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Case Report

Liver Cirrhosis–Associated Maxillary Mucormycosis: A Rare Case Report

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ABSTRACT

Mucormycosis is a rapidly progressive and life-threatening opportunistic fungal infection that predominantly affects immunocompromised individuals, most commonly those with uncontrolled diabetes mellitus. However, its occurrence in patients with liver cirrhosis is rare and inadequately documented in the literature. This report presents a case of a 35-year-old male with liver cirrhosis who developed maxillary mucormycosis characterized by sudden tooth exfoliation, palatal necrosis, and oroantral communication. Radiographic findings revealed extensive sinus involvement with bony destruction, and histopathological examination confirmed invasive fungal infection. This case highlights liver cirrhosis as an important but often overlooked predisposing factor and contributes to the limited documentation of such rare presentations. Early recognition of oral manifestations is crucial for timely diagnosis and management.

Introduction

Mucormycosis is an aggressive angioinvasive fungal infection caused by fungi belonging to the order Mucorales, including Rhizopus, Mucor, and Lichtheimia species[1]. The disease is characterized by rapid progression, vascular invasion, thrombosis, and extensive tissue necrosis[2].The infection primarily affects immunocompromised individuals, particularly those with uncontrolled diabetes mellitus, hematological malignancies, organ transplantation, or prolonged corticosteroid therapy[3]. In recent years, the incidence has increased significantly, especially in association with COVID-19 and indiscriminate steroid use[4].Although liver cirrhosis is not a commonly reported risk factor, it is increasingly recognized as a condition associated with immune dysfunction. Cirrhosis-associated immune dysfunction syndrome leads to impaired neutrophil function, reduced complement activity, and altered cytokine responses, thereby increasing susceptibility to opportunistic infections[5]. Additionally, elevated serum iron levels and malnutrition further predispose these patients to invasive fungal infections[6].Rhinocerebral mucormycosis is the most common form and frequently involves the maxilla due to its proximity to the paranasal sinuses[7]. Oral manifestations such as sudden tooth mobility, non-healing extraction sockets, palatal ulceration, and necrosis may represent early signs of the disease[8].

Despite increasing awareness, cases of mucormycosis associated with liver cirrhosis remain sparsely documented. The present report contributes to the

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existing literature by documenting a rare case of maxillary mucormycosis in a cirrhotic patient.

Case Report

A 35-year-old male patient presented with complaints of missing teeth in the upper front and back regions of the jaw for the past one month. The patient reported that he was apparently healthy prior to the onset of symptoms, after which he experienced sudden mobility of the maxillary teeth followed by spontaneous exfoliation. He also complained of foul odor and watery nasal discharge. There was no history of fever, pus discharge, trauma, or significant weight loss.

The patient's medical history revealed liver cirrhosis diagnosed four months earlier, for which he was undergoing medical management including corticosteroid therapy. He also had a history of cerebral infarcts and chronic alcohol consumption. These factors contributed to a compromised immune status.

On extraoral examination, face appeared symmetrical. Lips were competent. No signs of pallor, icterus, cyanosis, clubbing, or lymphadenopathy were noted.

A single, diffuse swelling was observed over the right middle third of the face, measuring approximately 4 × 5 cm. The swelling extended superiorly about 2 cm above the ala-tragus line, inferiorly up to the level of the right corner of the mouth, anteriorly up to the ala of the nose, and posteriorly up to the outer canthus of the eye.

The overlying skin appeared normal with no evidence of erythema, ulceration, or sinus formation. On palpation, the swelling was non-tender and soft to firm in consistency, with no local rise in temperature. No extraoral draining sinus was noted.

Temporomandibular joint examination revealed no abnormality. Mouth opening was within normal range.

Intraoral examination revealed multiple missing teeth in relation to 11–17 and 24–27 regions. A denuded, exposed necrotic yellowish bone was present in the mid-palatal region measuring approximately 1.5 cm in

Figure 1: Extraoral swelling on the right midface region.

Figure 2: Intraoral view showing exposed necrotic palatal bone and perforation.

