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UNRAVELING THE ENIGMA : PALATAL HISTOPLASMOSIS

Oral Pathology

Latha Mary Cherian Professor and HOD, Department of Oral Pathology and Microbiology, Government Dental College Kottayam

Vally M* Junior resident, Department of Oral Pathology and Microbiology, Government Dental College Kottayam *Corresponding Author

Hafeefa M Junior resident Department of Oral Pathology and Microbiology, Government Dental College Kottayam

Sudharani Junior resident, Department of OralPathology and Microbiology, Government Dental College Kottayam

Aishwarya Menon P Junior resident Department of OralPathology and Microbiology, Government Dental College Kottayam

ABSTRACT

Histoplasmosis is a systemic mycotic infection caused by the dimorphic fungus Histoplasma capsulatum. Oral lesions of histoplasmosis are generally associated with the disseminated form of the disease and can manifest as a fungating or ulcerative lesion of the oral mucosa. There have only been a few documented occurrences of histoplasmosis in India. In this article, we are reporting a case of palatal histoplasmosis in an HIV seronegative patient from a non-endemic region of India.

KEYWORDS

Fungal infection, Histoplasmosis, oral cavity.

INTRODUCTION

Histoplasmosis is a deep fungal infection caused by Histoplasma capsulatum. The first reported case of histoplasmosis in a human was reported by Samuel Darling in Panama in 1906.[1] Hence, the disease is also known as Darling's disease.

Histoplasmosis may occur in three forms: (i) the primary acute pulmonary form; (ii) the chronic pulmonary form; and (iii) the disseminated form.[2,]

Histoplasmosis is endemic in the central and eastern United States, especially the Ohio and Mississippi River Valleys, in Central and South America, and Africa, but is less frequently reported in Asia and Europe. Nonetheless, reports of the illness are also often coming from nonendemic regions, particularly from those with impaired immune systems.[5,6] Histoplasmosis in Indians tends to more commonly affect extrapulmonary sites, particularly the oral cavity. Reported cases of histoplasmosis have been low in India. In this article, we are reporting a case of palatal histoplasmosis in an HIV seronegative patient from a non-endemic region of India.

Case Report

A 65-year-old male presented to the outpatient department with a complaint of ulcers in the palatal area,pain and a burning sensation while swallowing since 3 months. Ulcer in the palatal region was insidious in onset; the size of the ulcer increased gradually; it was associated with pain and burning sensation while swallowing for a few weeks; it was not associated with any trauma, pus discharge, or bleeding. The patient has had a history of intermittent productive coughs for 1 month. There was no history of chest pain, dyspnea, wheeze and hemoptysis.

The patient was a known case of diabetes mellitus and is currently under medication for the same. There was no history of hypertension, tuberculosis, jaundice, epilepsy, or skin lesions.

Oral examination revealed an ulcerated area of size 2.5 x 1 cm noted over the junction of hard palate and soft palate, irregular in shape, and the margins of the ulcer appeared to be well demarcated from the normal mucosa (Figure 1). Part of the ulcer was covered with grayish white slough; the borders appeared punched out; and no bleeding or pus discharge was noted.

The ulcer was painful to palpate. The margin,edge and base of the ulcer were non-indurated. Right submandibular lymphadenopathy was noted. On extraoral examination, no relevant findings were detected.

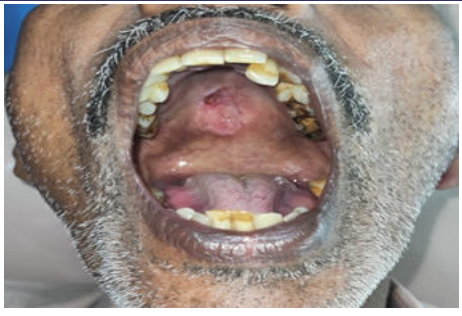

Figure1: Intraoral photograph of patient showing solitary ulcerated area over the junction of hard palate and soft palate

By correlating clinical findings with patient history, we came to the provisional differential diagnosis of chronic non-healing ulcers,diabetic ulcers,mucosal malignancies, and salivary gland malignancies.

As the patient was diabetic, fasting blood sugar was 173 mg/dl, and postprandial blood sugar was 245 mg/dl. Serological tests like HbsAg,HCV, and HIV tests were negative.

Microscopy of the H&E-stained soft tissue bit revealed a diffuse collection of numerous yeast-like tiny microorganisms of size less than 1 mm within the moderately collagenous connective tissue stroma (Figure 2) Organisms were seen infiltrating into mucous salivary gland aciniin certain areas. Gomori'sMethanimine Silver (GMS) special staining revealed dark, brown, and black-stained small yeast-like microorganisms of size less than 1mm in the epithelium and connective tissue (Figure 3)

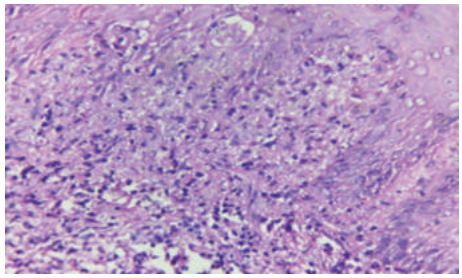

Figure 2; Photomicrograph shows diffuse collection of numerous

yeast –like tiny microorganisms of size less than 1 µm (H & E Stain, x 400)

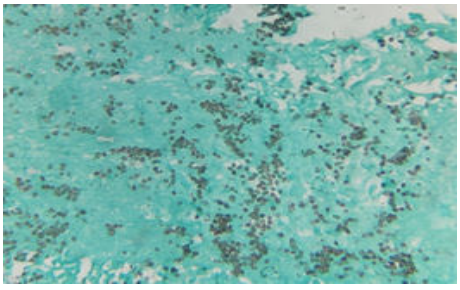

Figure 3: Photomicrograph shows diffuse collection of numerous dark, brown, and black-stained small yeast-like microorganisms of size less than 1mm (GMS Stain, x 400)

Depending upon microscopic findings, we came to the diagnosis of palatal histoplasmosis.

