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Case report

Successful management of pulmonary hemorrhage and aspergillosis in a patient with acute myeloid leukemia (AML-M3)

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ABSTRACT

A 35-year-old man presented with a one month history of gingival bleeding. He was diagnosed with Acute Myeloid Leukemia (AML-M3). During treatment he developed alveolar hemorrhage for which he was treated with a steroid. After the steroid treatment he developed a nodule, a cavitary lesion and atelectasia in the left lung. He was treated with voriconazole. After therapy with voriconazole his lesion significantly decreased. This case illustrates the efficacy and safety of antifungal therapy with voriconazole for aspergillosis complicated by AML.

1. Introduction

Acute myeloid leukemia (AML) is a hematopoietic neoplasm of the myeloid line of blood cells. Acute Promyelocytic Leukemia (APL) is a biologically and clinically distinct form of AML. APL is rarely seen in the first decade but increases with the second decade and into early adulthood [1]. APL patients present with symptoms such as pancytopenia, fatigue, infection, bleeding gums, bleeding, nosebleeds and disseminated intravascular coagulation [2].

Hematological malignancies are associated with many opportunistic infections including invasive aspergillosis (IA), an important destructive fungal infection [3]. IA complicates 5–29% of the cases of Acute Myeloid Leukemia (AML), and the risk is correlated with the degree of immuno-suppression following chemotherapy [3–5].

Voriconazole is a triazole derivative which is frequently used due to its potency, broad spectrum activity, clinical efficacy, safety and tolerance [6,7]. Numerous case studies and randomized control trials have shown that voriconazole is superior to amphotericin B in the treatment of IA and it is now considered the first-line therapy in many treatment centers [8]. I.

Below we present a case of AML-ML complicated by invasive aspergillosis treated with voriconazole.

2. Case report

A 35-year-old man presented with a one-month history of fatigue and gingival bleeding. Physical examination of the patient revealed hepatomegaly. Complete blood count (CBC) showed low counts with peripheral blasts. Subsequent bone marrow examination and immunophenotyping confirmed the diagnosis of Acute Myeloid Leukemia (AML-M3), but the patient refused treatment. Fifteen days later, the patient was admitted to hospital with deterioration of his overall condition. He received induction chemotherapy with All Trans Retinoic Acid (ATRA), cytarabine and idarubicin. On the thirtieth day of chemotherapy, chest pain, dyspnea and hemoptysis occurred. Results of laboratory examination were as follows: Hemoglobin: 8.5 g/dL, WBC: 1.4×10^9/L, Platelets: 60×10^9/L, PT 16 s, APTT: 26 s, INR of 1.3. On chest X-rays, ground glass opasities were seen in both lungs. In the parenchymal window on the thorax CT, diffuse bilateral pulmonary alveolar hemorrhaging combined with patchy opacities were observed (Fig. 1). The patient's condition was diagnosed as ATRA-dependent alveolar hemorrhage and steroid treatment was begun (Dexametazon 2×10 mg/day). About 15 days after initiation of steroid treatment, coughing and release of sputum started. On chest CT scans, in the

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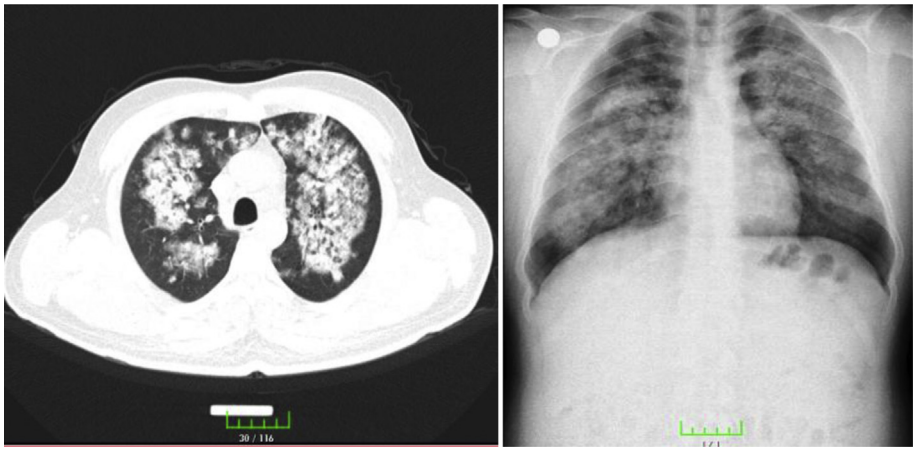

