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**Case Report**

# Juvenile post dengue mucormycosis - reporting a rare entity with review of literature

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## ABSTRACT

Invasive mucormycosis is a life-threatening fungal infection that most frequently occurs in patients with underlying comorbidities that impacts immune system function. It is an opportunistic and frequently fulminant fungal infection caused by a saprophytic organism belonging to class zygomycetes. These organisms are omnipresent and grow in their natural state on the variety of decaying organic materials. These organisms are present in the nasal passages and oral cavities of normal individuals. Here, we report a rare case of juvenile post dengue mucormycosis along with brief insight into the review of literature. Histopathological examination of the necrotic bone and overlying tissue was done in OPD in a juvenile patient who reported with purulent discharge and having a history of dengue. There were abundant PAS positive fungal hyphae. This case report will acquaint the oral care providers of a possible association of mucormycosis and dengue and highlight the importance of early diagnosis to optimize the treatment outcomes and prognosis in such cases. Mucormycosis is a rare opportunistic infection that spreads promptly and is life-threatening. Therefore, early diagnosis and prompt treatment is required to reduce the mortality and morbidity of this lethal infection.

**Keywords:** Mucormycosis, Fungal infection, Dengue, Opportunistic infection

## INTRODUCTION

Mucormycosis also referred to as zygomycosis or phycomycosis, is a rare opportunistic fungal infection caused by the oral mucorales.[1,2] These fungi are ubiquitous and the spores are found in soil, dust, manure, fruits and in decaying matter.[2,3] Humans get infected with these fungi through inhalation of spores, contaminated food ingestion or inoculation of disrupted skin or wounds.[3-5] It is among common angio-invasive fungal infections like candidiasis and aspergillosis.[6]

Previous literature reveals various predisposing factors including debilitating conditions like uncontrolled diabetes, malignancies (lymphomas and leukemias), renal failure, organ transplant, long-term corticosteroid and immunosuppressive therapy, cirrhosis, burns, protein-energy malnutrition, severe and prolonged neutropenia, deficient T cell immunity, immaturity and low birth weight and acquired immuno-deficiency syndrome (AIDS).[7,8]

Majority of cases are seen in older adults and occurrence in juvenile patients in absence of underlying debilitating chronic conditions is extremely rare.

Although quite rare, mucormycosis can also occur as an opportunistic infection. Dengue is one such infection that may result in compromised immune status thereby subsequently resulting in mucormycosis. The chief causative agent for dengue is virus dengue virus serotypes (DENVs 1–4).[9] Similar to other viral infections, the treatment for dengue is usually symptomatic. Opportunistic infections can occur in patients suffering from dengue due to associated comorbidities and immune-compromised status.

In this case report, we present a case of zygomycosis affecting the mandible in a juvenile patient who had recovered from dengue.

## CASE REPORT

A 14-year-old male patient reported to the outpatient department with chief complaint of pain and fluid discharge on the right side of his jaw since one month. A month back the patient was apparently asymptomatic when he developed pain in the lower right posterior tooth region. Extraction of the permanent mandibular right molar was done about one week back but patient did not get relief. The extraction socket remained unhealed and subsequently developed a purulent discharge from the same area (Figure 1).

Patient was an agricultural labor of lower socio-economic group and malnourished. There was no significant past medical history of any chronic disease and negative history of COVID-19 infection. Recent medical history revealed high-grade fever diagnosed as dengue for which the patient was hospitalized. Blood investigations during that period revealed anemia (Hb 6.9 g/dl), erythropenia ($2.64\times10^{12}$/l), mild leucocytosis ($11.4\times10^{9}$/l) with relative neutrophilia (90% of leucocytes), and thrombopenia ($42\times10^{9}$/l) for which the patient had undergone blood transfusion. The dental pain started about 1 week after discharge from the hospital and eventually for which the permanent right second molar tooth was extracted.

On intraoral examination the mandibular right second premolar and permanent right second molar were missing and the permanent mandibular right first molar was mobile. The mucosa over the extraction wound was denuded with exposed dull white color of bone extending from to distal aspect of mandibular right first molar extending up to retro-molar area. On palpation, the affected area was rough in texture with mild tenderness. Hematological parameters at this time showed relative improvement from the time of hospitalization (Hb 9.4 gm/dl, erythrocyte count-$3.48\times10^{12}$/l, leucocyte count-$9.75\times10^{9}$/l, and platelet count-$454\times10^{9}$/l. Blood sugar levels were within normal range, all viral markers and QuantiFERON TB gold assay for tuberculosis were negative.

Orthopantomogram was taken, and it showed incompletely healed socket of the mandibular right second molar but no other significant radiological findings (Figure 2).

**Figure 1: Intra oral photograph showing necrosis of the right mandibular bone.**

**Figure 2: Orthopantomogram showing incompletely healed socket of the mandibular right second molar.**

Based on the clinical and radiological findings a provisional diagnosis of suppurative osteomyelitis was considered. Patient was prescribed antibiotics and surgical debridement was planned. The mandibular right first molar was extracted and a deep curettage of the alveolar socket was done. The curetted soft tissue along with necrosed bone was sent for histopathological examination.

