Mycobacterium Chelonae Infection of the Parotid Gland

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ABSTRACT

Mycobacterium chelonae can cause numerous infections, including lung disease, local cutaneous disease, osteomyelitis, joint infections and ocular disease. With the exception of lung disease, these syndromes commonly develop after direct inoculation. The most common clinical presentation in immunocompetent individuals is skin and soft tissue infection. We present a case of *M. chelonae* infection of the parotid gland that was successfully treated with clarithromycin monotherapy. To our knowledge, this is the first case report of *M. chelonae* parotitis in an adult.

Key words: *Mycobacterium chelonae*, Parotitis, Immunocompetent

INTRODUCTION

Mycobacterium chelonae (*M. chelonae*), rapidly growing mycobacteria (RGM), primarily causehuman infection in immunosuppressed patients, including hematogenously disseminated disease. *M. chelonae* may also cause surgical wound infections and keratitis.[1] However, it rarely causes isolated lymphadenitis. Suppurative parotitis in an adult caused by *M. chelonae* has never been reported. Recently, we experienced a case of parotitis and parotid mass attributed to an *M. chelonae* infection.

CASE REPORT

We report a case of *M. chelonae*–associated parotitis in a 35-year-old woman. She presented with complaints of painful swelling and redness of the angle of her left jaw that started 1 month prior to admission. She reported that it started as a small 1-cm swelling in the middle aspect of her left jaw that gradually progressed to the angle. She was started on oral clindamycin and levaquin. There was no clinical improvement after a week of therapy. She started experiencing a pulsating pain that was aggravated by eating. A week prior to admission, she noticed progression of the redness and swelling to the left earlobe. She denied having any fever, chills, fatigue, weight loss or hearing impairment. She had a past medical history of neonatal portal vein thrombosis, splenectomy in 1992; and esophagogastric varices diagnosed by endoscopy two years ago. She was originally from Spain and had immigrated to the United States 15 years ago. She gave no personal or family history of tuberculosis. Her father had thyroid cancer. She did not have any history of recent travel. Physical examination [Figure 1] revealed a mobile and tender mass measuring approximately 3 cm and occupying the superficial lobe of the left parotid gland. There were overlying erythematous skin changes suggestive of parotitis. Facial nerve function was normal. Other physical findings were also normal, and there was no palpable cervical lymphadenopathy. The complete blood count, erythrocyte sedimentation rate, results of other biochemical/ autoimmune investigations and chest X-ray were normal. The result of the Human Immunodeficiency Virus (HIV) antibody test was negative. She had a tuberculin test done, which was measured at 4 mm (negative). CT scan [Figure 2] showed diffuse enhancement of the left parotid gland with an enlarged soft tissue mass lesion, measuring 3 × 4 cm in size, with a cystic component. A CT-guided fine-needle aspiration was performed, and the smear came back positive for acid-fast bacilli (AFB) [Figure 3]. The working diagnosis at that time was *M. tuberculosis*–related parotitis, and our patient was empirically treated with oral antitubercular meds (rifampin 300 mg, isoniazid 150 mg, ethambutol 400 mg

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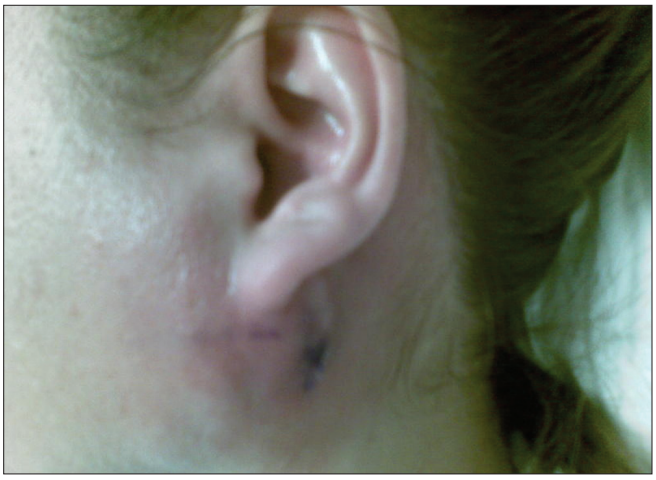

