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ПРИКАЗ БОЛЕСНИКА / CASE REPORT
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Successful Treatment of Pseudomembranous Necrotizing Aspergillus Tracheobronchitis in a Patient with Acute Myeloid Leukemia

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SUMMARY

Introduction Pseudomembranous necrotizing Aspergillus tracheobronchitis is a rare form of pulmonary aspergillosis which occurs in immunocompromised patients.

Case Outline A female patient aged 71, suffering from acute myeloid leukemia, developed the symptoms of progressive shortness of breath and inspiratory stridor. The diagnosis in our case was made on the histological findings from tissues obtained by bronchoscopy. A chest CT scan suggested the state of the compromised trachea and left principal bronchus lumen. The long-term regimen with itraconazole in the dose of 400 mg/24 hours proved efficient in our patient.

Conclusion Progressive shortness of breath and inspiratory stridor in immunocompromised patients along with radiological and CT changes should be also considered as pulmonary aspergillosis in differential diagnosis.

Keywords: aspergillosis; acute myeloid leukemia; trachea; large bronchi; immunodeficiency; antifungal medicament

INTRODUCTION

Pseudomembranous necrotizing Aspergillus tracheobronchitis (PNTB) is a rare form of invasive pulmonary aspergillosis which occurs in immunocompromised patients, primarily those with neutropenia, hematological disorders, acquired immunodeficiency syndrome (AIDS), and patients who underwent bone marrow or organ transplantation. The diagnostic criteria include *Aspergillus* isolation from the cultures of tracheobronchial tree samples, or the histological confirmation of *Aspergillus* in the affected tissue, along with confirmation of the absence of alternative diagnosis, as well as the absence of radiological, clinical and histological evidence of invasive parenchymal aspergillosis [1].

Aspergillus tracheobronchitis (ATB) can be manifested as obstructive tracheobronchitis, ulcerative tracheobronchitis and PNTB, the latter being the most unfavorable form of ATB. PNTB is known to have a fatal outcome in 90% of patients, regardless of the administered antifungal drug [2-5]. The degree of immune dysfunction is crucial for treatment outcome [4].

CASE REPORT

A 71-year-old female patient, non-smoker, was admitted at the Institute for Pulmonary Diseases of Vojvodina as an emergency case, with dyspnea, orthopnea and inspiratory stridor, immediately after completed second chemotherapy course (cytosine arabinoside + daunoblastin) according to the protocol for acute myeloblastic leucemia (AML). There were no other illnesses before. Laboratory findings revealed leukocytosis (WBC 13.9×10^9/L), erythrocyte count of 3.59×10^{12}/L, hemoglobin 113 g/L, and platelets 301×10^9/L. Biochemistry findings involving glucosa, urea, creatinine, sedimentation rate, C-reactive protein (CRP), fibrinogen, and coagulation status were within the reference values range, except for lactate dehydrogenase (LDH) that was 240 U/L. On admission the patient showed global respiratory failure with tachypnea within 24 min. Oxygen saturation was 94%, oxygen partial pressure of 10:00 kPa and carbon dioxide 6:54 kPa.

The standard chest X-ray finding revealed extended shadow of the upper mediastinum and cardiac vessel shadows, with reduced transparency at lung bases. The CT scan of the chest revealed a stricture in the lumen of the trachea and the left main bronchus, as well as a moderate mediastinal lymphadenomegaly. Extended cardiac structures and stain-like lesions of the lung parenchyma were detected primarily in the lower lobes of both lungs (Figure 1). Bronchoscopic examination showed normal findings of the larynx, but with strictures of the trachea and with hyperemic mucosa which abundantly bled during contact with the instrument. The tracheal lumen was covered with necrotic, soft yellowish masses. The masses were partially covering tracheal mucosa in the form of a pseudo membrane, which reduced the lumen of the trachea. This content was also present at

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