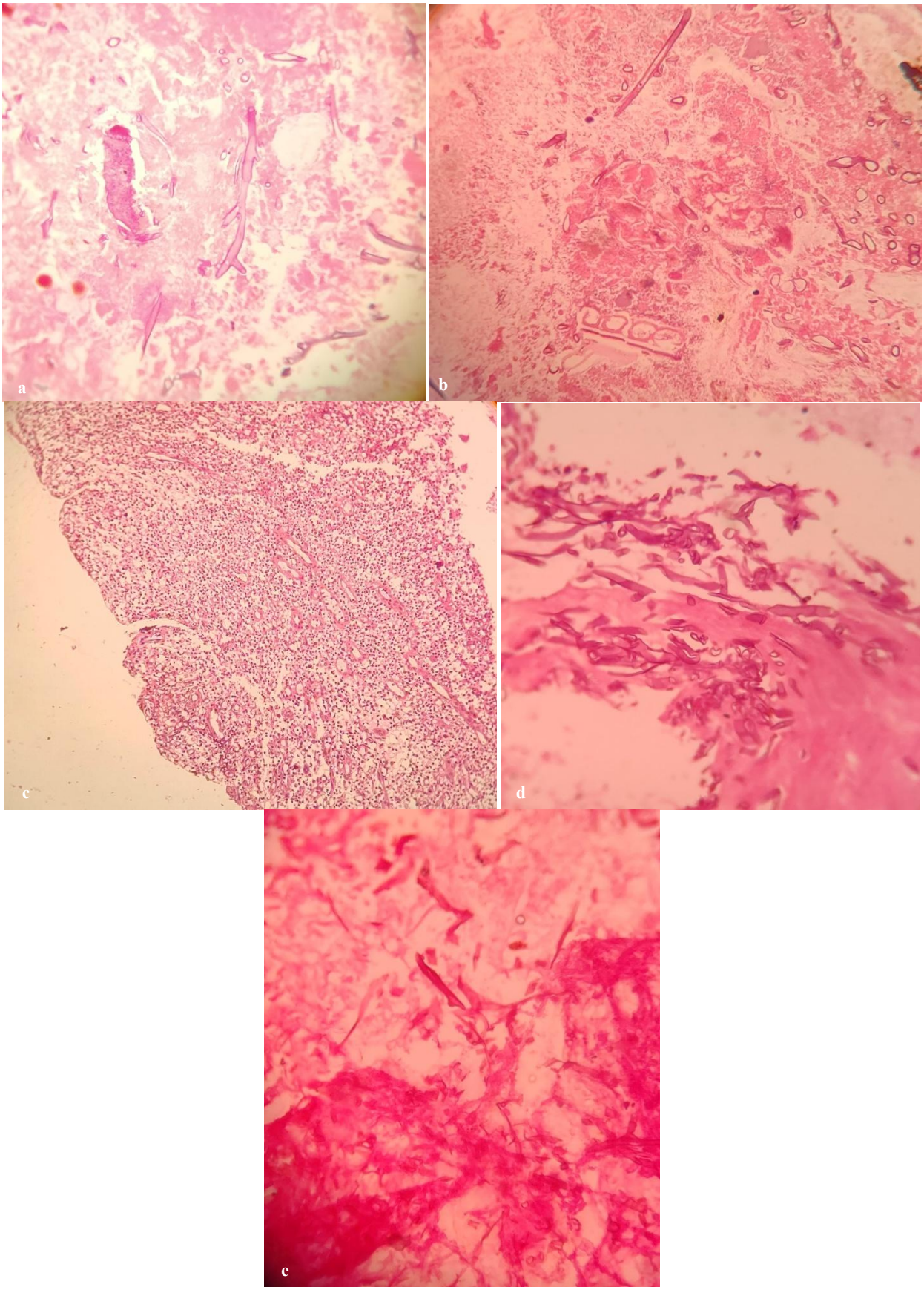


**Figure 3: (a, b) Hematoxylin and eosin section showing fungal hyphae branching at right angle, (c) Hematoxylin and Eosin section showing cellular debris along with dense mixed inflammatory cell infiltrate, and (d and e) periodic acid–Schiff (PAS) stained section showing magenta pink-colored aseptate fungal hyphae.**

The microscopic examination revealed spicules of necrotic bone surrounded by moderately collagenous, acellular, and avascular connective tissue exhibiting dense mixed inflammatory infiltrate. Interspersed within the tissue were large, broad, aseptate PAS positive fungal hyphae. These fungal hyphae showed branching in different patterns, majority of which were forming a right angle characteristic for mucormycetes (Figure 3c). In focal areas, these hyphae were seen invading into blood vessels (Figure 3d). PAS stained sections revealed aseptate PAS positive fungal hyphae (Figure 3e and f). Based on histopathological findings, a final diagnosis of zygomycosis/mucormycosis was made. The patient was prescribed antifungal therapy.

