# Rhino-orbito-cerebral mucormycosis in a child with diabetic ketoacidosis

Sir,

Rhino-orbito-cerebral mucormycosis is an unusual complication of diabetic ketoacidosis (DKA). Most of them have been fatal. High index of suspicion is critical for diagnosis and early treatment.

A 9-year-old, female child presented with fever, abdominal pain, and vomiting for 2 days. Examination revealed acidotic breathing and multiple furuncles over the face. She had hyperglycemia of 485 mg/dl, ketonuria, wide anion gap metabolic acidosis (pH 6.8) and polymorphonuclear leucocytosis.

She was diagnosed as Type 1 diabetes with DKA and treated according to the DKA management protocol of the unit. In view of persistent altered sensorium anti-edema measures were initiated. At 48 h of admission, she had a dark colored bloody nasal discharge. Injection amphotericin B was started empirically for suspected mucormycosis.

At 72 h, the child developed ophthalmoplegia of the right eye. A black necrotic palatal lesion was noted at 96 h [Figure 1]. Computed tomography (CT) brain was normal. CT paranasal sinuses showed right maxillary sinusitis. Nasal endoscopy revealed black necrotic eschar over the right middle and inferior turbinates, floor and lateral wall of nasal cavity. Despite surgical debridement child worsened with right facial palsy and left hemiparesis. Repeat CT brain revealed massive infarct involving right frontal, temporal and parietal regions. The child expired on day 9.

Histopathology of the necrotic lesion revealed broad, aseptate hyphae, branching at right angles with vascular invasion. Culture on saboraud dextrose agar medium grew black colonies typical of *Rhizopus*. On lactophenol cotton blue mount presence of sporangium and rhizoids typical of *Rhizopus* was seen. Postmortem cerebral spinal fluid was unusually orange-red colored with no cells, with 1250 mg% proteins and 35 mg% sugar. Hemoglobin A1c was high (12.6%).

Mucormycosis is an acute, frequently fatal, fungal disease in immunocompromised children due to *Rhizopus*[1] followed by *Mucor*. Risk factors include DKA, prolonged neutropenia, malignancy, chronic corticosteroid therapy and deferoxamine therapy. Rhinocerebral (most common in DKA),[2] pulmonary, cutaneous, gastrointestinal, and disseminated involvement can be seen.

In DKA, the chemotactic activity of neutrophils and their ability to produce reactive oxygen species is diminished. Acidosis disrupts the transferrin binding to iron,[3] releasing free iron favoring *Rhizopus* growth. As the advanced glycosylation end products increases the transendothelial migration of neutrophils decreases.

Following inhalation, the disease begins with facial pain and numbness followed by sinusitis and dark bloody nasal discharge. In this child, nasal discharge was the first sign. The infected tissue appears normal initially

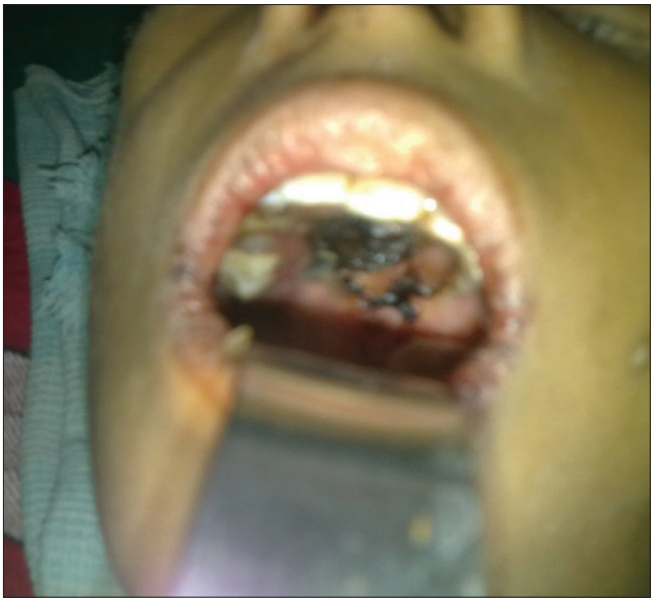

**Figure 1:** Black necrotic eschar in palate

then become erythematous, violaceous and finally turns black due to thrombosis and tissue infarction.[4] Rapid progression of the disease occurs in hours to days. Direct microscopic examination of the specimen and culture are diagnostic. Management includes reversal of the risk factor, surgical debridement along with antifungal therapy. Amphotericin B (polyene) is the drug of choice given over few weeks. Amphotericin B liposomal complex, echinocandins with polyenes, posaconazole and deferasirox can also be used.

Clinical suspicion warrants treatment without awaiting confirmation. Mortality is more than 40% with cerebral involvement. It is less than 10% without cerebral involvement.[2] Poor prognostic factors are delayed diagnosis, inadequate surgical debridement, spread beyond sinonasal cavity, and cerebral involvement.

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## References

1. Ribes JA, Vanover-Sams CL, Baker DJ. Zygomycetes in human disease. Clin Microbiol Rev 2000;13:236-301.
2. Nithyanandam S, Jacob MS, Battu RR, Thomas RK, Correa MA, D'Souza O. Rhino-orbito-cerebral mucormycosis. A retrospective analysis of clinical features and treatment outcomes. Indian J Ophthalmol 2003;51:231-6.
3. Artis WM, Fountain JA, Delcher HK, Jones HE. A mechanism of susceptibility to mucormycosis in diabetic ketoacidosis: Transferrin and iron availability. Diabetes 1982;31:1109-14.
4. Petrikkos G, Skiada A, Sambatakou H, Toskas A, Vaiopoulos G, Giannopoulou M, *et al*. Mucormycosis: Ten-year experience at a tertiary-care center in Greece. Eur J Clin Microbiol Infect Dis 2003;22:753-6.

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