

INTRODUCTION

Diabetic patients are more prone to infection. Prior to the advent of modern antimicrobial therapy, infection account for much of morbidity and mortality in diabetes. (*Top et al., 2005*).

Uncontrolled diabetes rapidly promotes infection, as sugar is a good medium for rapid and abundant growth of organisms. The occurrence of infection in a diabetic patient initiates a vicious cycle in which infection results in uncontrolled hyperglycemia which in turn causes further aggravation of infection. (*Trivedi, 2003*).

Many infections are common in patient with diabetes; as pyelonephritis, and soft tissue infection (Diabetic foot, necrotizing fasciitis, mucocutaneous candida infection) (*Bertoni et al., 2001*).

Foot infection is the most common soft tissue infection associated with diabetes, related peripheral neuropathy and peripheral vascular disease play a major role in this complication of diabetes mellitus (D.M.). More serious complications are osteomyelitis, amputation and death. Infection begins after minor trauma and may progress to cellulitis, soft tissue necrosis and may extend into bone (*Shah & Hux, 2003*).

A number of studies have found that staphylococcus aureus is the main causative pathogen in diabetic foot infection but recent studies reported a predominance of gram-negative aerobes, while role of anaerobes is unclear (*Abdulrazak, 2005*).

Increased susceptibility to infection in diabetic is multifactorial. Several immunological factors are related to increase risk of infection, first, neutrophil function is depressed affecting adherence to endothelium, chemotaxis, phagocytosis and bactericidal activity. Also cell mediated immunity (CMI), serum level of Igs (Immunoglobulins), complement

function all are reduced in diabetic patient, these impairment are exaggerated by hyperglycemia, academia , but reversed by normalization of pH (the measure of the acidity of a solution)and blood glucose level (*Tan , 2005*).

Furthermore, susceptibility of target tissue to hyperglycemia , vascular disease, and nerve damage cause proness to infection, and reduction of antibiotic absorption due to microangiopathy lead to persistence of infection (*Trivedi, 2003*).