International Journal of Internal and Emergency Medicine

Case Report
Published: 23 Aug, 2021

Mucormycosis Involving Anterior Abdominal Wall Following Insect Bite in a Diabetic Patient - A Case Report

Vijay B[1], Atul P[2], Amrish P[3], Sruja N[4], Ranjit Z[4] and Rahul K[4]*

[1]*Department of Burns and Plastic Surgery, Sterling Hospital, India*

[2]*Department of Infectious Diseases, Sterling Hospital, India*

[3]*Department of Pulmonary and Critical Care Medicine, Sterling Hospital, India*

[4]*Department of Burns and Plastic Surgery, SVP Hospital, India*

Abstract

Mucormycosis is a fatal fungal infection predominantly occurring in a diabetic and in immunocompromised patients. Infection is acquired by inhalation, ingestion or traumatic inoculation in a susceptible host. Impairment of host defence mechanisms affecting innate immune system facilitates invasive fungal infection. Skin and soft tissue mucormycosis, necrotizing fasciitis has been described in victims of road traffic accidents, natural disasters (Tsunami, tornado) who had dirt contaminated wounds. Clinical presentation of such patient is non-specific for mucormycosis and represents bacterial necrotizing skin and soft tissue infection. Early clinical suspicion and appropriate microbiological work up allow early diagnosis and appropriate antifungal therapy. With this case report we are describing mucormycosis involving anterior abdominal wall in a diabetic patients and challenges faced in the treatment.

Introduction

The *Zygomycoses* are infections caused by fungi of the class Zygomycetes, comprised of the orders Mucorales and *Entomophthorales*. The *Entomophthorales* largely afflict immunocompetent hosts in developing countries. In contrast, fungi of the order Mucorales are causes of mucormycosis, a life- threatening fungal infection predominantly affecting Immunocompromised but can produce infections in immunocompetent hosts as well. It is associated with high mortality with overall mortality approaches to 50%. Site of infection and underlying host immune status are two most important factors influencing mortality [1,2]. Combination of medical treatment, antifungal therapy and surgical debridement improves the outcome of mucormycosis [3]. With this case report, we are describing mucormycosis involving anterior abdominal wall in a diabetic patient to improve understanding of clinical presentation, pace of progression and treatment challenges involved in the management.

Case Presentation

A 58 year old male diabetic patient presented to Sterling Hospital for further management for the post debridement raw are on the left lower quadrant of the abdomen. Patient described a small, ulcerated nodular lesion of 0.5 cm × 0.5 cm following unidentified insect bite over left lower abdomen that rapidly progressed despite oral antibiotics and surgical debridement. Patient was referred for evaluation and treatment of worsening raw area over the abdomen to Sterling Hospital after day 3 of debridement with endotracheal tube *in-situ*. On admission, patient had a 23 cm × 25 cm raw area over abdomen (Figure 1). His laboratory reports revealed metabolic acidosis, raised white cells count and inflammatory markers. Patient underwent vigorous debridement 13 times; however debridement in the midline was done conservatively to prevent burst abdomen and regular dressings every alternate day from the day of admission in operation room to maintain all aseptic precaution. At each debridement the margin of the necrotic tissue involved by the fungus was increasing by 1 cm leading to a large raw area measuring 35 cm × 36 cm. Tissue sent for direct microscopic examination, showed broad aseptate mycelial filaments, tissue culture grew *Apophysomyces elegans* and histopathology confirmed tissue invasion with mycelia. Patient was treated with liposomal Amphotericin B (5 mg/kg/day) with Meropenem 1 gm IV q8h. Tablet Isavuconazole (200 mg three times a day for two days and then 200 mg once a day) was added to L-AmB on day 7 of hospitalization as culture grew *Apophysomyces elegans* which has high minimum

OPEN ACCESS

Correspondence:
Vijay Bhatia, Department of Burns and Plastic Surgery, Sterling Hospital, Ahmadabad, Gujarat, 380059, India, Tel: +91-9825073828; E-mail: bhatia101@gmail.com

