Pleomorphic adenoma of the soft palate

PAUL LOMEO, DO, and JUDITH FINNEMAN, LPN-CST, Muskegon, Michigan

An 18-year-old female presented to our otolaryngology clinic with dysphagia and difficulty breathing due to a mass in her throat. She was seen initially 4 to 5 years ago at the clinic with a small mass in her soft palate. A biopsy was recommended. However, she refused and did not return. When she became symptomatic, she returned with her mother. Dysphagia and partial airway obstruction was present. The CT scan showed a mass in the soft palate. On physical examination a 3-cm mass of the soft palate was noted obstructing the nasopharynx and impinging on the oropharynx (Fig 1). The remainder of her head and neck examination was unremarkable. She was taken to the operating room where the mass was completely removed under general anesthesia. The frozen section diagnosis was a pleomorphic adenoma. The soft palate was reconstructed, and the patient spent a 23-hour observation period in the hospital after which she was discharged to home. The postoperative course was uneventful, and there was no evidence of velopharyngeal insufficiency. Her evaluation revealed the soft palate to be healing well, and all preoperative symptoms had resolved.

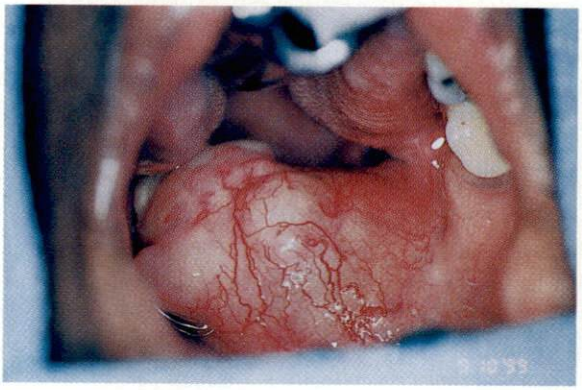

Fig 1. Pleomorphic adenoma of the soft palate involving the uvula.

DISCUSSION

Pleomorphic adenomas are benign tumors that mainly occur in the major salivary glands. They make up approximately 70% of all benign salivary gland tumors reported. They mainly occur in the parotid gland and are usually diagnosed on physical examination, image studies such as CT scans, or a fine-needle aspiration. The treatment of choice is complete removal of the pleomorphic adenoma with surrounding parotid gland. Benign pleomorphic adenomas do occur in minor salivary glands that are seen in the palate, upper lip, cheek, floor of the mouth, larynx, and trachea. Pleomorphic adenomas mainly occur in middle age, at approximately 40 to 60 years of age. Pleomorphic adenomas rarely occur in children, making up less than 2% of all benign salivary lesions reported.[1] Nevertheless, the treatment in the child population is the same as for adults with complete removal of the pleomorphic adenoma and surrounding tissue. The mass is surrounded by a capsule with small microscopic outgrowths known as pseudopodia that account for the recurrent rate of pleomorphic adenomas after resection. Histologically, these tumors are made up of a mixture of epithelial and myoepithelial cells with connective stroma. Pleomorphic adenomas are benign salivary masses that can be present for years. Such as in this case, this patient had a soft palate tumor since she was approximately 15 years of age. The mass continued to enlarge and became more symptomatic when she encountered adulthood. Even though this is a benign minor salivary gland tumor, it is still treated exactly the same as a major salivary gland tumor with a complete resection.

This section is made possible through an educational grant from Medtronic Xomed.

From Mercy General Health Partners, Muskegon, MI.
Reprint requests: Paul Lomeo, DO, Mercy General Health Partners, 1762 Wagner Ave, Muskegon, MI 49442; e-mail, shorent@gte.net.
Otolaryngol Head Neck Surg 2001;125:122.

