Case report

Leptospirosis and COVID-19 Co-Infection Manifesting as an Enhanced Thrombotic Phenomenon

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Abstract

Clinical manifestations of coronavirus disease 2019 (COVID-19) range from mild self-limiting illness to multi-organ dysfunction and acute respiratory distress syndrome (ARDS). Also, leptospirosis manifests as mild or asymptomatic infection, and only a small number of patients progress to ARDS and develop systemic manifestation. COVID-19 and leptospira co-infection can have fatal outcomes because of augmented pathophysiological manifestations of both the disease. We here describe a case of leptospira and COVID-19 coinfection in a patient who had poor results due to multi-organ involvement.

Keywords: COVID-19, Multi-organ Dysfunction, Leptospirosis

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Introduction

Leptospirosis is one of the leading causes of intensive care admission in developing countries (1). Leptospirosis has a biphasic presentation. The Anicteric phase lasts for 3-7 days and is characterized by fever, anorexia, headache, myalgia, abdominal pain, conjunctival congestion, or bleeding. It is primarily self-limiting, with 90% of the patient recovering from this illness. In the remaining 10% of the patients, the disease progress to a more lethal form called Weil's disease (2). COVID-19 and leptospira co-infection can have fatal outcomes which are discussed here.

Case Report

A 58-year-old male was admitted to COVID ICU after testing positive for the disease with 7 days history of cough, fever, shortness of breath, and jaundice. He was known to be diabetic and hypertensive but without any history of prior lung disease. Initial investigation showed the presence of thrombocytopenia (platelet-50000/cu mm), altered coagulation profile (INR-1.68, Aptt- 55.9 seconds, D-dimer- 14 mcg/ml), raised inflammatory markers (IL-6 -85 PG/ml, CRP - 6.8 mg/dl) and impaired renal as well as liver function (urea-71mg/dl, creatinine1.1 mg/dl, bilirubin- 7 mg/dl). Despite appropriate therapy, the patient's respiratory, hemodynamic, and laboratory status worsened gradually with increasing oxygen requirement, inotropic support, altered coagulation profile, and renal and liver function. He tested positive for ELISA for leptospira antigen when evaluated for tropical diseases endemic in the region. Chest radiograph showed the presence of wedge-shaped consolidation in the right middle lobe of the lung. (Fig.

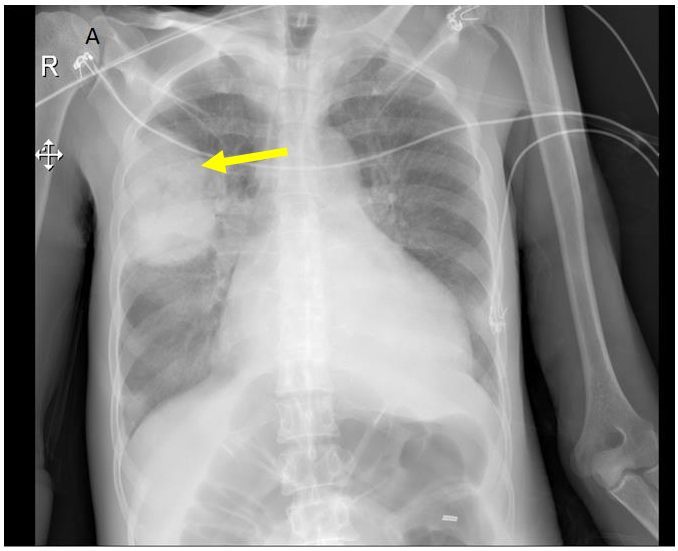

Figure 1. Chest X-ray showing the presence of wedge-shaped opacity of right middle lobe (Yellow Arrow).

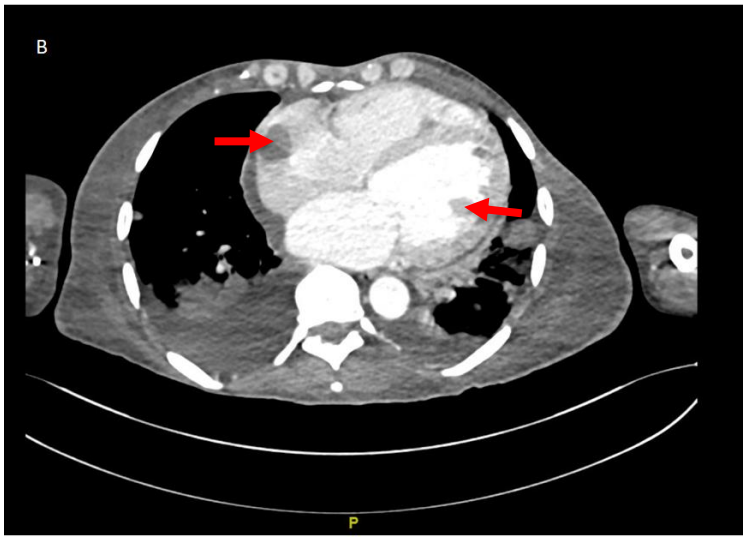

Figure 2. Transverse Section of CECT chest showing the presence of thrombus in the right atrium and left ventricle (Red Arrow).