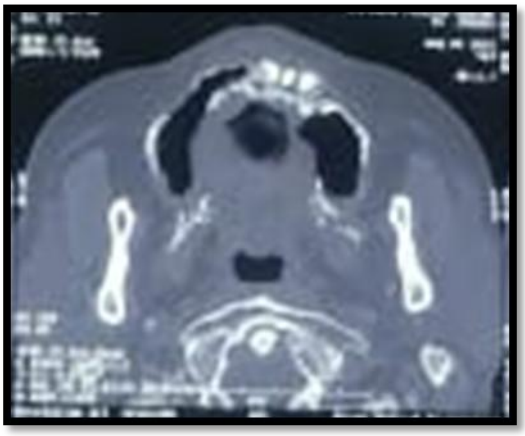

Figure 3: CT scan(Axial and 3D reconstruction) showing sinus involvement and maxillary bone destruction.

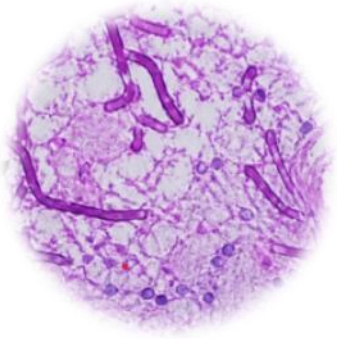

Figure 4: Histopathological section showing broad aseptate fungal hyphae with right-angle branching and angioinvasion.

diameter. The surrounding mucosa appeared normal with no significant inflammatory changes.

Bilateral palatal destruction was evident along with oroantral communication in the maxillary alveolar ridge. On palpation, the exposed bone was non-tender and rough in texture. Posterior mobility of the maxilla was noted, while teeth 21, 22, and 23 did not exhibit mobility.

Computed tomography imaging demonstrated mucoperiosteal thickening involving the maxillary, ethmoid, frontal, and sphenoid sinuses, along with erosion of the maxillary sinus floor extending into the alveolar bone and palate, suggestive of invasive fungal sinusitis[10].

An incisional biopsy was performed from the necrotic palatal region. Histopathological examination revealed extensive necrosis with broad, ribbon-like, aseptate fungal hyphae showing irregular right-angle branching. Evidence of angioinvasion and vascular thrombosis was observed. Periodic acid–Schiff (PAS) and Grocott's methenamine silver (GMS) staining highlighted the fungal elements clearly. These findings were consistent with mucormycosis[9].

Based on clinical, radiological, and histopathological findings, a final diagnosis of rhinocerebral mucormycosis involving the maxilla associated with liver cirrhosis was established. The patient was advised immediate antifungal therapy with liposomal amphotericin B along with aggressive surgical debridement and multidisciplinary management.

Discussion

Mucormycosis is a fulminant fungal infection associated with high mortality, particularly when diagnosis is delayed[1]. Although diabetes mellitus remains the most common risk factor, liver cirrhosis is an underreported but important predisposing condition.

Cirrhosis-associated immune dysfunction significantly impairs host defense mechanisms, including neutrophil dysfunction, complement deficiency, and altered cytokine response[5]. Additionally, increased free iron levels in cirrhotic patients enhance fungal proliferation[6].

The pathogenesis involves angioinvasion by fungal hyphae, leading to vascular thrombosis, ischemia, and necrosis[2]. In the present case, this resulted in maxillary bone necrosis, spontaneous tooth exfoliation, and palatal perforation, which are characteristic features of advanced disease[8].

Radiological imaging plays a crucial role in determining disease extent. CT imaging is useful for detecting bony destruction, whereas MRI is superior for soft tissue spread[10].

Histopathological examination remains the gold standard for diagnosis and confirmed mucormycosis in this case by demonstrating broad aseptate hyphae with angioinvasion[9].

Table 1: Comparison with Reported Cases

Author (Year)	Predisposing Factor	Site	Oral Findings	Outcome
Roden et al.[1]	Mixed	Rhinocerebral	Palatal necrosis	High mortality
Albillos et al.[5]	Cirrhosis	Systemic	Not specified	Increased risk
Therakathu et al.[10]	Immunocompromised	Sinuses	Bone destruction	Variable
Present case	**Cirrhosis + steroids**	**Maxilla**	**Tooth exfoliation, palatal necrosis, OAC**	**Under treatment**

This highlights the rarity of oral manifestations as initial presentation in cirrhotic patients.

Conclusion

This case highlights a rare presentation of mucormycosis in a patient with liver cirrhosis, emphasizing the importance of recognizing oral manifestations as early diagnostic indicators. Given the limited documentation of such cases, this report contributes to the existing literature and underscores the need for heightened clinical suspicion in cirrhotic patients presenting with maxillary necrosis or unexplained tooth mobility. Early intervention is critical to improving survival outcomes.

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