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Human Mucormycosis

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Introduction

Incidence of mucormycosis, the severe infection in immunocompromised and immunocompetent patients [1,2], is increased in recent years. In the United States, 63% of non *Aspergillus* mold infections in transplant recipients were caused by Mucoral family [3] with mortality rate of 22%, 25%, 48% and 79% for cutaneous, rhino-cerebral, pulmonary and disseminated infections, respectively [3]. In US and France, *Rhizopus* species are the most common genera [1,3].

The presentation type of this infection usually depends on the underlying host conditions [4]. Hyperglycemia and low pH in diabetic patients are the most important risk factors. The common risk groups include those on therapeutic interventions corticosteroid therapy, solid tumors and solid organ transplant recipients, acquired or congenital neutropenic patients, those with hematologic disorders and hematopoietic stem cell transplant recipients. In last decades, a link has been reported between infection with the *Rhizopus* species and resistant gram-negative bacteria which can produce endosymbiotic toxin [4,5]. Hemodialysis patients under treatment with deferoxamine, due to high concentration of free iron in plasma and tissues are at risk for mucormycosis [6]. The common presentations are rhino-orbital-cerebral involvement, pulmonary and gastrointestinal infection, and invasive mucormycosis (central nervous system infection).

Mucoral spores with 3-11μm diameters can be easily aerosolized and dispersed throughout the environment and inhaled by human. Human may also be infected by taking infected food through the gastrointestinal tract, and also by direct inoculation through skin (trauma, burn) or central venous catheters [7]. Invasion to human endothelial cells is related to host receptor (the glucose-regulated protein 78) and iron (expression of the high affinity iron permease gene and its product). Therefore, diabetic patients with high serum iron level and glucose concentrations are more susceptible to mucormycosis than other high risk patients. Mucorales could invade the tissues by proteolytic, lipolytic, and glycosidic enzymes and also with metabolite products like mycotoxins such as agroclavine [6,8].

The first barriers to immune defense in human are ciliated bronchial cells and their mucus that lead sporangiospores away from alveoli by coughing. Macrophages and neutrophils play the major role in immunedefense. Spores are phagocyted by macrophages oxidative killing and hyphae are damaged by neutrophil oxidative cytotoxic system and chemotactically attracted. Pulmonary alveolar macrophages damage the invading organisms and to regulate innate immune response secrete cytokines and chemokines to destroy sporangiospores before they terminate to become hyphae. In hosts with diabetes, the monocytes/macrophages are dysfunctional and fail [5,9,10].

The gold standard test for diagnosis is the isolation of organism from clinical samples. The hyphae of this family are delicate and frequently destroyed in the process of sample preparation, therefore, isolation of organisms from clinical samples remains difficult. Serological diagnostic method for these infections is not available but use of molecular methods like a polymerase chain reaction technique is recommended in the literature [11].

Conclusion

Mucormycosis has emerged as severe infection in immunocompromised and immunocompetent patients in many health centers worldwide. Epidemiology, diagnosis and treatment are with many unresolved issues and to guide the best management newer diagnostic tests to initiate appropriate treatment are suggested.

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