0194-5998/2001/$35.00 + 0 **23/11/116784**
doi:10.1067/mhn.2001.116784

REFERENCES

1. Austin JR, Crockett BM. Pleomorphic adenoma of the palate in child. Head Neck 1992;14:58-61.
2. Gupta AK, Singhal SK, Mann SBS, et al. Pleomorphic adenoma presenting as a base of the tongue mass. J Laryngol Otol 1997;111:1177-8.
3. Lopez-Cedron JL, Gonzalez-Landa G, Birichmaga B. Pleomorphic adenoma of the palate in children: report of a case. Intranasally. J Oral Maxillofac Surg
4. Beunting J, Holmes DK. Giant pleomorphic adenoma of the parotid gland: case report and review of the literature. Ear Nose Throat J 1998;77:634-40.

by systemic symptoms that may last several weeks but usually resolves in immunocompetent patients without medical intervention. The elusive diagnosis may be achieved with the aid of a complement fixation test, tissue culture, special histologic stains, or examination of biopsy specimens, which may reveal characteristic granulomatous inflammation and spherules.[2]

Extrapulmonary or disseminated disease occurs in 0.5% of cases and typically involves the skin, lymph nodes, and bones, although it has been identified in every organ system in the body excluding the GI tract. Dissemination will most often occur within 1 year of the primary infection but may also develop many years later, primarily in association with impaired immunity. Any cause of T cell suppression—chemotherapy, corticosteroid therapy, thymectomy, or HIV infection—may lead to aggressive disease and extrapulmonary spread.[2] Organ transplant recipients on immunosuppressive drug regimens are at the highest risk for infection or reactivation within 1 year after the transplant.[3] It has been postulated that prophylactic antifungal therapy may be warranted in those transplant patients with potential exposure to or history of coccidioidomycosis. Severe disseminated coccidioidomycoses in this patient population may necessitate cessation of immunosuppression, accepting the likelihood of transplant failure.[3]

Those patients with severe pulmonary disease or dissemination should be managed with antifungal therapy. Amphotericin B has been the mainstay of therapy and the "gold standard" for treatment of *C. immitis* infection. The azole antifungals—fluconazole, itraconazole, and ketoconazole—are an attractive alternative because they are associated with less systemic toxicity and can be given orally. However, the efficacy of these medications for disseminated disease has yet to be proven.[2] The treatment duration for relapsing coccidioidomycosis is also unknown.

To our knowledge, this is the first published case of long-term relapse of a coccidioides infection of the larynx and the first published picture of laryngeal infection with this organism. A review of the literature revealed only 14 reported cases of coccidioidomycosis involving the larynx, none reactivated.[4,5] It remains unclear whether laryngeal coccidioidomycosis results from primary inoculation of the laryngeal mucosa or from hematologic dissemination.[4] In all of the reports that discussed initial presentation, symptoms of hoarseness, stridor, or dyspnea were present. Additional common symptoms included fever, malaise, cough, and dysphagia. Head and neck involvement with coccidioidomycosis may include skin lesions on the face or scalp, mucosal lesions in the nose or mouth, cervical lymphadenopathy, and soft tissue or deep neck space abscesses.[4] Of these 14 reported cases in the literature, 6 patients required airway intervention with either tracheotomy or intubation, 4 required no airway intervention, and 4 cases did not report airway management.[4,5] Although *C. immitis* infection is a seemingly rare cause of illness requiring evaluation by an otolaryngologist, it is important to remain cognizant of this disease, especially in immunocompromised patients from endemic areas.

REFERENCES

1. Hajare S, Rakusan TA, Kalia A, et al. Laryngeal coccidioidomycosis causing airway obstruction. Pediatr Infect Dis J 1989;8:54-5.
2. Stevens DA. Coccidioidomycosis. N Engl J Med 1995;332:1077-82.
3. Schroter GP, Bakshandeh K, Husberg BS, et al. Coccidioidomycosis and renal transplantation. Transplantation 1977;23:485-9.
4. Boyle JO, Coulthard SW, Mandel RM. Laryngeal involvement in disseminated Coccidioidomycosis. Arch Otolaryngol Head Neck Surg 1991;117:433-8.
5. Oliver, EA. Coccidioidomycosis with cutaneous involvement. Arch Dermatol 1954;70:537-8.