Received Date: *20 Jul 2021*
Accepted Date: *19 Aug 2021*
Published Date: *23 Aug 2021*

Citation:
Vijay B, Atul P, Amrish P, Sruja N, Ranjit Z, Rahul K. Mucormycosis Involving Anterior Abdominal Wall Following Insect Bite in a Diabetic Patient - A Case Report. Int J Intern Emerg Med. 2021; 4(1): 1040.

inhibitory concentration for Amphotericin-B. He underwent multiple debridements along with antifungal therapy. Patient responded to his treatment, showing improvement from day 17 of hospitalization further disease progression stopped. He received split thickness graft from bilateral thighs, anterior, medial and lateral on day 21 of hospitalization. He underwent dressing of the spit thickness graft in the operation theatre. He received 4 weeks of L-AmB and 42 days of Isavuconazole. The patient was discharged uneventfully on day 38 post admission.

Pathogenesis

The phagocytic cells like mononuclear and polymorphonuclear, phagocyte and kill Mucorales in a normal healthy host by oxidative metabolites. Neutropenic patients and patients with dysfunctional phagocytes are at higher risk of developing mucormycosis. Hyperglycemias and metabolic acidosis are known to impair the functional ability to eliminate and fight the organism by oxidative and non oxidative mechanisms, leading to increased susceptibility towards grave infection.

Mucormycosis is angioinvasive infection, more marked in a neutropenic host. Angioinvasive capability resulting in to vessel thrombosis and tissue necrosis. This angioinvasion is also associated with the ability of the organism to spread hematogenously to a distant organ apart from a local spread. Hence, damage of and penetration through endothelial cells lining blood vessels is likely a critical step in the organism's pathogenic strategy [2].

Discussion

Mucormycosis is an aggressive, rare, opportunistic infection caused by *Zygomycetes* class of fungi, order mucorales. Genera most commonly responsible for mucormycosis usually are *Rhizopus*, *Mucor*, *Rhizomucor*, *Licthemia Corymbifera*, *Apophysomyces*, *Cunnighamella* and others. Rhino-orbito-cerebral mucormycosis is commonest form, occurring in conjunction with the paranasal sinus or nasal infection with local spread to orbit and brain [4]. Primary cutaneous mucormycosis in humans is limited in most cases to, patients with severe immunocompromised state, diabetes mellitus or trauma [5]. The fungi invade the blood vessel lumen and cause thrombosis through inflammatory cascade and this is the pathognomic feature of this entity. There is often a mixed suppurative and necrotizing inflammatory reaction in the dermis and subcutaneous tissue (Figure 2).

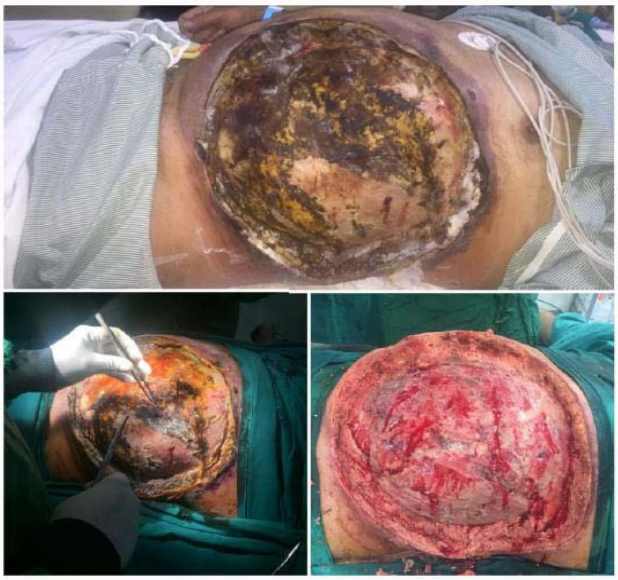

Figure 1: Wound at the time of presentation requiring vigorous debridement.

