Background. Oral and nappy Candida infections are common in neonates and infants, but candidaemia and its consequences are more often seen in children with risk factors for immunosuppression. This case presentation illustrates that exposure to HIV, without infection, should be considered one of those risk factors.

Objectives. To determine whether HIV-exposed, but uninfected, children have immune dysfunction that could alter their disease profile, and to elucidate the interactions of the gastrointestinal tract (GIT) with Candida infections.

Methods. Keywords/key phrases searched on databases were: candidiasis; GIT and Candida; HIV-exposed infants; immunity and HIV-exposed neonates.

Results. Several detailed original studies confirm an impaired immunological response in neonates and infants born to HIV-infected mothers. Impairment extends to children born to mothers on antiretroviral medication. The duration of immune dysfunction is unclear, but it appears to persist for several years. Homeostasis of the GIT is essential in order to prevent the translocation of Candida into the bloodstream. GIT immunity plays a critical role in the clearance of fungi. The HI virus interferes negatively with this ability.

Conclusion. If HIV-exposed but uninfected children have a degree of immunodeficiency, then the risk of opportunistic infections is higher than in HIV-unexposed infants. The clinician should bear this in mind when these patients present, in order to decrease the morbidity and mortality associated with delayed diagnosis and treatment of candidaemia.

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Candida becomes pathogenic when the gastrointestinal immune defences are compromised. Changes in local conditions and immune responses result in mucosal disease, and this in turn increases the chance of invasive disease developing. Only slight alterations are necessary in order for Candida to transform from a commensal organism to a pathogenic one. For this reason, even superficial Candida infections should be regarded in a serious light when present in immunocompromised individuals.

Changes in immunocompromised individuals include immune and cellular interaction at a gastrointestinal level that affect the initiation of a systemic inflammatory cascade and clearance of a pathogen. Gut-associated lymphoid tissue contains T and B cells and immunoglobulin A. Differentiation and proliferation is stimulated when these cells interact with C. albicans. A low concentration of immune cells is proportional to a weaker immune response.

When the Candida-mucosal epithelial cellular interaction is altered, under-stimulation of neutrophils (important in stimulating immune pathways and direct killing of fungi) and subsets of T and B cells, results. Production of pro-inflammatory cytokines and chemokines by the host cells in response to C. albicans plays a critical role in recruitment and activation of immune cells and final clearance of organisms. T-helper lymphocytes play an integral role in defence against fungi. Once activated, these cells produce pro-inflammatory cytokines. Since the immune response plays such an important role in maintaining homeostasis, a decrease in the immune system’s arsenal in HEU infants is a significant concern when treating superficial Candida infections. This is especially true when the infant presents with signs of systemic infection and a superficial candidiasis is also diagnosed. Perforation of the GIT is a natural progression of the invasive form of Candida infection, and this diagnosis should be higher up on the differential diagnosis list than it would be for an immunocompetent infant.

Conclusion

A growing body of evidence suggests that HEU neonates and infants are immune impaired when compared with HUU individuals – this is despite the use of PMTCT. It is also well established that gut-Candida interaction and local immunity play an important role in the establishment of invasive candidiasis. HEU children may already be at a disadvantage when encountering other illnesses that stress their already weakened immune system, and may have an increased risk of intestinal perforation from C. albicans. Superficial Candida infections are common, but in HEU infants they should be closely monitored and their resolution ensured, as they may be an external indicator of an internal process.

Further studies should be encouraged to determine whether Candida overgrowth of the GIT is associated with invasive disease in HEU patients, as opposed to a just a pathological change of the fungus in normal concentrations. These studies would guide treatment methods, determining whether options such as use of non-absorbable antifungals to eradicate Candida overgrowth in the GIT, should be utilised in HEU patients presenting with superficial Candida infection.

References


