ISOLATED CANDIDA ALBICANS PNEUMONIA IN AN ELDERLY FEMALE

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ABSTRACT
Candida pneumonia is a rare lung infection, majority of cases seen to be occurred in immunocompromised individuals secondary to haematogenous dissemination from a distant sites like skin, gastrointestinal tract or female genital tract. Invasive candida pneumonia without heavy colonization in a particular anatomical site is rarely reported. We report an isolated case of candida albicans pneumonia in an elderly female of 65 yrs. with mucosal erosion in left cheek of oral cavity in absence of heavy colonization.

KEYWORDS
Candida albicans, Pseudohyphae, Pneumonia, Invasive.

INTRODUCTION
Candida exists in unicellular forms as yeasts which reproduce by budding and in multicellular forms as pseudohyphae. The former types exist as commensals in oral cavity, gastrointestinal tract, respiratory tract, female genitourinary tract and skin of human, but take the invasive pathogenic pseudohyphal forms, when there is reduction in normal microbial flora in the body due to antibiotics or debilitation of the host.[1] Following impaired defence due to disease/drugs or there is mucosal/skin damage due to infection/injury. There are various pathogenic species of Candida i.e. C. albicans, C. krusei, C. parapsilosis, C. tropicalis, C. lusitaniae, C. glabrata, C. guilliermondii, C. pseudotropicalis, and C. dubliniensis,[2] among which the most prevalent one is C. albicans that account for over 80% of all oral yeast isolates.[3] The species of candida are strictly opportunistic. It has been reported that no superficial or systemic forms of Candida infections could be initiated in the absence of an underlying pathology.[4] Immunosuppressive diseases like leukaemia, diabetes mellitus, HIV or nicotine/alcohol abuse or steroids/cytotoxic/immunosuppressive drugs and prolonged antibiotics use have been reported to have some relation with increased colonization by Candida species at various anatomical sites. Invasive candida albicans pneumonia as an isolated case in absence of involvement of any other anatomical site is very rare.

CASE REPORT
A 65 year-elderly woman married, weighing 47 kg, addicted to tobacco chewing without history of diabetes and hypertension but a known case of right knee arthritis without any treatment admitted to pulmonary medicine department, SCB medical college, Cuttack with rhinorrhoea, low grade night fever (39.5°C) accompanied by chill & rigor, cough with scanty mucopurulent expectoration, exertional dyspnoea, loss of appetite and generalised weakness. All symptoms developed since one month except exertional dyspnoea for last 15 days duration. She had no history of TB/malignancy, prolonged use of any broad spectrum antibiotics or immunosuppressives.

During hospitalization, she had clinical signs of consolidation on left lower lobe and patchy inspiratory crackles and Ronchi on both hemithoraces. URT was normal except mucosal erosion on left cheek in oral cavity. Haematological examination showed TLC 9x10^9/L, Hb 10.4 gm% and differential count revealed neutrophils 70%, lymphocytes 25%, eosinophils 3% & monocytes 2%. Fasting blood sugar was 96 mg/dL. HIV, HBsAg, HCV, and RA factor were negative. C3 was 242 mg% and C4 was 40 mg%. Absolute neutrophil count 6.3x10^9/L, absolute lymphocyte count 2.25x10^9/L and total platelet count was 240x10^9/L There was hypoalbuminemia (3.3 gm%). Urine examination was normal.

Renal and liver profiles were within normal ranges. Ultrasound abdomen and pelvis only showed bilateral early medical renal disease and menopausal uterus. X-ray chest PA view showed homogenous airspace opacities bilaterally with left lower lobar consolidation. HRCT displayed bilateral multiple patchy airspace consolidations with surrounding ground glass opacities and feeding vessel sign located in peripheral lung associated with multiple mediastinal lymph nodes suggesting fungal granuloma along with left lower lobe consolidation. ECG and Echocardiogram revealed no abnormality.