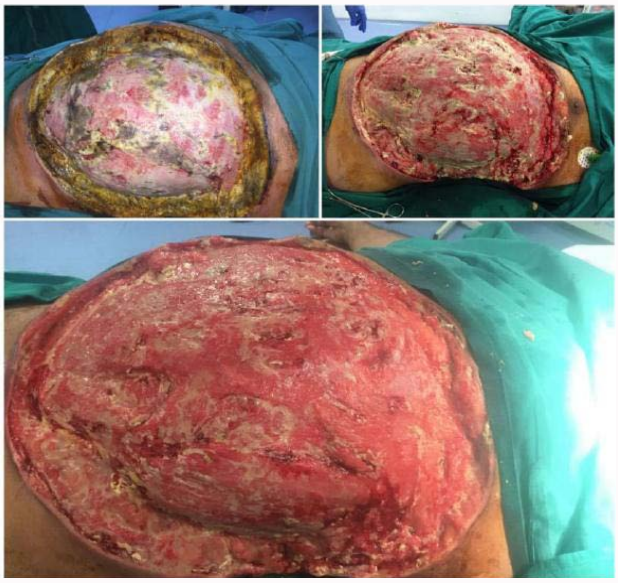

Figure 2: Wound requiring multiple debridement and dressings.

In the present case, the patient had history of uncontrolled diabetes mellitus and insect bite over the abdomen both acting as pre disposing factors for fungal infection. Clinical manifestation appeared as expanding necrotic ulcers with black eschars. Histopathology examination showed hypha with thick, non-septate, vertical branch. Tissue culture found fungal growth of *Apophysomyces elegans*. *Apophysomyces elegans* is commonly found in dirt, being first isolated from the soil in India in 1979 [6]. *A. elegans* most commonly causes infections in apparently healthy individuals [6-8]. Infection of cutaneous and subcutaneous tissues with *A. elegans* is predominantly the result of the introduction of spore-containing soil and vegetation into wounds arising from trauma or surgery, burns, injection, or an insect bite [6-8].

Infection in our case was rapidly spreading by 1 cm in 24 h and was frightening because of the risk of burst abdomen.

Primary Cutaneous Mucormycosis (PCM) is often reported in patients with local trauma or predisposing conditions including diabetes mellitus, hypo-immune functions and chronic renal failure. Our patient had history of local trauma in form of insect bite complicated by uncontrolled diabetes mellitus. Cutaneous mucormycosis is segregated further into two subtypes according to the cutaneous manifestation. The superficial variant presents with a gradual onset and slow progression of symptoms [9]. The gangrenous form of cutaneous mucormycosis advances rapidly like in our case, causing painful ulcers and eschars [10].

In order for fungus to cause disease, it must acquire sufficient iron for the growth from the host and evade the phagocytic defence to access the vasculature. In hosts with co-morbidities like diabetes mellitus, the iron is released from sequestering protein [8]. Acidotic conditions decrease the iron- binding capacity, suggesting that acidosis per se disrupts the capacity of transferrin to bind iron, probably by proton-mediated displacement of ferric iron from transferrin. Fungi can

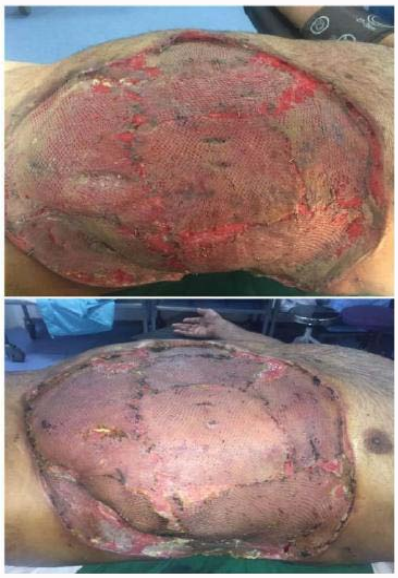

Figure 3: Split thickness grafting done after multiple vigorous debridement.

obtain iron from the host by using high-affinity iron permeases or low-molecular-weight iron chelators (siderophores).