The patient was referred to the Department of Infectious Diseases, MCH Kottayam. The patient was initially treated with injection amphotericin B 150 mg OD for 2 weeks, and the patient has been advised to take tab oral itraconazole 200 mg TID for 1 year.The patient was put under follow up since then. After a period of 8 months from the start of treatment, the palatal ulcer showed an uneventful healing

DISCUSSION

Histoplasmosis is a rare opportunistic infection in India, caused by Histoplasma capsulatum. Histoplasma capsulatum is a dimorphic fungus, growing as a mold in its natural environment and as a yeast at body temperature in the human host. The fungi known to cause human infections include H. capsulatum var. capsulatum and H. capsulatum var. duboisii.[7] Africa is home to the equine pathogen H. capsulatum var. farciminosum, which is the third variant and causes infection. Histoplasmosis is endemic in Africa, Central and South America, the Ohio and Mississippi River Valleys of the United States, and Asia; reports from Asia and Europe are less common. Nonetheless, reports of the disease are also being made regularly from nonendemic regions, particularly from immunocompromised people and returning tourists.[5,6]

The fungi usually live in warm, humid habitats that are also home to bats and bird droppings.[8] Histoplasmosis has historically been linked to carpenters, agricultural laborers, engagement in outdoor activities, such as chicken runs, collecting bird or bat guano, and living in bat-infested homes.[9,10] H. capsulatum can be acquired by inhaling the fungus's mycelial fragments. Histoplasmosis is mostly a lung pathogen, although in immunocompromised individuals, it can spread to extrapulmonary organs. Lesions manifest as necrotizing granulomas and diseases resembling tuberculosis in imm unocompetent patients. The most frequently affected areas are the lungs (single nodules might mimic neoplasms), skin (resembles Molluscum contagiosum), bone marrow, mediastinal lymph nodes (can mimic lymphoma), and tongue (ulcerative lesion with heaping borders).[11]

Oral manifestations of the disease sporadically appear.[12] They sometimes appear as a localized lesion, which may be the first, or they occur in conjunction with the disseminated type.

According to Padhye et al., oral cavity infections are the main extrapulmonary sites where histoplasmosis likely to arise in Indians.[8] The areas where oral histoplasmosis occurs most frequently are the palate, tongue, buccal mucosa, gingiva, and pharynx. Goodwin et al. state that oropharyngeal lesions, particularly when the disease is disseminated, are often the first signs of the illness. It is suggested that patients with localized oral histoplasmosis should have regular evaluations for any systemic involvement. Reddy et al. (1970) observed that all of the patients who originally presented with mouth lesions later developed disseminated disease.[8]

Oral lesions can appear as nodules, papular, ulcerative, furunculoid, granulomatous, or plaque-like lesions; however, a superficial or deep infiltrating ulceration with a pseudomembrane is the most typical presentation. A biopsy of the lesion may be necessary if the clinical presentation resembles that of a cancer. A lesion of oral histoplasmosis has a wide clinical differential diagnosis. Benign entities should be taken into consideration, ranging from non-specific ulcers to malignancy. The differential diagnosis for the current patient included chronic non-healing ulcers,diabetic ulcers,mucosal malignancies, and salivary gland malignancies.

Since the detection of histoplasmic organisms in the section offers definitive proof of the disease, histopathology is the primary investigative modality.[13] The presence of microscopic 2-4 µm spores in the cytoplasm of macrophages and occasionally in large cells is the hallmark of histoplasmosis as identified by histopathology. Hematoxylin and eosin sections stained with periodic acid Schiff (PAS) or Giemsa stain (GMS) are used to visualize the spores of H. capsulatum. The spores are shaped as ovoid or spherical entities with a distinct space surrounding them. This initial appearance led to the idea that the spores were capsules, hence the name H. capsulatum.

Leishmaniasis, Penicillium marneffei, Cryptococci neoformans, and Candida glabrata infections are included in the histopathologic differential diagnosis of histoplasmosis.[14] The lack of a distinct halo surrounding spores distinguishes leishmaniasis from histoplasmosis. Penicillium marneffei can be identified by its septate yeast forms, which reproduce by binary fission while H. capsulatum divides via budding.[15] H. capsulatum is roughly 2-4 µm in diameter, whereas cryptococci are enclosed, spherical to oval yeasts that measure 5-10 µm. Compared to histoplasmosis, Candida glabrata may exhibit greater size variability.

However, the culture of the pathogen on SDA(Sebaroud's dextrose agar) will be confirmatory.

Amphotericin B is still the drug of choice for histoplasmosis.[16] For patients who cannot tolerate amphotericin B, itraconazole is an effective and alternative therapy. In the present case, the patient was initially treated with injection amphotericin B 150 mg OD for 2 weeks and has been advised to take oral itraconazole 200 mg TID for 1 year.

CONCLUSION

Histoplasmosis is an underdiagnosed infection that affects both seemingly healthy and immunocompromised individuals. The disseminated form of histoplasmosis is typically linked to oral lesions, which can manifest as ulcerative or fungal lesions of the oral mucosa. In order to diagnose and prevent the disease's spread, which would otherwise lead to its fulmination, a comprehensive clinical understanding of oral histoplasmosis is crucial.

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