Sputum smear for AFB was negative. Gram staining showed pus cells, epithelial cells and few gram positive cocci. Sputum was sent for fungal culture and awaited. Broad spectrum antibiotics like amoxiclav, azithromycin and metronidazole in adequate doses were administered basing on gram staining that showed no remission of fever or radiological improvement. Subsequently CT guided transthoracic needle aspiration (TTNA) was done over left infrascapular region and the specimen sent for AFB, bacterial and fungal culture. Then imtraconazole 200 bd was started empirically considering the HRCT findings, age and renal status.
Aerobic blood culture and fungal culture were found to be negative but after 8 weeks incubation of sputum and TBNA specimen on Sabouraud Dextrose Agar (SDA) media showed growth of candida albicans, confirmed by germ tube test. By the time, the patient was reviewed after one month and two months showing clinical and radiological improvement. The drug in same doses was extended for another month. This time after completing 3 months treatment, the patient discontinued the medication for 1.5 months and again came with fever and generalised weakness and subsequent investigations revealed appearance of new shadows on chest imaging, polyclonal hypogammaglobinaemia (Decreased Albumin 51.9%, α13.8%, α2 11.1%, increased β17.5%, β25.3%, increased γ20.4% in serum) on serology and no fungal growth in urine on culture. The patient was restarted the same medication for another 2 months. On follow up, she was found to develop complete wellbeing with radiological improvement. FOB was normal and BAL showed no growth of bacteria or fungus. The patient was advised to continue the same medication.
DISCUSSION

Candida albicans is the commonest endogenous human commensal in oral cavity, existing in yeast forms. In presence of immunodeficiency/mucosal injury, the transition from yeast forms to pathogenic pseudohyphae forms occur followed by local or systemic infection. Candida albicans is the predominant species responsible for primary candida pneumonia from oral cavity that accounts 40-70% of all candida pneumonia. It is also a predominant species in secondary candida pneumonia due to haematogenous dissemination, followed by C. glabrata, C. tropicalis, C. krusei and C. parapsilosis.[5]

Invasive candidia pneumonia occurs as a primary infection following aspiration from heavily colonized oral cavity or as a part of disseminated candidiasis from blood stream. Typical risk factors for invasive candidiasis include prolonged neutropenia, recent surgery, broad spectrum antibiotic therapy, intravascular catheters and IV drug use.[6] Other predisposing clinical circumstances are haematological malignancy like leukaemia, Hodgkin’s lymphoma, DM and severe immunosuppressive states. It has been at reported that alteration in mental states is the most important contributing factor than other risk factors.[5] Immunocompetent subjects are resistant to aspiration induced pneumonia, but are succumbed to infection by a small inoculum of IV injection.[5] It has been reported that defective neutrophil phagocytosis i.e. leukaemia rather than corticosteroids, which causes defective margination, is extremely susceptible to candida pneumonia. T-Cell defect leads to mucocutaneous infection in oral cavity as in case of HIV infection, but defects in neutrophil phagocytosis leads to invasive candidiasis.[8] Alkalinity, malnutrition and lack of oestrogen may result in growth of invasive and pathogenic pseudohyphae forms affecting different organ system either locally or systemically. In elderly aging mucosal pattern, age related cellular immunity and frequent silent aspiration in normal sleep due to impaired physical defence are most important contributing factors in development of candida pneumonia.

Clinical manifestations in candida pneumonia are non-specific and depend on the site of infection, extent of involvement and the host immune response. Isolation of candida from sputum although not diagnostic, but is an index of suspicion for LRTI infection. Chest x-ray usually shows patchy consolidation due to bronchopneumonia, sometimes lobar consolidation and miliary nodulations. HRCT thorax displays patchy consolidations, CT halo signs, feeding vessel signs and miliary nodulations suggesting invasive forms of candida pneumonia. In contrast, the feeding vessel sign is rarely reported in lung carcinomas and granulomas.
The candida species are weakly gram-positive and stain with PAS and Grocott’s methamine silver, but diagnosis of candida albicans is usually confirmed by culture or histopathological demonstration of yeasts and pseudohyphae in target tissues. Candida is reported to colonize the pre-existing pathological lesions. It has also been reported upto 40% of cases with disseminated candidiasis that candida from blood may not be isolated by multiple blood culture.\[5\]

Our case is an old menopausal lady, presented with bilateral diffuse patchy consolidations, “CT halo sign” and “feeding vessel sign”, mucosal erosion on left cheek without obvious haematological disorders, immunosuppressive therapy, prolonged antibiotics and DM. There was no fungaemia or funguria. But the culture from target tissue showed growth of candida albicans, confirmed by germ tube test with good response to antifungal therapy implying the diagnosis of isolated candida albicans pneumonia possibly due to recurrent aspirations or haematogenous dissemination.

CONCLUSION
Entry of small inoculum of organisms into lung parenchyma either by recurrent silent aspirations or by haematogenous dissemination from site of injury can give rise to invasive candida pneumonia as an isolated lesion without any evidence of other organ involvement.

Sputum culture displaying fungal growth shouldn’t be ignored as a diagnostic clue for LRT infection.

REFERENCES