CASE STUDY

The Coccidioidomycosis Conundrum: A Rare Parotid Mass

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Introduction

Coccidioides—either of the species Coccidioides immitis or Coccidioides posadasii—is endemic to the southwestern US and desert regions of Mexico, Central America, and South America. Primary infections are often respiratory in nature, because Coccidioides infection is typically achieved via inhalation of arthroconidial spores. These infections are frequently asymptomatic and often go unrecognized. Extrapulmonary coccidioidomycosis is virtually always disseminated from a primary pulmonary infection, frequently becoming evident within weeks to 2 years after initial exposure. Risk of dissemination ranges from 0.2% to 4.7%, with the immunosuppressed and those of African or Filipino ancestry as the groups at highest risk for dissemination. The incidence of coccidioidomycosis continues to increase; primary coccidioidal pulmonary infection accounts for 17% to 29% of all cases of community-acquired pneumonia in endemic regions. Lesions are commonly known to hematogenously and/or lymphatically spread to skin and subcutaneous soft tissues, meninges of brain and spinal cord, and skeleton. Rarer and more atypical sites of dissemination have been documented. In the face and neck region, lesions to the eye and thyroid have been reported. Other atypical sites of dissemination to other regions of the body have included the adrenals, liver, peritoneal cavity, male genitourinary tract, and kidneys. To our knowledge, this is the second documented report of coccidioidomycoses of the parotid.

Abstract

A man, age 62 years, presented to the clinic with a 2-week history of increased nontender, nonerythematous, indurated right-sided parotid swelling. A 4 × 6-cm firm, well-circumscribed mass was palpated in the right parotid gland. A fine-needle aspiration biopsy was performed on the parotid mass with aspiration of 0.5 cc of purulent fluid with some blood. Cultures from the aspirate revealed Coccidioides immitis confirmed by DNA probe. Pathology slides revealed fungal spores. The patient was treated with 800 mg of fluconazole every day for 3 months with resolution of the parotid swelling. However, persistent cervical adenopathy remains.

Although this is a rare case of acute parotid swelling, Coccidioides immitis should be considered in the differential diagnosis of parotid masses in a patient with previous coccidioidomycosis. There may be a potential for an increase in frequency and variety of atypical extrapulmonary manifestations of coccidioidomycosis that parallels the increase in coccidioidomycotic pulmonary infections. Long-term antifungal therapy appears essential for control.

Observations

An African-American man, age 62 years, presented to the clinic with a 2-week history of increased nontender, nonerythematous, indurated right-sided parotid swelling (Figure 1a). The patient was initially given augmentin, but after histology, magnetic resonance imaging, and DNA probe confirmation, the treatment was switched to 800 mg fluconazole every day. At 3 months, parotid swelling was noted to be greatly improved.

Figure 1a. (left) Initial presentation. A man, age 62 years, presented to the clinic with a 2-week history of increased nontender, nonerythematous, indurated right-sided parotid swelling. A 4 × 6-cm firm, well-circumscribed mass was palpated in the right parotid gland.

Figure 1b. (right) After treatment. The patient was initially given augmentin, but after histology, magnetic resonance imaging, and DNA probe confirmation, the treatment was switched to 800 mg fluconazole every day. At 3 months, parotid swelling was noted to be greatly improved.

Figure 2. Coronal magnetic resonance imaging of the parotid mass. Multiplanar multisequence magnetic resonance images of the soft tissues of the neck were obtained using a 1.5-T scanner without and with Magnevist 20 mL intravenous gadolinium. A coronal T1-weighted sequence with fat saturation is seen here.
is an office worker and had a history of pulmonary coccidioidomycosis/valley fever several years ago, treated at another facility. During that time the patient had a complicated stay in an intensive care unit, where he required intubation with mechanical ventilation. After 18 months of fluconazole, his pulmonary symptoms resolved. At this visit, the patient denied pain, fever, chills, facial droop, or sicca symptoms. On examination, a 4 × 6-cm firm, immobile, well-circumscribed mass was palpated in the right parotid gland, without mucosal lesion or ulceration in the oral cavity. No pus was milked out of the Stensen duct.

Multiplanar multisequence magnetic resonance images of the soft tissues of the neck were obtained using a 1.5-T scanner without and with gadolinium. In both the coronal T2-weighted sequence (Figure 2) and axial T1-weighted sequence (Figure 3) with fat saturation after gadolinium, the right parotid gland was diffusely enlarged and edematous with an ill-defined peripherally enhancing 2.4 × 2 × 3-cm fluid collection in the superficial lobe, nonspecific at imaging. Associated matted-appearing lymphadenopathy extended posteriorly from the enlarged parotid gland involving predominantly level 1b and V.