1) Contrast-enhanced computed tomography of the chest and abdomen was done, which showed the presence of multiple thrombi in the right and left ventricle and the right atrium. (Fig. 2) It also showed the presence of bilateral pleural effusion with a wedge-shaped zone of pulmonary infarction of the right middle lobe. (Fig. 3) The possible mechanism could be COVID-19, and Leptospirosis induced disseminated intravascular coagulation (DIC), leading to thrombi formation in various heart chambers and their dissemination in the pulmonary vasculature, causing lung infarction. Gradually, there was multiorgan involvement with DIC, acute kidney injury (AKI), and liver dysfunction. Later, the patient manifested with upper gastrointestinal bleeding and expired due to refractory shock and hypoxemia.

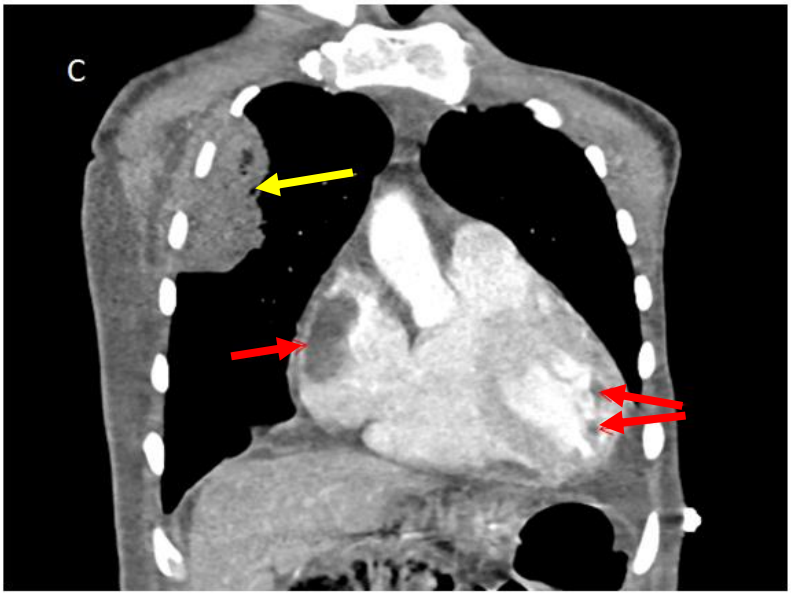

Figure 3. Saggital Section of CECT chest and abdomen showing the presence of multiple intracardiac thrombi (Red Arrow) along with right lung middle lobe infarction (Yellow Arrow).

Discussion

Leptospirosis is characterized by increasing jaundice, AKI, thrombocytopenia, DIC, acute respiratory distress syndrome, and multi-organ dysfunction (1-3). A sudden episode of hemoptysis in stable patients of Leptospirosis is a predictor of poor outcomes, with DIC being a contributing factor. Sometimes, severe upper gastrointestinal bleeding may occur as a preterminal event. Our patient had the presence of thrombocytopenia, AKI, DIC, and an acute episode of upper gastrointestinal bleeding.

On the other hand, COVID-19 clinical manifestation varies from mild self-limiting illness to severe disease. Mild cases present with fever, cough, myalgia, and loss of taste and smell. Severe cases may manifest with dyspnoea, hypoxia, respiratory failure with acute respiratory distress syndrome, shock, and multi-organ failure (4). The endothelial damage due to the raised level of circulating cytokines and microcirculation dysfunctions leads to the development of microthrombi and DIC (5).

In our case, the aggravation pathophysiological manifestation of COVID-19 and leptospira coinfection resulted in enhanced disseminated intravascular coagulation leading to thrombi formation in various heart chambers and their dissemination in the pulmonary vasculature, causing lung infarction.

Conclusion

The leptospira and COVID-19 co-infection can have a fatal outcome if not diagnosed and treated early. The pathophysiological alteration due to Leptospira and COVID-19 can lead to the augmented manifestation of both diseases resulting in AKI, liver dysfunction, ARDS, and DIC. Therefore, critical care clinicians must diagnose such rare co-infections to start adequate treatment on time before any irreversible injury sets in.

Acknowledgment

None.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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