276. Detection of Rhizopus oryzae-Specific Antigen (RSA) in Serum and Bronchial Alveolar Lavage Is a Potential Early Diagnostic Marker in Mucormycosis by R. oryzae
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Session: 40. Fungal Diagnostics
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Background. The diagnosis of mucormycosis was made by the identification of an organism in the histopathology with culture confirmation. However, culture often yields no growth, and histopathological identification of organism with typical of mucorales is sometimes difficult. Therefore, a reliable new diagnostic tool is expected. We reported a novel Rhizopus-specific antigen (23kDa, named protein RSA) by screening with a signal sequence trap was detected at significantly higher concentrations in serum and in lung homogenates in the infected mice on day 4. And the results indicated that RSA was a possible diagnostic marker of mucormycosis (Satoh K, et al. Medical Mycology, 2017, 55:113–119). Here, we examined whether the RSA was detected on early stage in sera and bronchial alveolar lavage (BAL) of infected mice.

Methods. We developed the ELISA Kit using monoclonal antibody for RSA. The mice were infected with cortisone acetate and cyclophosphamide; and R. oryzae was infected intratracheally. Mice sera and BAL was obtained from infected mice on day 1, 2, 3, and 4. Then the concentration of RSA in sera and BAL was evaluated using the ELISA Kit for RSA.

Results. The RSA was detected in sera and BAL on day 1, 2, 3, and 4. The concentration of RSA in sera and BAL were significantly higher on day 4 as compared with uninfected mice. And the concentration of RSA in sera was the upward trend through day 1 to 4. However, the concentration of RSA in BAL was stable through day 1 to 4.

Conclusion. The RSA is a potential early diagnostic marker in mucormycosis by R. oryzae.

Disclosures. All authors: No reported disclosures.

277. Identification and Antifungal Susceptibility of Candida Species Isolated from Bloodstream Infections Over a 14-Year Period
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Session: 40. Fungal Diagnostics
Thursday, October 3, 2019: 12:15 PM

Background. Invasive infections caused by Candida species are associated with significant morbidity and mortality. Historically, C. albicans has been the predominant species recovered from patients with candidemia. However, the changing epidemiology of invasive candidiasis now includes more non-C. albicans species, which may exhibit intrinsic resistance or reduced susceptibility to antifungal agents used for therapeutic intervention. We sought to evaluate the epidemiology and susceptibility of invasive Candida spp. isolates causing bloodstream infections at the NIH Clinical Center over a 14-year period.

Methods. Candida spp. isolates causing bloodstream infections between 2004 and 2018 were identified. Retrospective chart review was performed for infected patients in accordance with the IRB. All Candida isolates were recovered from frozen storage, obtained on Sabouraud Dextrose Agar, and isolate identities were confirmed by MALDI-TOF MS. Antifungal susceptibility testing was performed by broth microdilution and MICs were interpreted using current CLSI criteria.

Results. Between 2004–2018, we identified 98 unique clinical isolates from 77 patients with candidemia. Records from 75 of these patients were able to be reviewed, and the 27 day and 90-day mortalities were 24% and 52%, respectively. The average age at the time of culture positivity was 41.3 years (range 6.5 to 76.9 years). Thirty-one of 41 (70%) were identified behind C. albicans species associated with significant morbidity and mortality. Renal insufficiency was a known risk factor associated with the development of candidemia and azole resistance. As expected, fluconazole resistance was prevalent among C. glabrata (70%, N = 16) and C. krusei (100%, N = 6); however, a sizable proportion of C. parapsilosis (11%, N = 3), C. tropicalis (63%, N = 5) and C. albicans (22%, N = 4) strains also exhibited fluconazole resistance.

Conclusion. Our findings illustrate a high prevalence of non-C. albicans Candida spp. as the causative agents of bloodstream infections among patients at our institution. The clinical risk factors associated with the development of candidemia and azole resistance, as well as the molecular mechanisms of antifungal resistance are under investigation.

Disclosures. All authors: No reported disclosures.