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COVID-19 Associated Pulmonary Fungal Ball and Arterial Thrombosis: A Rare Two Case Report

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Abstract

COVID-19 is regarded as one of the worst pandemics, which has consumed human lives. COVID-19 infection mainly affects the lungs triggering severe hypoxemic respiratory failure, also providing a nidus for superimposed bacterial and fungal infections. We report the case of a 71 years old male who presented with progressive dyspnea, fever, myalgia, haemoptisis, diagnosed with COVİD-19 related complicated with lung cavitations *Aspergillus* sp. COVID-19, to our knowledge, has rarely been associated with pulmonary aspergillomas ball with asendan aorta thrombosis.

Keywords: COVİD-19; Pulmoner aspergillosis; Thrombositosis; Fungal infections

Introduction

Coronavirus Disease 2019 (COVID-19), caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has been an emergency global public health events. Coronaviruses are a group of enveloped, nonsegmented, single stranded RNA viruses COVID-19, which may range from asymptomatic to a fatal, multi-organ disease. A dysregulated immune response not only compromises the ability of the host to resolve the viral infection, but may also predispose the individual to secondary bacterial and fungal infections. Rarely has a superinfection association of COVID-19 with pulmonary fungal ball-like aspergilloma and arterial thrombositosis been reported [1].

Case Presentation

Case 1

A 71 years old male. Past medical history of tuberculosis sequela lesion, hypertension, diabetes, hypothyroidis diagnosed with COVID-19 infection 3 days prior to admission, presented to the emergency room with progressive worsening of shortness of breath. He endorsed 3 days of subjective fevers, chills, productive cough, generalized weakness, and decreased oral intake [2]. On presentation to the emergency room, he was tachypneic to 32 breaths per minute with oxygen saturation of 83% on room air. He was immediately placed on a non-rebreather with improvement in oxygenation. Blood work showed elevated inflammatory markers with C-reactive protein: 58.3 mg/dL (0 mg/dl-5 mg/dl), Ferritin: 338 (13-150) ng/ml and Lactate Dehydrogenase (LDH): 476 (125 U/L-245 U/L), White Blood Cell (WBC): 4600 (4-10.6 10^3 µ/L), lymphocyte: 600, Hemoglobin: 12.7 gr/dl (12 g/dl-16.8 g/dl), PLT: 248000 (139000 µ/L-346000 µ/L), Sedimentation: 96 mm/saat (0 mm/hr-15 mm/hr), D-Dimer: 1650 ng/ml (0 ng/ml-500 ng/ml), Fibrinogen: 236 ng/ml (193-412), Aspartate Amino Transferase (AST): 22 (0 U/L-35 U/L), Alanine Amino Transferase (ALT): 18 (0 U/L-45 U/L), Gamma Glutamyl Transferase (GGT): 36 (12 U/L-64 U/L), Alkaline Phosphatase (ALP): 48 (40 U/L-150 U/L), Calcium: 7.9 (8.2 mg/dl-10.6 mg/dl), Albumin: 3.2 (3.5 mg/dl-5 mg/dl), Troponin: <0.01 (0.00 ng/ml-0.06 ng/ml), glycated Hemoglobin (HbA1C): 7.4 (4-6). Chest radiograph on day of admission showed bilateral peripheral infiltrates suggestive of COVID-19 lesions (Figures 1 and 2).

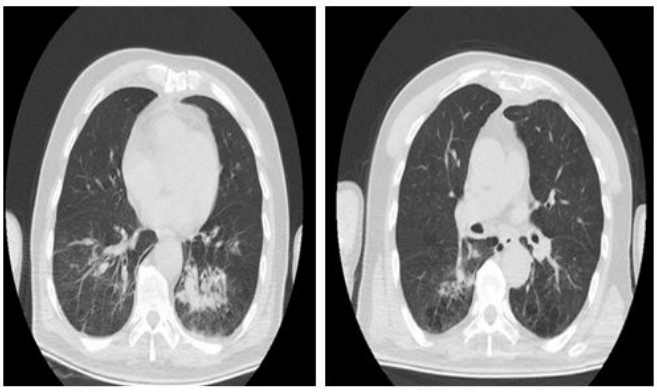

Figure 1: Computed tomography of chest axial view. Ground-glass opacities, paving pattern and patchy consolidations.