Figure 1: Erythema and fluctuance overlying the parotid gland area

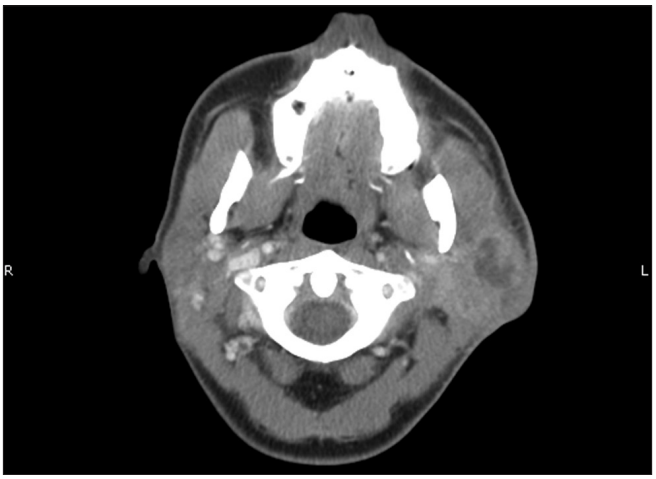

Figure 2: CT scan evidence of acute inflammation of the left parotid gland (acute suppurative parotitis)

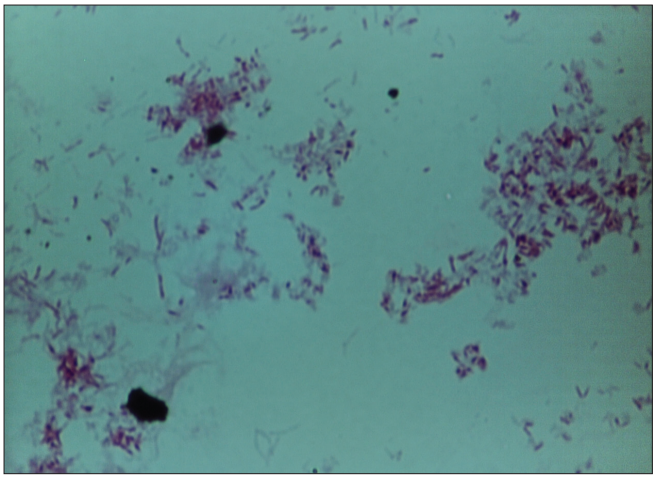

Figure 3: CT-guided aspirate of the left parotid gland revealing acid-fast bacilli organisms

and pyrazinamide 1000 mg once a day). Ten days later, the cultures grew AFB; and utilizing the genotype TB system (Hain, Nehren), the AFB was identified as *Mycobacterium chelonae*. Drug susceptibility report showed the organism to be resistant to imipenem, amikacin, ciprofloxacin, cefoxitin and linezolid; and sensitive to clarithromycin. The antitubercular regimen was discontinued, and she was started on oral clarithromycin 500 mg twice a day, with some evident resolution of symptoms, size and inflammation after 3 months of therapy. The management plan wasto continue this antibiotic regimen for at least six months and to regularly follow up with her during the duration of therapy.

DISCUSSION

Rapidly growing mycobacteria (RGM) are ubiquitous environmental organisms that usually take a week to grow in subculture. They have been isolated from soil, dust, water, terrestrial and aquatic animals, hospital environments and contaminated reagents and pharmaceuticals.[2] These RGM include three clinically relevant species: *M. fortuitum, M. chelonae and M. abscessus.* On review of the literature of RGM-associated infections, *M. fortuitum* was found to be most commonly associated with clinical disease.

