Trichosporon asahii Causing Skin Lesions upon Ileostomy and Ureterostomies in an Immunodeficient Patient: A Case Report and a Mini Review of the Literature

Stergios Karapsias
Clinical Microbiology Laboratory, 251 General Air Force Hospital, Athens, Greece

*Corresponding author: Stergios Karapsias, Clinical Microbiology Laboratory, 251 General Air Force Hospital, Athens, Greece, Tel: 0306983521853; E-mail: kast594594@yahoo.gr

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Abstract

A case of superficial skin lesions due to T. asahii is reported, concerning an immune-deficient patient while receiving micafungin. Yet, T. asahii is assumed as intrinsically resistant to echinocandines. Thus, compared to echinocandines, azoles should be preferred in antifungal prophylaxis of immunosuppressed patients because they treat a wider variety of opportunistic fungi, including T. asahii. Additionally, T. asahii should be regarded as a potential pathogen causing skin lesions in immune-depressed or surgical patients.

Keywords: Trichosporon asahii; Skin lesions; Immunodeficiency

Introduction

Trichosporon asahii is basidiomycetous yeast causing various opportunistic infections in immunodeficient patients. The present short review is based upon a case of fungal skin infections due to T. asahii in an immunosuppressed patient.

Case Study

A 70 year old male patient had been admitted in surgical wards for treatment of urine bladder cancer. After surgical removal of urine bladder and part of small intestine, an ileostomy and two ureterostomies were created, which macroscopically appeared inflammatory and consequently were microbiologically examined via cultures. Skin lesions included erythema, edema, locally increased temperature and mild pain. All cultures (ileostomy, two ureterostomies) showed Trichosporon asahii as the responsible pathogen for all superficial skin inflammatory lesions. Susceptibility testing against T. asahii was performed by calculating minimum inhibitory concentrations (MIC) to azoles (sensitivity), amphotericin B (sensitivity) and echinocandines (resistance).

Before T. asahii diagnosis was established, the patient had been receiving micafungin 100 mg X 1 iv (an echinocandine) in a preventive antifungal basis due to his immunodeficiency (cancer, chemotherapy, several surgeries). Yet, T. asahii bibliographically presents endogenous resistance to echinocandines and such resistance has been proved by our laboratory, too. So, despite antifungal treatment, superficial surgical wounds (ileostomy, ureterostomies) were opportunistically colonised and finally infected by T. asahii. As soon as fungal susceptibility testing was completed, micafungin treatment was replaced by a miconazole nitrate 2% cream, locally applied upon infected wounds and skin lesions. After five days of topical treatment, T. asahii was eradicated from infected skin wounds.

Discussion

T. asahii has been described as a pathogen causing fatal septic shock in a diabetic patient without cancer or neutropenia in the USA [1], nosocomial urinary tract infections in intensive care unit patients in China [2], community urinary tract infections in diabetic patients in India [3] and bacteraemia in a premature neonate in Greece [4]. Yet, T. asahii has been reported to cause urinary tract infections in immunocompetent patients in India [5].

Trichosporon species seem to colonize normal perigenital region as well as urine and catheters of hospitalised patients at the Intensive Care Unit in Brazilian hospitals [6]. Though, T. asahii rather prefers to contaminate urine and catheters than perigenital skin [6]. As a consequence of such a contamination following a potential colonization, our rare report -if not the first one- has presented T. asahii skin lesions in an immunodeficient operated patient.

T. asahii bibliographically is intrinsically resistant to echinocandines. For that reason, outbreaks of T. asahii infections, including our case, have been reported in immunosuppressed patients while receiving echinocandines [7, 8].

In conclusion, T. asahii appears mostly as a pathogen in immunodeficient patients. Since echinocandines are ineffective against such opportunistic fungal infections, they shouldn’t be used in a preventive antifungal basis. Compared to echinocandines, azoles should be preferred in antifungal prophylaxis because they treat a wider variety of opportunistic fungi, including T. asahii and remain more cost-effective, too. In addition, T. asahii should be regarded as a potential pathogen causing skin lesions in wounds of immunodepressed or surgical patients.

References