Covid-19 Infection Complicated with Aortic Thrombosis: A Case Report

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Abstract
Thromboembolic complications are well recognized events in Covid-19 infection. Most of the case fatalities are due to this event. Although any blood vessels may be involved, larger vessels are less commonly involved in this process. Here we describe a case of thrombosis of the arch of the aorta in association with Covid-19 infection in a hospitalized patient. The aim was to ensure timely identification and therapeutic intervention to prevent distal thromboembolic sequelae. [Journal of National Institute of Neurosciences Bangladesh, July 2020;6(2): 143-145]

Keywords: Covid-19; thromboembolism; LMW heparin; aortic arch

Introduction
Since the outbreak of Covid-19 pandemic, increasing evidence suggests that infected patient presents with a high incidence of thrombotic complications such as deep vein thrombosis¹, pulmonary embolism² or microvascular thrombosis³ which leads to limb ischemia, myocardial ischemia, ischemic stroke, mesenteric ischemia. Aortic thrombosis⁴ is an uncommon but not rare complication of Covid-19 infection. It has been reported that the incidence of thrombotic event is as high as 31% cases⁵ among the critically ill patient. Hypercoagulability and endothelitis caused by Covid-19 seems to be associated with all these complications⁶. Here we describe a case of thrombosis of the arch of the aorta in association with Covid-19 infection in a hospitalized patient. The aim was to ensure timely identification and therapeutic intervention to prevent distal thromboembolic sequelae.

Case Presentation
A 38 yrs old non-diabetic, normotensive, non-smoker, male from Dhaka got admitted into Kurmitola General Hospital a dedicated Covid hospital at Dhaka, with history of fever, shortness of breath, cough and a progressive deterioration of oxygen saturation for 4 days. He gave H/O diarrheal illness 7 days back. On admission he was found to have dyspnea with a respiratory rate of 26 breaths/min, pulse 103/min, BP was 120/75mmhg, temperature 37.2⁰c & O2 saturation 88% without O2 which was corrected immediately with O2 supplement of 5L/min. After admission he became more dyspnic & O2 requirement gradually increased & was settled to O2 10L/min. All routine test including RT-PCR for Covid-19 were sent & a HRCT chest was done. His
initial treatment includes, complete bed rest, O₂ inhalation through face mask to maintain O₂ saturation >95%, I/V fluids, I/V antibiotics and s/c LMW heparin for prophylaxis of thromboembolism. The patient was maintaining his O₂ saturation with a stable condition. RT-PCR for Covid-19 was positive. Other laboratory reports revealed, neutrophil leukocytosis with mild elevation of ESR Hb-13.8 gm/dl, ESR-31 in 1st hour, TC - 13000/cumm, neutrophil - 88%, lymphocyte- 9.3% and platelet count - 174000/cumm), normal Urine R/E, RBS - 7.3mmol/l, ALT-190 IU, (S. creatinine-0.6mg/dl). CRP (12 mg/dL), serum ferritin (913) and LDH (579 U/L), with a normal D-dimer level (D-dimer-0.11 ng/L). HRCT chest revealed: Bilateral ground glass opacities, advance stages of pneumonitis: CT score-4 & Aortic thrombosis (arch of the aorta).

With lack of further laboratory facilities for thrombophilia screening the patient was ultimately diagnosed as a case of Covid-19 pneumonia (severe) with thrombosis of the arch of the aorta. The patient was managed in the hospital with: complete bed rest, O₂ inhalation, intravenous fluid, intravenous broad spectrum antibiotics, s/c LMW heparin in therapeutic dose with oral anticoagulant and inj. Remdisivir. The patient was closely monitored for any systemic embolic events. His condition improved gradually from 5th day onward without developing any further complications. The patient was discharged with a negative Covid19 report and without any supplemental O₂. He informed over telephone that he was doing well during writing the report and was advised for a follow up HRCT chest.

**Discussion**

To the best author’s of knowledge this is the first reported case of aortic thrombosis (arch of the aorta) in Covid-19 infection in Bangladesh. Although prognosis of hospitalized Covid-19 patient is determined by the extent of pulmonary involvement, vascular thrombosis also greatly influences their outcome. The pathophysiology of thrombosis here is not well elucidated, however recent data suggest the existence of a hypercoagulable state in patients with Covid-19 patients. Excessive inflammation, platelet activation, endothelial dysfunction & stasis all contributes to the event of thrombosis⁷. Alternatively it was suggested that formation and polymerization of fibrin are responsible for this hypercoagulability⁸. Viral induced endothelitis has also been implicated in a recent study⁹. Therefore thromboprophylactic measures have been recommended in the guideline to prevent thromboembolism⁹,¹⁰,¹¹. Finally outcome of these complicated Covid-19 cases may vary with or without systemic sequelae.

**Conclusion**

Early diagnosis and therapeutic intervention of covid-19 cases complicated by arterial and venous thrombosis is highly recommended to save the life of the patient as many of these complicated cases undergoes a catastrophic outcome.

**References**