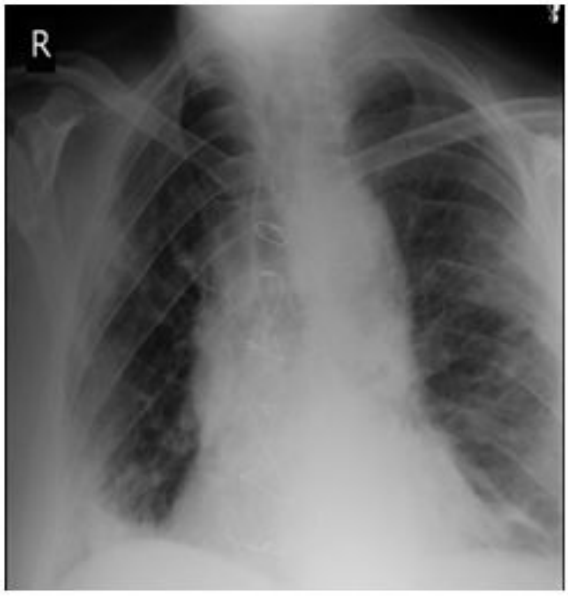

Figure 2: Chest X-Ray view. Consolidations and patch lesions.

Nasopharyngeal swab polymerase chain reaction for COVID-19 tested positive. RT-PCR test was evaluated by the Turkish ministry of health, department of microbiology reference laboratory of using BIO RAD, CFX real-time PCR system, (ABD). COVID-19 RT-PCR kit, Bioeksen (Turkey). Blood and respiratory cultures were drawn. Due to progressive respiratory distress, the patient was initially placed on high-flow nasal cannula [3]. He started on faviprevir, prednisolon based on current COVID-19 recommendations. With persistent fever spikes to 38°C, he was initiated on broad-spectrum antibiotics. After treatment he was discharged. Two weeks later despite appropriate antibiotics, the patient continued to have persistently high-grade fevers and progressively higher oxygen requirements and cough with haemopthisis. Computed tomography scan of the chest was performed 2 weeks after admission demonstrating extensive airspace disease with bilateral peripheral ground glass pattern, along with newly formed thin-walled cavitary lesions in the right middle lobe and left upper lobe (Figure 3) [4].

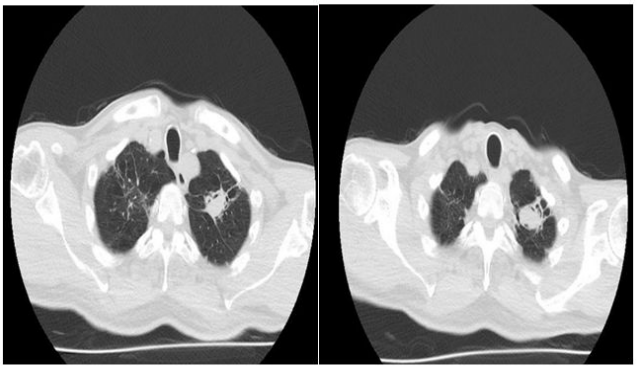

Figure 3: Computed tomography of chest axial view.

Aspergillus fungal ball within thin-walled cavities. At the exit level of the aortic arch, a hypodense thrombus in the form of a cracentric, measuring 7 mm in its widest part, was observed. Partly bronchiectasis changes were observed in both lung apices. The lesion appearance at the apex of the left lung was ~3 cm × 2 cm in length, with air values, circumferential linear extensions, fungal ball-like aspergilloma. Peribronchial thickening was observed in the lower lobe posterobasal segment of the right lung, with a minimal consolidated area with a pleural base and its neighborhood. Emphysematous and linear fibrotic changes are present in both lungs. Linear-reticular density changes were observed in the lower lobe basal segments of both lungs [5].

These cavities were occupied by fungal ball-like lesions, and galaktomannan test was positive. Without any fungal growth in sputum cultures. The patient was then started on ampiric intravenous voriconazole for pulmonary aspergilloma. Due to persistence of the COVID-19 and complicated by pulmonary fungal ball with asendan aorta thrombosis, the patient further was discharged to chest surgery clinic for operating.

