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Virulence factors and Azole-Resistant mechanism of *Candida Tropicalis* isolated from Candidemia

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Limited knowledge exists on the virulence factors of Candida tropicalis and the mechanisms of azole resistance that lead to an intensified pathogenicity and treatment failure. We aimed to evaluate the virulence factors and molecular mechanisms of azole resistance among C. tropicalis isolated from patients with candidemia. Several virulence factors, including extracellular enzymatic activities, cell surface hydrophobicity (CSH), and biofilm formation, were evaluated. Antifungal susceptibility pattern and expression level of ERG11, UPC2, MDR1, and CDR1 genes of eight (4 fluconazole resistance and 4 fluconazole susceptible) clinical C. tropicalis isolates were assessed. The correlation between the virulence factors and antifungal susceptibility patterns was analyzed. During a 4 year study, forty-five C. tropicalis isolates were recovered from candidemia patients. The isolates expressed different frequencies of virulence determinants as follows: coagulase 4 (8.9%), phospholipase 5 (11.1%), proteinase 31 (68.9%), esterase 43 (95.6%), hemolysin 44 (97.8%), biofilm formation 45 (100%) and CSH 45(100%). All the isolates were susceptible to amphotericin B and showed the highest resistance to voriconazole. There was a significant positive correlation between micafungin minimum inhibitory concentrations (MICs) and hemolysin production ($rs = 0.316$). However, we found a negative correlation between fluconazole MICs and esterase production ($rs = -0.383$). We observed the high expression of ERG11 and UPC2 genes in fluconazole-resistant C. tropicalis isolates. C. tropicalis isolated from candidemia patients extensively displayed capacities for biofilm formation, hemolysis, esterase activity, and hydrophobicity. In addition, the overexpression of ERG11 and UPC2 genes was considered one of the possible mechanisms of azole resistance.