## DISCUSSION

Mucormycosis was discovered and described in humans by German pathologist Paltauf and the term 'mucormycosis' was coined by Baker.[9-11]

Mucor is often referred to as 'Black fungus' owing to clinical appearance of the necrosed tissue. However, the actual black fungus is the pigmented fungus (Dematecious fungi) exhibiting black/brown coloration of the tissue due to presence of melanin pigmentation.[12]

Fungi of the order mucorales have unique features that distinguish them from other clinically relevant fungi. The hyphae of mucorales are known as coenocytic hyphae as they are broad, ribbon-like multinucleated cells with absent or rare septa. The hyphae develop from the germinal tube by apical extension, and during tissue invasion may occasionally form septa to delimit reproductive structures or swollen areas.[13]

They produce unicellular asexual spores known as sporangiospores endogenously without the involvement of pre-existing cell walls.[14] These spores are found ubiquitously in soil, dust, manure, fruits and decaying matter.[15] The spores enter into the organism through inhalation or consumption of contaminated food. It may also get inoculated via contaminated needles or catheters into the cutaneous tissue, or due to implantation of injured skin following trauma.[12]

These tiny spores get settled upon the oral and nasal mucosa in humans.[16] In majority of immunologically competent hosts, these spores are unable to develop further due to host phagocytic response.

However, in compromised patients having low immunity, this fails leading to germination and thereby causing development of hyphae followed by angio-invasion and dissemination.[16] Therefore it is well understood that the immune status of the individual determining the host pathogen interaction plays a crucial role in the development of the disease.[6]

Mucormycosis of the oral cavity in majority of cases arise due to direct wound contamination and may lead to dissemination of the spores to other viscera as a common complication. Alternatively, another gateway of entry is inhalation (through the nose). It is a disseminated infection and presents as palatal ulceration leading to necrosis and black appearance of the affected area.[7]

Literature review on mucormycosis revealed that the age of majority of patients reported to be infected with mucormycosis was in/after third decade of life.[17]

However, in the present case, patient age was fourteen years (juvenile) wherein immune status is seldom questionable. Mucormycosis usually presents as a pulmonary, gastrointestinal, disseminated or rhinocerebral infection.[18]

In the presented case, infection was localized to the mandible with subsequent necrosis without any other reported symptoms. As per literature, the most common oral location of mucormycosis is maxilla. Isolated mandibular mucormycosis is rare with very few cases reported.[19] The pathogenesis of mucormycosis in mandible (which is mainly dependent on endosteal blood supply) appears to be different from that of involvement of maxilla.

Dissemination of mucormycosis is observed in diabetics with ketoacidosis, which favors rapid proliferation of fungus and its invasion into the orbit and cerebrum.[20]

In the present case although the patient was malnourished, he did not had ketoacidosis thereby restricting the dissemination of mucormycosis.

Literature review also revealed several cases of mucormycosis that were linked to direct inoculation after trauma (primarily observed for cutaneous infection). Also several cases are reported in patients exhibiting having iron overload, intravenous drug use, and malnourishment, even in the absence of diabetes and immunosuppression.[20]

Another major bulk of literature on mucormycosis comprises plethora of cases reporting mucormycosis in patients post COVID-19 infection.

However, in this patient no such history for COVID-19 positivity for reported. The history of previous infection with dengue virus is suggestive of the fact that dengue infection could have instigated development of mucormycosis infection similar to the findings of Toppo et al.[21]

Dengue is a viral infection that gets transmitted via mosquito and is a leading cause of death in tropical and subtropical countries.[22] It presents as a self-limited febrile illness in majority of cases, however the viral infection can induce virus-mediated host changes, hampering the

immuno-competency of the person leading to susceptible to life threatening fungal infections.[23]

It is observed that in post dengue patients who exhibit lack of adequate phagocytic activity or those having impaired phagocytic functions, there is a higher risk of development of mucormycosis. The immune-compromised status of the patients is reported to be the chief contributing factor.

Histopathologically, the lesion demonstrated broad aseptate fungal hyphae that exhibited branching at right angles. Also round or ovoid sporangia were frequently seen in tissue sections and a preference of the fungi for invasion of small vessels was observed.

The histopathological findings of the fungus were confirmed by PAS stain. The histopathological differential diagnosis includes aspergillosis where the hyphae of Aspergillus species are septate, smaller in width and branch at more acute angles.

Early diagnosis of mucormycosis is key to rapid and appropriate treatment and improved outcomes.

## CONCLUSION

The malnourished status of this juvenile patient, his habit of eating soil and subsequent infection by dengue virus compromised his immunity by hampering the phagocytic function. The immuno-compromised status, hospitalization and negligence towards timely reporting of the symptoms promoted development of bone necrosis following tooth extraction and subsequently mucormycosis.

It therefore becomes responsibility of the clinician to carefully record the history of infections particularly dengue and adequately evaluate the immune status of the patient wherever mucormycosis is suspected.

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