Host response to Candida albicans bloodstream infection and sepsis.

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Details

Abstract

Candida albicans is a major cause of bloodstream infection which may present as sepsis and septic shock - major causes of morbidity and mortality world-wide. After invasion of the pathogen, innate mechanisms govern the early response. Here, we outline the models used to study these mechanisms and summarize our current understanding of innate immune responses during Candida bloodstream infection. This includes protective immunity as well as harmful responses resulting in Candida induced sepsis. Neutrophilic granulocytes are considered principal effector cells conferring protection and recognize C. albicans mainly via complement receptor 3. They possess a range of effector mechanisms, contributing to elimination of the pathogen. Neutrophil activation is closely linked to complement and modulated by activated mononuclear cells. A thorough understanding of these mechanisms will help in creating an individualized approach to patients suffering from systemic candidiasis and aid in optimizing clinical management.

Beteiligte Forschungseinheiten

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