Aspergillus mastoiditis

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Mycotic infections of the head and neck region are being reported much more frequently in the last decade. The majority of patients are immunocompromised and the most familiar examples to the otolaryngologist are rhinocerebral mucormycosis and invasive external otitis. Aspergillus has been recognized in infections of the paranasal sinuses since 1891,1 and in invasive external otitis since 1985.2 Documented cases of Aspergillus mastoiditis are exceedingly rare3,4 and the majority of these cases represent either hematogenous or direct spread to the temporal bone from another primary source. Only one of these reports4 documents primary Aspergillus mastoiditis with no evidence of any other source.

CASE REPORTS

Case 1. A 24-year-old man with AIDS had an 8-month history of intermittent left otorrhea, left facial weakness for 3 weeks, and fevers, chills, and headache for 1 week. He denied any tinnitus, vertigo, or otalgia, and noted a mild subjective hearing loss. Bilateral tympanostomy tubes had been placed approximately 6 weeks earlier by another otolaryngologist for chronic middle ear effusion. Physical examination revealed a temperature of 102.2° F, mild left facial nerve paresis involving all branches (incomplete eye closure, symmetry at rest), no evidence of nystagmus, and purulent drainage through the left tympanostomy tube. There was no granulation tissue in the ear canal.

The chest radiograph (x-ray film) was normal and initial laboratory workup revealed Hct 21.0, WBC 3800/mm³ (93% neutrophils), and elevated liver enzymes as a result of AZT toxicity. An initial culture of the drainage in the external canal revealed light growth of Staphylococcus aureus, and a gram stain revealed no WBCs. Blood cultures × 4 demonstrated no growth. A CT scan showed an ill-defined, decreased attenuation of the left cerebellar hemisphere, with a slight mass effect on the fourth ventricle, bony destruction of the left sigmoid plate and decreased attenuation of the sigmoid sinus (Figs. 1 and 2). An MRI revealed a 3.5 × 3 × 2.5 cm left cerebellar abscess surrounded by a large zone of edema, mastoiditis, and lateral sinus thrombosis (Fig. 3).

The patient was placed on broad-spectrum antibiotics and underwent a left canal wall-down mastoidectomy, retrosigmoid drainage of the cerebellar abscess, and resection of the sigmoid and lateral sinus. An intraoperative gram stain revealed a few WBCs and no organisms. Fluid and tissue cultures both revealed Aspergillus fumigatus, and the patient was placed on a regimen of 30 mg amphotericin B daily. He was discharged on postoperative day 8 at his request and arrangements were made for a total of 2 gm amphotericin B to be administered by means of Hickmann catheter. The patient was lost to followup.

Case 2. A 65-year-old woman with a lifelong history of intermittent otorrhea was referred for evaluation of cholesteatoma of her right ear. She had a history of right tympanic membrane perforation since childhood and had noted continuous otorrhea for 4 months. The patient had mild fluctuating hearing loss, tinnitus, vertigo associated with quick head movements, and no otalgia. Physical examination revealed a right anterior tympanic membrane perforation and a posterior retraction pocket that extended into the hypotympanum. A cholesteatoma was noted around the stapes, extending into the attic. Tonal testing produced rotatory nystagmus in the headback and head-to-the-right hanging positions and an audiogram demonstrated a PTA air conduction of 75 dB and bone conduction at 30 dB on the right, and PTA air and bone conduction of 25 dB. Tuning forks suggested a right conductive hearing loss.

The patient was treated with boric acid powder for 1 month and underwent a right radical mastoidectomy and type IV sandwich graft tympanoplasty. Findings at surgery included an extensive cholesteatoma with complete destruction of the ossicular chain, including the posterior footplate, dehiscence of the pyramidal segment of the facial nerve and a fistula of the horizontal semicircular canal with exposed endosteum. All cholesteatoma was thoroughly removed, the fistula was covered with fascia before reconstruction, and erythromycin was given postoperatively for 7 days. The patient noted moderate dizziness and nausea 1 week postoperatively, and her cavity was clean. Two

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weeks later, necrotic tissue was noted in her mastoid cavity, she was placed on cortisporin otic suspension. Approximately 5 weeks postoperatively, the patient noted the onset of right facial weakness and less dizziness. Examination revealed mild weakness of all branches of the facial nerve with persistent necrotic tissue of the mastoid cavity. The paresis progressed to complete paralysis over the next week, despite treatment with steroids and topical antibiotics. Cultures of the mastoid bowl were obtained. The aerobic swab demonstrated *Aspergillus fumigatus*; however, tissue cultures revealed acute ulceration and inflammatory reaction and no evidence of fungus on special stains. A CT scan revealed some demineralization of the remaining bone. Conservative management was continued until 12 weeks postoperatively, at which time otalgia and right pinna perichondritis developed.