M. chelonae can cause lung disease, local cutaneous disease, osteomyelitis, joint infections and ocular disease. With the exception of lung disease, these syndromes commonly develop after direct inoculation. The most common clinical presentation in immunocompetent individuals is skin and soft tissue infection. Disseminated disease with cutaneous manifestations has been linked to immunosuppression related to defects in cell-mediated immunity (e.g., HIV) or tothe Interleukin 12 / Interferon gamma pathway.[3]

Systemic findings of fever, chills and marked toxicity are not typical of RGM-related parotid gland infections, and chest radiographs are almost always normal. Some of these cases are associated with fistulae formation and/ or cervical lymphadenitis.

The histopathologic and microbiologic diagnoses of non-tuberculous Mycobacteria (NTM) parotitis are made by fine-needle aspiration of parotid gland material. CT scans and Magnetic Resonance Imaging (MRI) may help if they reveal subcutaneous necrotic, ring-enhancing lesions with typically minimal stranding of subcutaneous fat, which are suggestive of NTM infections.

NTM-related parotid gland infections are very rare; and to our knowledge, this is the first case report of *M. chelonae* parotitis in an adult, and there are no guidelines in the

medical literature specifically for this infectious entity. The diagnosis of this rare condition was made by fine-needle aspiration; and although the presence of *M. chelonae* may have been incidental rather than etiologic, the fact that the patient responded very well to oral clarithromycin antibiotic therapy supports the latter.

Lindeboom *et al.* showed that complete surgical excision of the gland had higher rates of complete regression when compared to medical antibiotic therapy alone (96% *vs.* 66%, respectively).[4] There are a few cases of NTM parotitis that were successfully treated with clarithromycin-based antibiotic regimen alone; or if necessary, along with drainage of an abscess.[5] When the diagnosis was made by fine-needle aspiration, we opted to treat the patient medically with antibiotics first because (1) we had established an etiologic agent and there is literature to support that these cases can be treated medically; and (2) our patient's concerns with having to go through a surgical resection of the parotid gland.

Most strains of *M. chelonae* have *in vitro* susceptibility to tobramycin, linezolid and tigecycline; whereas imipenem, doxycycline and ciprofloxacin are less effective. All strains of *M. chelonae* are susceptible to clarithromycin; therefore, it should be part of any antibiotic regimen for *M. chelonae* infections.

CONCLUSION

To our knowledge, this is the first case report of unilateral parotitis caused by *Mycobacterium chelonae* in an HIV-negative immunocompetent adult that was successfully treated solely with clarithromycin antibiotic therapy. We conclude that rare organisms from the rapidly growing mycobacteria family, like *M. chelonae,* should be included in the differential diagnosis for etiologic agents of suppurative parotitis.

REFERENCES

1. Chang MA, Jain S, Azar DT. Infections following laser *in situ* keratomileusis: An integration of the published literature. Surv Ophthalmol 2004;49:269-80.
2. Wolinsky E. Nontuberculous mycobacteria and associated diseases. Am Rev Respir Dis 1979;119:107-59.
3. Remus N, Reichenbach J, Picard C, Rietschel C, Wood P, Lammas D, *et al.* Impaired interferon gamma-mediated immunity and susceptibility to mycobacterial infection in childhood. Pediatr Res 2001;50:8-13.
4. Lindeboom JA, Kuijper EJ, Bruijnesteijn van Coppenraet ES, Lindeboom R, Prins JM, *et al.* Surgical excision versus antibiotic treatment for nontuberculous mycobacterial cervicofacial lymphadenitis in children: a multicenter, randomized, controlled trial. Clin Infect Dis 2007;44:1057-64
5. Green PA, von Reyn CF, Smith RP. Mycobacterium avium complex parotid lymphadenitis: Successful therapy with clarithromycin and ethambutol. Pediatr Infect Dis J 1993;12:615-7.

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