Acute Pulmonary Embolism post COVID-19 pneumonia – a case report

Abstract

COVID-19 has been associated with multiple venous thromboembolism events such as pulmonary embolism and deep vein thrombosis. Here we report a 64-year male with COVID-19 pneumonia who developed pulmonary thromboembolism following the COVID illness. This patient developed VTE complication in spite of receiving anti-coagulation therapy during admission. This case brings out the need for evidence-based post-discharge VTE prophylaxis approach and guidelines in patients who recover from COVID-19.

Keywords: COVID-19; VTE; CT Pulmonary Angiogram

Introduction

The hypercoagulability state seen in COVID-19 has been associated with thromboembolic complications. [1] The VTE risk appears highest in those with critical care admission. [2] No clear-cut evidence-based recommendations exist for prevention of venous thromboembolism events. Usually institutional protocol provides thromboprophylaxis to COVID-19 patients during admission. We report a case of thromboembolism complicationfollowing COVID-19.

Case Report

A 64-year-old male presented with breathlessness for the past 20 minutes to the ER. The breathlessness was sudden in onset, not aggravated on exertion. No H/o fever, cough or cold. H/o chest pain, retrosternal, non-radiating, pleuritic type of pain was present. No h/o palpitations Patient had any prior co-morbidity. Patient gives H/o of admission 1 month ago for COVID-19infection and confined to bed for 13 days. CT chest taken during the COVID infection showed CORADS-5 with 10-20% lung involvement. His D-Dimer was 1652 ng/dL (Normal <500ng/dL) during the course of COVID illness and he received injection Heparin 5000 units twice daily for five days. A follow-up CT at 10 days later revealed resolution of COVID changes (Lung involvement - 5%).

On examination patient was conscious, oriented and afebrile. He had tachycardia and tachypnoea. However, saturation was within normal limits. BP was 130/80mmHg involvement and). ECG showed sinus tachycardia.With suspicion of Pulmonary Embolism, CT Pulmonary Angiogram was done and it showed evidence of pulmonary artery embolism in the main right pulmonary artery extending through the bifurcation into the left pulmonary artery and into the interlobar branches. Patient underwent thrombolysis with Unfractioned Heparin and switched to Injection Low Molecular Weight Heparin 60mg twice daily. D-dimer was 10000ng/dL (Normal <500ng/dL). Patient improved following therapy and he was switched over to oral anti-coagulant dabigatran after 5 days of parenteral anticoagulation. Patient was discharged and advised to follow-up in out-patient department.

Discussion

COVID-19 pandemic has been ravaging the world for months already and now post infection complications are beginning to be found. Individuals with COVID-19 may have a number of complex and varied coagulation abnormalities (in the direction of an underlying hypercoagulable state). [2] The predominant coagulation abnormalities in patients with COVID-19 suggest a hypercoagulable state and are consistent with uncontrolled clinical observations of an increased risk of venous thromboembolism. [3] It has been named as a COVID-19 associated coagulopathy (CAC) or thrombo-inflammation. [4,5] The pathogenesis of the hypercoagulability is still not understood. However, an autopsy study that compared pulmonary pathology from seven individuals who died of COVID-19 found a severe endothelial injury (endotheliitis), widespread thrombosis with microangiopathy and alveolar capillary microthrombi, and increased angiogenesis, all of which were significantly more prominent in the lungs of the patients who died of COVID-19. Endothelial injury found in these patients is a hallmark of Virchow's triad. [6] However the development of the thromboembolism raises many questions about the incidence, risk factors for developing the complication and the prevention and management strategies for the same. Similar cases have been reported elsewhere. In a large study that involved over 3000 individuals admitted to the hospital, most of whom received prophylactic-dose anticoagulation, risk factors for VTE on multivariate analysis were older age, male sex, Hispanic ethnicity, coronary artery disease, prior myocardial infarction, and higher D-dimer (>500 ng/mL) at hospital presentation. [1] VTE was associated with an increased mortality rate (adjusted hazard ratio [HR], 1.37; 95% CI 1.02-1.86).

A case series of COVID-19 patients with VTE found VTE events more likely in patients admitted in ICU, sometimes despite receiving anti-coagulant therapy in ICU. Many reports of DVT have also been noted during and following the COVID infection. Patients who developed VTE were found to have high D-dimer, high fibrinogen, normal or mildly prolonged PT and normal or decreased platelet count. [7] However, these laboratory derangements do not provide sufficient incentive to intervene. Currently, no high-quality studies exist to dictate streamlined management strategies. Hence,clinicians resort to institutional practices which may vary in aggressiveness in approach. Two new autopsy studies, together including a total of 33 individuals who died of COVID-19, have revealed common causes of death to be pneumonia and pulmonary embolism. Hence prevention of VTE will be very essential in bringing down the mortality of COVID-19.

Conclusion

While, thromboembolic complications have been found in patients with COVID-19, there still exists no proven preventive therapy for the same. Clinical trials to establish must be carried out to find the same.

References

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