Acute mastoiditis with temporomandibular joint effusion

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Acute otitis media has a markedly lower incidence of intra- and extratemporal sequelae today than in the pre-antibiotic era. Among the complications of acute otitis media, mastoiditis continues to be the most common intratemporal complication. The classic description of acute mastoiditis involves a child with antecedent acute otitis media who develops high fever, severe otalgia, postauricular erythema, and tenderness. There is frequently a degree of postauricular swelling, suggesting the presence of subperiostial abscess.

Contemporary studies of mastoiditis describe the clinical presentation with several prominent clinical features, including otalgia (98% incidence), fever (83%), abnormal appearance of the tympanic membrane (88%), abnormal appearance of the external auditory canal (80%), postauricular edema (76%), auricular proptosis (42%), and otorrhea (26%).[1]

Because the overall incidence of mastoiditis has plummeted in the postantibiotic era, it is less frequently encountered by the otolaryngologist. Atypical presentations, therefore, have become proportionately more scarce and present a diagnostic challenge to the clinician.

We describe a case of acute, coalescent mastoiditis whose atypical presentation, with massive TMJ effusion and trismus, highlights the anatomic relationship of this joint to the mastoid and middle ear.

CASE REPORT

An 11-year-old female with no otologic history and no significant medical history presented to the emergency department with a 4-day history of left-sided otalgia. She had arrived from Eastern Europe 4 weeks earlier and shortly after arrival developed bilateral otalgia and a low-grade fever. She was diagnosed with bilateral acute otitis media and given a 10-day course of antibiotics orally with complete resolution of her symptoms. Four days before presentation, she developed left-sided otalgia and was again diagnosed with acute left otitis media and treated with amoxicillin. She presented to our emergency department with increased left-sided otalgia. History revealed daily ocean swimming. Her physical examination was significant for marked tragal tenderness and fullness, and an edematous external auditory canal (EAC). A small posterosuperior crescent of her tympanic membrane was visible; however, mobility could not be adequately assessed because of significant canal edema. Her mastoid tip was nontender and nonerythematous, and the auricle was nonproptotic. She had normal hearing bilaterally, and no vestibular symptoms. The remainder of her otolaryngologic examination was normal. She was diagnosed with otitis externa and given cortisporin otic suspension and placed on water precautions. Because her middle ear was not able to be thoroughly assessed, the presence of infection was presumed, and she was continued on oral antibiotics. Thirty-six hours later, she returned with worsening otalgia, marked trismus (15 mm), left-sided neck tenderness, and tenderness over the left mastoid tip. On occlusion, her mandible deviated 3 mm to the right.

A CT scan with contrast revealed coalescent mastoiditis with destruction of the mastoid bony septations. There was a large, rim-enhancing lesion occupying the TMJ, consistent with a septic effusion (Fig 1). The middle ear was occupied with soft tissue, suggesting suppuration.

The patient underwent simple mastoidectomy, myringotomy, and aspiration of the TMJ. Several milliliters of purulent material were evacuated from the middle ear cavity, and the mastoid cavity contained a moderate amount of granulation tissue. The TMJ aspirate appeared as a cloudy liquid. Cultures from the middle ear grew group A *Streptococcus* mucoid colonies.

She was treated with broad spectrum intravenous antibiotics; coverage was narrowed when cultures demonstrated appropriate sensitivity. Her occlusion returned to normal over the course of several days. She did well with a 7-day course of intravenous antibiotics and was discharged on a course of oral antibiotics on postoperative day 7.

DISCUSSION

Infection in any anatomic space, including the mastoid, is prevented from disseminating by a series of anatomic barriers.[2] Because bone provides the best of these barriers, the presence of bony destruction or the absence of complete bony partitioning (as a result of

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congenital factors or trauma) increases the likelihood of spread of infection to adjacent areas.

In children, dense barriers of bone may not yet have developed to the point where this prevention of extension occurs. The very young are most at risk for suppurative otologic infections due to immature eustachian tube function, and they have not yet undergone complete bony development of the temporal bone, glenoid fossa, and TMJ. This combination of factors puts this age group at high risk for spread of infection between these two anatomically distinct regions. Keith reports that complete maturation in this region does not occur until well into the second decade of life.[3] Other investigators report that the tympanosquamosal suture can remain open medially, and is divided into both petrosquamous and petrotympanic portions by the tegmen tympani.[4] Still others have examined the timing of ossification of the tympanic plate, and report that this can be delayed until beyond age 10, and in 20% of skulls, may never completely ossify well into adulthood.[4]

Our case demonstrates that the relationship between the mastoid, middle ear cavity, and TMJ is not simply theoretical, but must be borne in mind in all cases of otologic infection. A 1914 discussion of how the mandibular articulation becomes involved during an acute otitis media included direct extension (through the foramen of Huschke), mandibular osteitis extending to the glenoid cavity, or hematogenous metastasis to the TMJ from the focus of infection.[4] Review of a series of cases of TMJ ankylosis revealed that infection, including acute otitis media and mastoiditis, was second only to trauma as a cause for delayed TMJ ankylosis.[5]

This case illustrates the close anatomic relationship of the middle ear and mastoid cavity to the glenoid fossa and TMJ. It is critical that this anatomic relationship be appreciated in order to correctly diagnose mastoiditis when the presenting symptoms, physical examination, or radiographic evaluation implicate TMJ pathology.

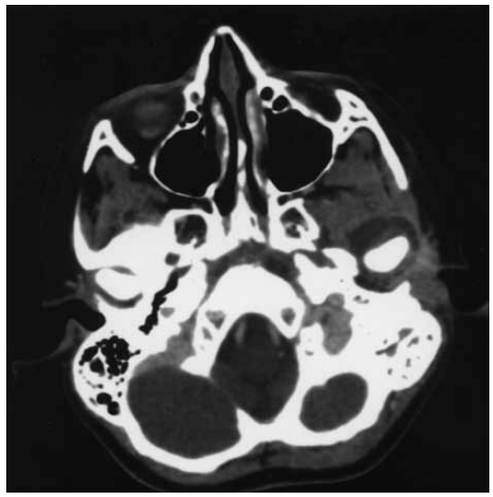

Fig 1. Computed tomography through the TMJs reveals a 3-cm effusion in the left joint space. Also note the ipsilateral coalescent mastoiditis.

Because mastoiditis is now an uncommon clinical problem, the practicing otolaryngologist has less experience in diagnosis. One must have a high index of suspicion in cases where a sign or symptom of middle ear infection recurs several weeks after an initial episode of acute otitis media.

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