Cutaneous disease can be very invasive locally and penetrate from the cutaneous and subcutaneous tissues into the adjacent fat, muscle, fascia, and even bone. Secondary vascular invasion may also lead to hematogenously disseminated infection of the deep organs. Cutaneous and subcutaneous disease may lead to necrotizing fasciitis, which has a mortality approaching 80%. However, isolated cutaneous mucormycosis (i.e., not disseminated disease) has a favorable prognosis and a low mortality if aggressive surgical debridement is done promptly [11]. The large post debridement raw area needs coverage by split thickness grafting, as performed in the presented case (Figure 3).

The successful management of infections caused by *Mucormycosis* requires an early diagnosis, control or reversal of any predisposing factors or underlying disease, antifungal therapy (amphotericin-B being the drug of choice), and aggressive surgical debridement which may have to be repeated until all infected necrotic tissue is removed. This case is being presented because of its rarity, and to emphasize the role of early diagnosis and appropriate treatment, in a life threatening disease.

Conclusion

Mucormycosis of skin and soft tissue usually has favorable prognosis especially with early diagnosis and treatment. Mucormycosis as a possibility should be kept in patients with unusual progressive necrotizing skin and soft tissue infection. Debrided tissue should be sent to microbiology laboratory for direct microscopic examination with KOH preparation and routine stain, along with tissue culture and histopathology. Timely intervention with vigorous regular debridement of the dead and necrotic tissue and appropriate antifungal medication improves the outcome and reduces the morbidity. Prompt control of hyperglycemia and reversal of ketoacidosis are important component of mucormycosis treatment.

References

1. Ribes JA, Vanover-Sams CL, Baker DJ. Zygomycetes in human disease. Clin Microbiol Rev. 2000;13(2):236-301.
2. Roden MM, Zaoutis TE, Buchanan WL, Knudsen TA, Sarkisova TA, Schaufele RL, et al. Epidemiology and outcome of zygomycosis: A review of 929 reported cases. Clin Infect Dis. 2005;41(5):634-53.
3. Patel A, Kaur H, Xess I, Michael JS, Savio J, Rudramurthy S. A multicentre observational study on the epidemiology, risk factors, management and outcomes of mucormycosis in India. Clin Microbiol Infect. 2020;26(7):944.e9-944.e15.
4. Gilbard SM, Della Rocca RC. Paranasal sinus disease and the orbit. In: Black EH, Nesi FA, Gladstone G, Levine MR, Calvano CJ, editors. Smith's ophthalmic plastic and reconstructive surgery. 2nd Ed. CV Mosby, St. Louis, MO: 1998:896-930.
5. Alan MS. Agents of mucormycosis & related species. In: Gerald LM, John EB, Raphael D, editors. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. 6th Ed. Churchill Livingstone, Philadelphia: 2005:2973-84.
6. Prakash H, Chakrabarti A. Epidemiology of mucormycosis in India. Microorganisms. 2021;9(3):523.
7. Prakash H, Chakrabarti A. Global epidemiology of mucormycosis. J Fungi (Basel). 2019;5(1):26.
8. Steinbrink JM, Miceli HM. Mucormycosis. Infect Dis Clin North Am. 2021;35(2)435-52.
9. Ledgard JP, van Hal S, Greenwood JE. Primary cutaneous zygomycosis in a burns patient: A review. J Burn Care Res. 2008;29(2):286-90.
10. Prabhu RM, Patel R. Mucormycosis and entomophthoramycosis: A review of the clinical manifestations, diagnosis and treatment. Clin Microbiol Infect. 2004;10(Suppl 1):31-47.
11. Chakrabarti A, Kumar P, Padhye AA, Chatha L, Singh SK, Das A, et al. Primary cutaneous zygomycosis due to Saksenaea vasiformis and Apophysomyces elegans. Clin Infect Dis. 1997;24(4):580-3.