was unremarkable.

Neurological examination revealed no signs of meningeal irritation or focal neurological deficits. Facial nerve function was normal (House–Brackmann grade I) and no other cranial nerve abnormalities were detected. Vestibular examination showed no spontaneous nystagmus. Romberg testing demonstrated deviation toward the right side, whereas both the Fukuda stepping test and fistula test were negative.

Examination of the head and neck revealed no retroauricular swelling, erythema, fluctuation or tenderness over the mastoid region. No cervical lymphadenopathy was identified.

Laboratory investigations demonstrated an inflammatory syndrome, with a white blood cell count of $10.19 \times 10^9/L$, including $7.67 \times 10^9/L$ neutrophils and an elevated C-reactive protein level of 6.5 mg/dL.

Pure-tone audiometry demonstrated moderate conductive hearing loss of the right ear, with an average air-conduction threshold of approximately 50 dB HL and preserved bone-conduction thresholds, while hearing in the contralateral ear was within normal limits (**Figure 2**).

Contrast-enhanced computed tomography (CT) of the temporal bones revealed complete opacification of the right external auditory canal, middle ear cavity, aditus ad antrum and mastoid air cells, associated with destruction of the mastoid septa and focal erosion of the mastoid cortex, consistent with acute coalescent mastoiditis. In addition, a well-defined rim-enhancing epidural collection measuring 22.5×10 mm was identified along the right temporal region, compatible with a temporal epidural abscess (**Figure 3**). No evidence of cerebritis, brain abscess,

hydrocephalus, midline shift, posterior fossa involvement or dural venous sinus thrombosis was observed (**Figure 1**).

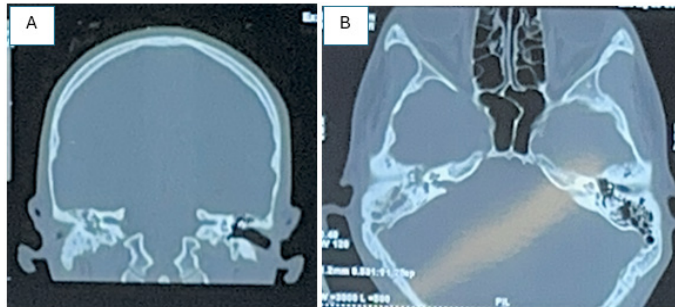


Figure 1: Preoperative contrast-enhanced computed tomography (CT) of the temporal bone.

(A) Coronal CT image demonstrating complete opacification of the right mastoid air cells and middle ear cavity, associated with cortical mastoid erosion (arrow), consistent with acute coalescent mastoiditis.

(B) Axial CT image showing complete opacification of the right mastoid and middle ear, with an adjacent right temporal epidural collection (arrow), compatible with a temporal epidural abscess.

Given the radiological findings and the risk of further intracranial extension, the patient underwent urgent cortical mastoidectomy under general anesthesia. Intraoperatively, the mastoid cavity contained abundant purulent material associated with extensive inflammatory granulation tissue, confirming the diagnosis of acute coalescent mastoiditis. Complete drainage of the infectious focus was achieved without intraoperative complications.

Postoperatively, the patient received intravenous broad-spectrum antibiotic therapy for 10 days, together with regular local wound care and close clinical monitoring. His postoperative course was uneventful, with rapid resolution of fever, progressive disappearance of headache and vertigo and marked improvement in otologic symptoms. No facial nerve dysfunction or neurological complications developed during hospitalization. The patient was discharged in good clinical condition and remained asymptomatic during follow-up, with no evidence of recurrent infection.

Discussion

Although the incidence of acute mastoiditis has markedly declined since the introduction of antibiotics, it remains the most frequent suppurative complication of acute otitis media (AOM) and continues to represent the principal source of otogenic intracranial infections¹⁻³. Recent evidence suggests that intracranial complications occur in approximately 6-23% of patients with complicated mastoiditis, with epidural abscess, brain abscess, meningitis and lateral sinus thrombosis representing the most frequently encountered entities^{2,9-11}. Despite major advances in antimicrobial therapy, high-resolution imaging and surgical techniques, these complications remain associated with significant morbidity and a reported mortality approaching 11% in contemporary series².

The pathophysiology of otogenic epidural abscess is closely related to the progression of acute mastoiditis toward coalescent mastoiditis, characterized by osteitis and destruction of the mastoid trabeculae. As the inflammatory process progresses, infection may spread directly through erosion of

the tegmen mastoideum or tegmen tympani into the epidural space. Alternatively, microorganisms may disseminate through thrombophlebitis involving the diploic or emissary veins without obvious cortical destruction^{3-5,10,12}. The dura mater initially limits further extension of infection, explaining why epidural abscesses may remain localized and clinically silent despite their intracranial location^{4,6,13}.

One of the major diagnostic challenges lies in the nonspecific clinical presentation of epidural abscesses. Headache, fever, persistent otalgia, hearing loss and otorrhea constitute the most common presenting symptoms, whereas focal neurological deficits, seizures, altered mental status or papilledema usually develop only after considerable intracranial extension^{2,9,13-15}. In their systematic review of 1,307 otogenic brain abscesses, Duarte et al. identified headache as one of the most frequent presenting complaints, whereas neurological deficits were considerably less common during the initial stages of disease⁹. Similarly, Gkrinia, et al. reported headache in nearly two-thirds of patients with otogenic intracranial complications, emphasizing that the absence of neurological findings should never exclude intracranial spread².