No serology was obtained during the visit because a histopathologic diagnosis was seen as key. Fine-needle aspiration of the mass yielded less than 0.5 cc of fluid, showing minimally purulent material with admixed blood. Microscopy at high power with oil immersion (Figure 4) with characteristic histopathology is shown.

Microscopy revealed spherules consistent with *Coccidioides immitis* (≤ 70 µm in diameter), containing the classic endospores, with macrophages and polymorphonuclear neutrophils dominating the tissue reaction. Initially, the patient was given augmentin, but after histopathology, magnetic resonance imaging, and DNA probe confirmation of coccidioidomycosis, the treatment was soon switched to 800 mg fluconazole every day and the patient was followed-up closely. After 3 months, parotid swelling diminished (Figure 1b).

**Conclusions**

The differential diagnosis for unilateral parotid enlargement is vast—varying across such broad categories as infectious, neoplastic, or autoimmune. Infectious causes of parotid enlargement are best divided into suppurative versus nonsuppurative, with the former characterized by the clinician milking out suppurative fluid from the Stensen duct. Suppurative causes are almost always bacterial and are much more common; top pathogens involved in suppurative bacterial parotitis include *Staphylococcus aureus* and mixed oral aerobes and/or anaerobes. Nonbacterial causes of infectious parotitis are nonsuppurative and rarer. The following viral, mycobacterial, and fungal causes have been reported in prior literature. Viruses noted are human immunodeficiency virus, influenza, Coxsackievirus, Epstein-Barr virus, lymphocytic choriomeningitis virus, parainfluenza viruses, herpes simplex virus, and cytomegalovirus. Mycobacteria listed are *Mycobacterium tuberculosis* and *M. avium-intracellulare*. Fungal causes are *Candida albicans*, *Candida glabrata*, *Cryptococcus neoformans*, *Coccidioides immitis*, and *Histoplasma capsulatum*.

Because less than 50% of primary pulmonary coccidioidomycoses cases come to medical attention, and because of the indolent nature and nonspecific symptoms associated with extrapulmonary lesions, diagnosis of extrapulmonary coccidioidomycosis is often delayed by weeks or even months. Nonetheless, a detailed history, especially a history of
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Disclosure Statement
The author(s) have no conflicts of interest to disclose. This case was presented as a poster at the 2013 Combined Otolaryngology Spring Meeting for the Triological Society; April 10-14, 2013; Orlando, FL. Publication of this manuscript is not a result of this meeting presentation.

Acknowledgment
Mary Corrado, ELS, provided editorial assistance.

References

endemic exposure or prior coccidioidomycosis, and physical examination can generate enough clinical suspicion to initiate the appropriate further evaluation. Serologic antibody detection testing is an adequate means to diagnose the condition in ambulatory patients. If available, enzyme-linked immunoassays for IgM and IgG are highly sensitive screening tests and should be ordered before the more specific complement-fixing antibodies or immunodiffusion tests; the latter tests can also be used to monitor treatment response. Definitive cultures from clinical specimens, though the gold standard and useful for hospitalized inpatients, are often logistically difficult. In patients presenting similarly to this case with suitable masses, fine-needle aspiration biopsy can be a critical step in diagnosis and has been previously documented as an effective tool for diagnosis of extrapulmonary coccidioidomycosis.

On the basis of the 2005 Infectious Diseases Society of America published guidelines on coccidioidomycosis, oral azole antifungal agents are customary initial drugs of choice. Although ketoconazole has been approved for the treatment of coccidioidomycosis by the US Food and Drug Administration, studies have shown similar efficacy for use of fluconazole or itraconazole for chronic pulmonary and soft-tissue coccidioidal lesions. For patients worsening rapidly or whose lesions are located in vital areas like the spine, amphotericin B is recommended. There has been documented risk of relapse after discontinuation of therapy; however, most patients do not relapse, as cure rates approach 66%. In summary, in patients with parotid abscess, coccidioidomycosis of the parotid should be incorporated into the differential, most especially in those with typical geographic risks or known prior exposure. To our knowledge, this is only the second case report of a Coccidioides abscess of the parotid, with diagnosis affirmed by histopathology and imaging. Long-term antifungal therapy is essential for control.

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