



An Unusual Cause of Hoarseness of Voice in an Immunocompetent Individual

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A 43-year-old female patient presented with the complaints of progressive hoarseness of voice for 6 months. There was no apparent evidence of immunodeficiency, abuse of voice, systemic infection, or trauma. Fiberoptic laryngoscopy revealed whitish plaques mimicking leukoplakia that involved both the vocal cords. Investigations ruled out malignancy and confirmed primary vocal cord aspergillosis. The lesions responded dramatically to oral antifungal drugs. High index of suspicion and micropathological awareness regarding such an entity are of utmost importance since the management depends on accurate diagnosis and timely introduction of proper antifungal therapy.

Key words: Primary vocal cord aspergillosis, laryngeal aspergillosis, fiberoptic laryngoscopy

INTRODUCTION

Hoarseness of voice usually arouses a suspicion of upper respiratory tract infection including viral or bacterial laryngitis, vocal cord nodule, premalignant or malignant association, underlying systemic disease, or neuromuscular and psychiatric etiologies. Clinicians tend to overlook the diagnosis of fungal laryngitis in immunocompetent individuals. Aspergillosis is primarily a pulmonary infection with the involvement of other body sites such as paranasal sinuses and cutaneous tissues as well.¹ Secondary laryngeal involvement has been found in few cases of extensive bronchopulmonary aspergillosis, but primary laryngeal involvement is rare. In the absence of any immunodeficiency, such cases have been found to be associated with certain causes that lead to a breach of laryngeal epithelium such as voice abuse, laryngeal trauma, gastroesophageal reflux disease, and even oral sex.²⁻⁴ Rarely, the disease presents itself without any underlying cause or event.² We report this rare cause of hoarseness following primary laryngeal aspergillosis, i.e., without the involvement of other parts of the airway in an immunocompetent individual.

CASE REPORT

A 43-year-old homemaker presented with the complaints of progressive hoarseness of voice for 6 months at the ENT outdoor of a tertiary care hospital. On interrogation, she did not have tuberculosis, history of voice abuse and substance abuse, or laryngeal trauma. There was no history of any steroid or immunosuppressant use. History of recurrent bacterial, viral, or fungal infections was absent. Her general survey and systemic examination were noncontributory.

Indirect laryngoscopy (IDL) revealed whitish plaque-like lesion on the whole length of vocal cords with some irregularities on both margins. Vocal cord mobility was normal bilaterally, the airway was adequate, and both subglottis and supraglottis showed normal mucosa. IDL impression was that of a leukoplakic patch. Rest of the head and neck examination was unremarkable. She was euglycemic, and her complete hemogram was normal. Chest X-ray posteroanterior view and contrast-enhanced computed tomography (CT) thorax did not show any abnormality. X-ray of paranasal sinuses was also normal. Direct laryngoscopic biopsy was performed

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Primary vocal cord aspergillosis

under general anesthesia, and the tissue specimen was sent for histopathological and microbiological evaluation. Later, fiberoptic laryngoscopy was done to visualize postpunch biopsy residual patch [Figure 1].

Microscopic examination of hematoxylin and eosin stained smear from the tissue sample revealed fragments of hyperplastic squamous epithelium with areas of necrosis, ulceration, and dense inflammation. Septate fungal hyphae with a dichotomous branching pattern at acute angle were embedded within the exudates [Figure 2]. Microscopic examination of 10% potassium hydroxide preparation also revealed dichotomously branching, hyaline, septate hyphae. Fungal culture on Sabouraud dextrose agar showed growth of green, velvety, and powdery colonies highly suggestive of *Aspergillus fumigatus*. Microscopic examination with lactophenol cotton blue mount from the colony showed smooth conidiophores with uniseriate phialides on the upper part of vesicle, hence confirming the actual fungal etiology [Figure 3].

HIV serology was found to be nonreactive on two occasions. The patient's CD4 count (764 cells/mm³ as estimated by flow cytometry), CD4:CD8 ratio (1.6), serum IgG (850 mg/dl), IgA (130 mg/dl), IgM (150 mg/dl), and IgE (12.6 kU/L) all were within normal limits. In this background, the diagnosis of primary aspergillosis of the vocal cords was made, and she was advised oral itraconazole 100 mg twice a day for 2 months. Dramatic improvement following 1 month of therapy was noted. Further, 1-year follow-up did not reveal any recurrence.

DISCUSSION

Hoarseness of voice is a common presenting symptom of laryngeal leukoplakia. Occasional association of leukoplakia with malignancy necessitates laryngoscopic biopsy for its confirmation. Histopathological findings and fungal culture from the biopsy specimen were the key aspects in the confirmation of the present diagnosis.