Our patient illustrates these observations remarkably well. Despite harboring a 22.5 × 10 mm temporal epidural abscess, he remained neurologically intact throughout his presentation. Persistent temporal headache associated with fever and progressive conductive hearing loss represented the principal warning signs leading to further investigation. This emphasizes the importance of maintaining a high index of suspicion whenever severe headache appears disproportionate to otologic findings, particularly in patients who fail to respond to initial antibiotic therapy^{2,7,9,14,16}.

Radiological imaging remains the cornerstone for early diagnosis. Contrast-enhanced computed tomography (CT) is widely considered the first-line imaging modality because of its rapid acquisition, broad availability and excellent ability to demonstrate mastoid opacification, cortical erosion and associated intracranial collections^{3,7,10,17}. MRI, particularly with diffusion-weighted imaging and gadolinium enhancement, provides superior visualization of epidural collections, dural inflammation, cerebritis, venous sinus thrombosis and early brain abscesses, making it the preferred complementary examination whenever neurological deterioration is suspected or CT findings remain equivocal^{5,9,17,18}. In the present case, contrast-enhanced CT accurately identified acute coalescent mastoiditis together with a temporal epidural abscess while excluding parenchymal abscess, hydrocephalus and dural venous sinus thrombosis, allowing immediate surgical management.

Surgical management

The optimal management of otogenic epidural abscess remains controversial because of the rarity of this condition and the absence of universally accepted treatment guidelines. Nevertheless, there is broad consensus that coalescent mastoiditis associated with intracranial extension requires prompt surgical eradication of the primary infectious focus, in combination with prolonged intravenous antimicrobial therapy^{1,2,8,18,21}. Cortical mastoidectomy remains the cornerstone of treatment, as it provides adequate drainage of infected mastoid air cells, removes necrotic bone and eliminates the source of ongoing intracranial contamination^{1,8,21,26}.

The role of neurosurgical drainage depends primarily on the size of the epidural abscess, the presence of mass effect, neurological deterioration, associated cerebral abscess or failure of conservative management. Several authors have demonstrated that small epidural abscesses without neurological deficits may be successfully managed with mastoidectomy alone, provided that complete source control is achieved and close clinical and radiological follow-up is ensured^{9,10,21,30}. Conversely, larger collections or those associated with significant intracranial complications generally require combined otologic and neurosurgical intervention^{2,4,18}.

In the present case, the epidural abscess measured 22.5 × 10 mm and the patient remained neurologically intact without evidence of mass effect, cerebritis, brain abscess or dural venous sinus thrombosis. These findings supported the decision to perform urgent cortical mastoidectomy without neurosurgical drainage. Complete surgical eradication of the mastoid infection, followed by 10 days of intravenous broad-spectrum antibiotic therapy, resulted in rapid clinical recovery and complete resolution of symptoms. The favourable outcome observed in our patient is consistent with recent reports indicating that carefully selected patients with limited epidural abscesses can be successfully treated through mastoidectomy alone, thereby avoiding unnecessary intracranial surgery^{13,21,30}.

Although the optimal duration of antimicrobial therapy remains undefined because of the rarity of this condition, most authors recommend broad-spectrum intravenous antibiotics for at least 2-6 weeks, with treatment tailored according to microbiological findings, radiological evolution and clinical response^{6,18}. In our patient, the excellent postoperative clinical evolution, absence of neurological complications and complete symptom resolution after 10 days of intravenous therapy supported the effectiveness of the chosen therapeutic strategy.

The microbiology of otogenic intracranial infections has evolved over recent decades. Historically, *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Streptococcus* species and anaerobic bacteria predominated, particularly in chronic suppurative otitis media^{9,19}. In contrast, acute mastoiditis is more frequently associated with *Streptococcus pneumoniae*, *Streptococcus pyogenes* and *Staphylococcus aureus*^{1,3,20}. Previous antibiotic exposure frequently results in sterile intraoperative cultures, reported in up to one-third of cases, highlighting the importance of initiating empirical broad-spectrum intravenous therapy without delaying surgical intervention^{9,20}.

Management requires prompt multidisciplinary collaboration between otolaryngologists, neuroradiologists, infectious disease specialists and neurosurgeons^{2,9,18}. Current evidence supports the early administration of intravenous broad-spectrum antibiotics covering aerobic and anaerobic organisms, followed by culture-directed therapy whenever microbiological results are available^{18,21}. Surgical eradication of the primary infectious focus by mastoidectomy remains the cornerstone of treatment for coalescent mastoiditis with intracranial extension^{1,3,10}. Neurosurgical drainage is generally reserved for patients presenting with large epidural collections, significant mass effect, neurological deterioration or concomitant brain abscess^{9,13,18,21}. Interestingly, several recent series have demonstrated successful management of selected epidural abscesses through mastoidectomy alone, provided that adequate source control is achieved and close radiological follow-up is ensured^{13,21}.

Our patient underwent urgent cortical mastoidectomy combined with ten days of intravenous antibiotic therapy, resulting in complete clinical recovery without neurological sequelae. The favourable outcome can probably be attributed to early radiological diagnosis, timely surgical intervention and the absence of associated intracerebral abscess or venous sinus thrombosis. Similar outcomes have been reported in recent case series emphasizing the prognostic importance of early diagnosis before neurological deterioration occurs^{2,9,13,21}.

Finally, this case highlights an important clinical message. Although intracranial complications of AOM have become exceedingly rare in the antibiotic era, they should still be considered whenever patients present with persistent headache, fever or worsening symptoms despite adequate medical treatment. The absence of neurological deficits should not reassure clinicians, as epidural abscesses may remain clinically occult until advanced stages. Early contrast-enhanced CT, followed, when necessary, by MRI, together with prompt mastoidectomy and appropriate intravenous antimicrobial therapy, remain the cornerstone of successful management and are associated with excellent functional outcomes^{2,3,5,9,18,21}.

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