Case 2

A 56-year-old male patient has known diabetes and hypertension. The COVID-19 PCR test was positive when she applied to the hospital with complaints of fever, shortness of breath, cough, and weakness. The patient, who was discharged after a week of hospitalization, reapplied to the hospital with hemoptysis approximately 2 weeks later. C-reactive protein: 7.2 mg/dL (0 mg/dl-5 mg/dl), Ferritin: 367 (13-150) ng/ml and Lactate Dehydrogenase (LDH): 185 (125 U/L-245 U/L), White Blood Cell (WBC): 8700 (4-10.6 10 µ/L), lymphocyte: 3500, Hemoglobin: 13.2 g/dl (12 g/dl-16.8 g/dl), PLT: 354000 (139000 µ/L-346000 µ/L), Sedimentation: 107 mm/hr (0 mm/hr-15 mm/hr), D-Dimer: 0.80 ng/ml (0 ng/ml-500 ng/ml), Fibrinogen: 463 ng/ml (193-412), Aspartate Amino Transferase (AST): 14 (0-35 U/L), Alanine Amino Transferase (ALT): 13(0 U/L-45 U/L), Albumin: 3.5 (3.5 mg/dl-5 mg/dl), Troponin: <0.08 (0.00 ng/ml-0.06 ng/ml), glycosylated hemoglobin (HbA1C): 79.5 (4-6). In the imaging performed, 4 cm nodular lesion in the upper lobe of the left lung, sequelae of TB and 4 cm pleural effusion on the right were evaluated. *Aspergillus fumigatus* grew in the sputum culture taken. Galactomannan test was positive. The patient was evaluated as pulmonary aspergilloma. He was referred to the thoracic surgery clinic for preoperative evaluation [6].

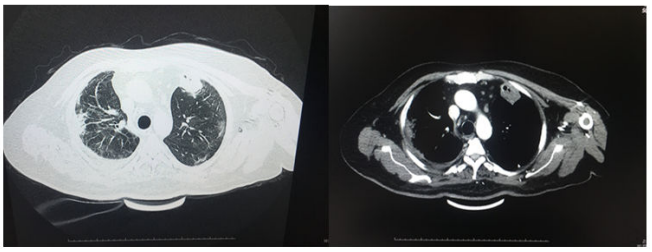

Figure 4: 4 cm nodular lesion suggesting a ball of manar on the left apex in computed tomography.

Discussion

Beta coronavirus-COVİD-19 has rapidly spread around the world, causing a global pandemic of coronavirus-19 disease. The diffuse alveolar damage with severe inflammatory exudation, COVID-19 patients always have immunosuppression with a decrease in CD4 T and CD8 T cells. Critically ill patients, especially the patients who were admitted to the intensive care unit and required mechanical ventilation, or had a longer duration of hospital stays, even as long as

50 days, were more to develop fungal co-infections. Hence, it is important to notice that COVID-19 patients can develop further fungal infections during the middle and latter stages of this disease, especially severely ill ones. An increasing risk for COVID-19 patients is represented by the occurrence of co-infections and superinfections, which may deteriorate the clinical picture. Indeed, bacterial, fungal, and viral infections have been detected in COVID-19 patients, although at a low incidence. Risk factors described in CAPA patients include older age, lymphopenia, chronic respiratory diseases, corticosteroid therapy, antimicrobial therapy, or cytokine storm. In a study due to the severity of the clinical situation, thoracic CT scans were performed only in 5/8 (62.5%) patients and globally in 31/96 patients (32.3%). Thoracic CT scans may be difficult to assess in patients with ARDS associated COVID-19, usually showing ground-glass opacities, a crazy-paving pattern and patchy consolidations [7]. Typical COVID-19 radiological findings are usually present in COVID 19 related Aspergillosis cases, sometimes with consolidating peribronchial patterns. However, classic findings of angioinvasive fungal infection are anecdotic. Other methods to date include attempting to recover *Aspergillus* spp. on culture media of Bronchoalveolar Fluid (BALF) and tracheal aspirate, as well as utilizing serologic biomarker testing such as the conventional Galactomannan (GM), tracheal aspirate, and serum specimens. Other diagnostic tests that may prove useful also include *Aspergillus* PCR, serum (1,3)-β-d-glucan. Histological examination of lungs coming from seven deceased patients affected by COVID-19 was compared with that obtained from patients who died from influenza infection and uninfected control lungs. Vascular features were instead distinctive of COVID-19 and consisted of severe endothelial injury, widespread thrombosis with microangiopathy, alveolar capillary microthrombi, and neoangiogenesis.

Conclusion

Severe COVID-19 is associated with a dysregulated immune response that may not only impact the clinical deterioration of patients, but also modulate the susceptibility to secondary infections, for instance by impairing host antifungal defences and increasing the risk of fungal infection. Pulmonary Aspergillosis as a superimposed infection in patients with COVID-19 and arterial thrombositosis are rare entity. In this study we aimed to remain about aware of COVID-19 and fungal superinfections.

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