This rarity of laryngeal aspergillosis emphasizes the natural resistance of larynx to colonization by fungi, including *Aspergillus*, despite the presence of spores in inhaled air.⁵ *Aspergillus* laryngitis is most commonly seen as part of a broader infection involving the respiratory system in immunocompromised people, but *Aspergillus* may rarely infect the larynx of healthy individuals.² Normal chest X-ray and CT-thorax ruled out pulmonary involvement in our case.

Immunocompetent status of our patient was supported by normal CD4 count, CD4:CD8 ratio, normal serum immunoglobulin levels, euglycemic status, repeated HIV seronegativity, and the absence of other opportunistic

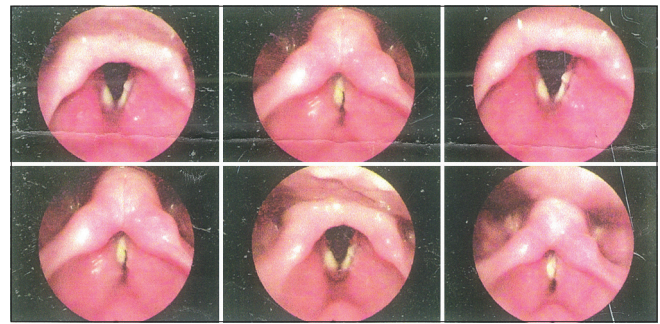


Figure 1: Postpunch biopsy residual patch visualized under fiberoptic laryngoscopy

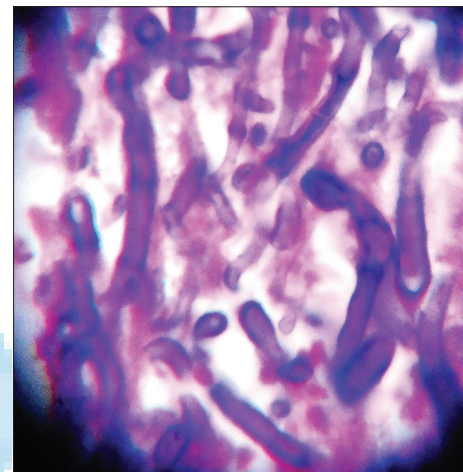


Figure 2: Photomicrograph of laryngeal tissue section showing septate fungal hyphae with a dichotomous branching pattern at acute angle, embedded within the exudates, (H and E, $\times 1500$)

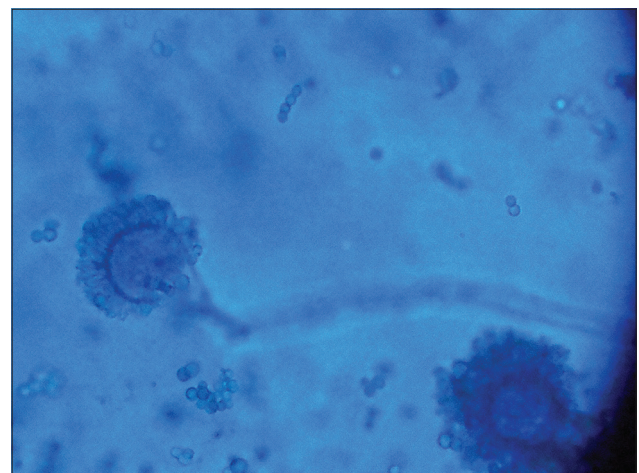


Figure 3: Photomicrograph showing smooth conidiophores with uniseriate phialides on the upper part of vesicle, (lactophenol cotton blue mount, $\times 600$)

infections. The upper airway epithelium acts as a physical barrier further supported by mucous lining and protects against colonization or infection by potential pathogens. Thus,

any factor that alters the mucosal barrier may predispose to laryngeal mycosis even in the absence of immunodeficiency. In immunocompetent individuals, predisposing factors are the factors altering mucosal barrier which include previous radiotherapy, antibiotic therapy with changes to local flora, inhaled corticosteroids, gastroesophageal reflux disorder or trauma (e.g., intubation), and smoking.⁴ We did not find any of these predisposing factors in our patient.

No satisfactory epidemiological study is available regarding the overall incidence of such conditions. In healthy patients, debridement alone or voice resting, stopping antibacterial antibiotics, and applying antifungal agents have been considered important in the treatment of aspergillosis in the vocal cord.⁵ We also found a very good response with oral antifungal therapy.

Primary laryngeal location of the lesion, local tissue invasion, no systemic involvement, immunocompetent state, no underlying event which may cause a breach in laryngeal epithelial integrity, and a good response to antifungal therapy are the notable features in our case. The possibility of primary laryngeal aspergillosis should be kept in mind while dealing with nonhealing and chronic laryngeal leukoplakic lesions presenting with hoarseness, even in patients with a good immune status.

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Conflicts of interest

There are no conflicts of interest.

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