The woman was returned to surgery and underwent a right revision radical mastoidectomy, total facial nerve decompression, excision of 1.5 cm of necrotic pyramidal/vertical segment of facial nerve with greater auricular nerve reconstruction, and a temporalis muscle flap obliteration of the mastoid cavity.

Preoperative cultures demonstrated staphylococcus and no evidence of fungus. Intraoperative cultures revealed few colonies of *Aspergillus*. Tissue pathology showed necrosis, chronic inflammation with large aggregates of fungi with branching, septate hyphae that completely replaced the facial nerve, and demonstrated vascular invasion (Fig. 4). The patient was treated with amphotericin B (30 mg daily) for a total dose of 1.5 gm and had an uneventful recovery.

Immunologic workup, including CBC/manual differential, serum immunoglobulin levels with subclasses, total
hemolytic complement, and nitroblue tetrazolium, revealed no deficiencies. She regained facial nerve function (House grade II) 13 months postoperatively.

**DISCUSSION**

Aspergillus is a ubiquitous saprophyte that is found in soil, water, dust, and decaying matter. It is a filamentous fungus that is recognized by septate hyphae, with dichotomous branching at 45° angles. Aspergillus was first described as a human pathogen by Virchow (1844-56). The most commonly isolated species in Europe and North America is *Aspergillus fumigatus*. Other species that can be pathogenic to man include *Aspergillus flavus*, which is most common in the Middle East, *Aspergillus niger*, and *Aspergillus oryzae*.

The classification of aspergillus infections was initially described by Hora and modified by McGill et al. for paranasal sinus aspergillosis, but can easily be expanded to include temporal bone infections. Noninvasive aspergillosis is a localized infection without tissue invasion that responds to simple conservative removal. Invasive aspergillosis represents a destructive disease characterized by bony invasion, a granulomatous response, and marked fibrosis in patients without immune deficiency. Fulminant aspergillosis occurs in immunocompromised patients and represents an aggressive disease that demonstrates progressive tissue and angioinvasion with little tissue reaction and no granulomatous response.

Aspergillus infections of the head and neck are being reported with increasing frequency; the most common site is the paranasal sinuses. Invasive external otitis resulting from Aspergillus has been described within the last decade. All but one of the reported cases to date involved immunocompromised patients. In most of these cases the temporal bone involvement can be attributed to direct spread from other sources, such as the sinus or external auditory canal, or result from hematogenous spread. Other potential routes of invasion include tympanic and meningogenic.

Well-documented case reports of primary aspergillus mastoiditis resulting from a tympanic source have been rare. The clinical presentation of our patients was typical for chronic ear disease and included minimal otalgia, otorrhea not responsive to treatment, and hearing loss. Signs of intracranial involvement such as spiking temperatures, lethargy, and dull chronic headache were also noted. None of these signs or symptoms are suggestive of fungal infection. Both patients were noted to have facial nerve involvement, and case 2 had complete replacement of her facial nerve with inflammatory tissue containing fungal elements. Phillips et al. report that 100% of the four patients with documented invasive external otitis resulting from aspergillus had facial nerve involvement. Evidence of facial nerve involvement in patients with mastoiditis—particularly those who are immunocompromised and have not responded to antibiotics—should cause one to consider Aspergillus in the differential diagnosis.

The diagnosis of invasive or fulminant Aspergillus requires histopathologic confirmation on deep tissue biopsy or isolation from blood cultures. Superficial fungal otitis externa and saprophytic fungi in chronic otitis media are common, so that a single positive culture from the external canal or middle ear with a chronic perforation is meaningless. In case 2, initial cultures revealed aspergillus, but the tissue biopsy was negative, causing a delay in recognition and treatment.

Treatment of Aspergillus mastoiditis is threefold. Attempts should be made to control any underlying
immunologic conditions. Aggressive surgical debridement and resection is required, and antifungal chemotherapy should be instituted once the diagnosis is made. While amphotericin B is currently the most effective available agent, Itraconazole, a new oral triazole with fungicidal activity, has recently been reported effective against invasive Aspergillus.11

REFERENCES