Laryngeal tuberculosis and stridor in a patient with AIDS

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Abstract

We present the first reported case of respiratory distress and stridor due laryngeal tuberculosis to occur in a patient with the Acquired Immunodeficiency Syndrome (AIDS). As such, larygeal tuberculosis should considered as a possible cause when an AIDS patient develops respiratory distress.

Key words: tuberculosis, laryngeal, acquired immunodeficiency syndrome.

Resumo

Referimos o primeiro caso da literatura de tiragem por tuberculose laríngea que surgiu num doente com SIDA. Como tal a tuberculose laríngea deve ser considerada como causa possível quando o doente desenvolve dificuldade respiratória.

Palavras chave: tuberculose laríngea, síndrome de imunodeficiência adquirida.

Introduction

Tuberculosis (TB) of the larynx is now considered a rare entity in industrialized countries. ^{1,2} It is so unusual that some otolaryngologists no longer include TB in the differential diagnosis of patients with complaints related to the larynx. ¹ Persons infected with the Human Immunodeficiency Virus (HIV) develop a deficiency in cell mediated immune defense that predisposes them to TB. Extrapulmonary sites of TB infection are common in these patients. ³ We recently cared for a patient with the Acquired Immunodeficiency Syndrome (AIDS) who developed stridor due to laryngeal TB, to our knowledge the first such case to appear in the literature.

Case Report

A 29 year old female with a history of intravenous drug abuse and AIDS-related complex presented with three weeks of fever, weight loss, progressive shortness of breath and "wheezing". On admission, the patient was in respiratory distress with a temperature of 38.3c. Her physical examination was significant for cervical adenopathy and laryngeal stridor. Pulmonary and cardiac examinations were normal. Initial laboratory values of significance were a white blood cell count of 3.3 x 10 /mm3, hemoglobin of 10.5 gm/dl, and normal serum chemistries. An arterial blood gas on 50% inspired oxygen via ventimask was pH 7.45, pC02 32mmHG, pO2 171mmHG, 99% saturation. The admission chest xray showed bands of atelectasis in both lung bases.

The patient was treated with aerosolized epinephrine, cefazolin 1 gram IV q8H, and dexamethasone 20mg IV (one dose only). Respiratory distress persisted and an emergency tracheostomy was performed on the first hospital day. During the procedure, a right sided subglottic mass was noted. Sputum cultures, blood cultures, and sputum for acid fast bacilli (AFB) were negative.

On the third hospital day a biopsy of the laryngeal mass was performed. The specimen consisted of necrotic tissue with areas of acute inflammatory infiltration by lymphocytes and plasma cells. No well formed granulomas or histiocytic giant cells were noted. Ziehl-Neilsen staining was positive for many acid fast bacilli. Cefazolin was discontinued and anti-TB treatment was started with isoniazid 300mg qD and rifampin 600mg qD. The patient became afebrile and, over the next five weeks, the tracheostomy site was gradually closed. Tissue cultures for AFB were negative. Anti-TB medications were continued for nine months without adverse effect. The patient remained stable and there was no clinical evidence of opportunistic infection or recurrent tuberculosis during 21 months of followup.

Discussion

Recent series of patients with laryngeal TB demonstrate that the average age of onset is in the 5th to 7th decades of life and that there is a male:female predominance of at least 2:1. More than 90% of patients present with several months of hoarseness and/or odynophagia. Although symptomatic stridor is considered uncommon, it has been reported in 5-20% of patients. Chest xrays vary but most commonly are consistent with advanced pulmonary TB. Response to treatment with anti-TB medications is excellent and only a rare patient requires surgical intervention. 1,2,4,5 TB of the larynx most often occurs as a consequence of active pulmonary TB; primary laryngeal tuberculosis is rare. Biopsy specimens usually show the classic findings of caseating granulomas with histiocytic giant cells. However, these findings may not always be present and the diagnosis must be based on the direct visualization of AFB in the tissue or positive tissue cultures for AFB.2

The patient we present is the first reported AIDS patient to develop respiratory distress and stridor due to

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laryngeal TB. As such, laryngeal TB should be considered in the differential diagnosis of an AIDS patient presenting with shortness of breath and/or stridor. Her symptoms and clinical course are somewhat unusual for laryngeal TB, no doubt because of concurrent HIV infection. Without a positive tissue culture result we cannot be sure of the causative species, but given our patient's improvement on a standard anti-TB regimen the species was most probably Mycobacterium tuberculosis.

Previous autopsy studies have demonstrated that 30% of patients in the general population with extensive pulmonary TB have biopsy evidence of laryngeal involvement. Because of the high prevalence of pulmonary TB among HIV positive individuals, laryngeal TB may therefore be an under-diagnosed condition in AIDS patients. This case demonstrates that further study is needed to determine whether laryngeal TB is a clinically significant entity in patients with AIDS.

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