Fig. 1. Alveolar hemorrhage after treatment of ATRA.

parenchymal window, a thick-walled peripheral ground-glass cavitary lesion approximately 27 × 18 mm in size was observed in the left upper lobe. In the left lower lobe mediobazal segment, an atelactasis 7.5 × 4.5 cm in size was found, and in the right lower lobe irregular nodules from 10 mm to 16 mm in size of the area were observed (Fig. 2). Bronchoscopy was performed and bronchial lavage taken. Tuberculosis culture and galactomannan were negative. Serial galactomannan antigen tests on the patient's blood were performed. After 20 days galactomannan was positive and treatment with voriconazole was started (Table 1). One mounth later under voriconazole treatment, the left upper lobe cavitary lesion and lower lobe atelectasis were found to have significantly decreased (Fig. 3). The patient is still being monitored in our clinic.

3. Discussion

Differentiation syndrome (DS), previously known as retinoic acid syndrome, is the main life-threatening complication of therapy with differentiating agents [either trans retinoic acid (ATRA) or arsenic trioxide (ATO)]in patients with acute promyelocytic leukemia (APL) [9]. Abnormal findings in chest radiography or computerized tomography are very common during the course of DS [10]. The typical findings on a chest radiograph are interstitial infiltrates (i.e., septal lines and peribronchial cuffing, ground glass opacity) and pleural effusion. An increased cardiothoracic ratio (up to 87%) and parenchymal consolidation are also frequently encountered (47%), with or without air bronchogram [11]. Also, congestive heart failure and pneumonia in a febrile neutropenic patient should be excluded from this pattern. In such cases eco-cardiography, microbiologic isolates, patterns of fever, further response to intravenous dexamethasone or antibiotics, and the clinical and radiological course will aid in diagnosis. Notably, an association between the occurrence of DS and disseminated intravascular coagulopathy and haemorragic syndrome, including

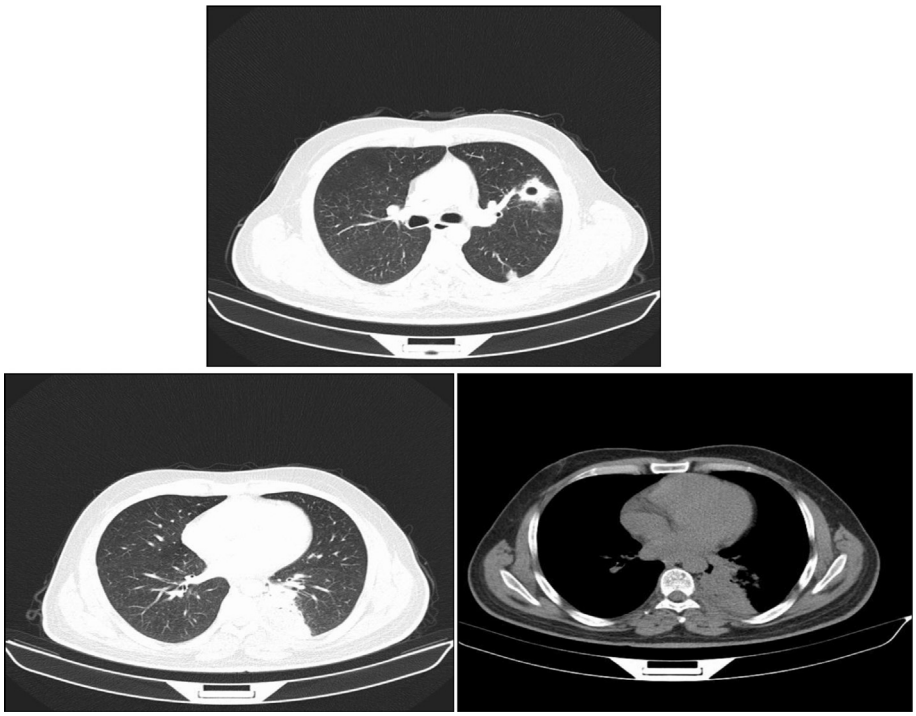

Fig. 2. Before treatment.