Case report

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TANAFFOS

Extensive Left Ventricular Thrombosis after Recovery from COVID-19: A Case Report

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Coagulation disorder is common among COVID-19 patients, particularly those afflicted with the severe form of the disease. Manifestations of thrombotic complications such as pulmonary embolism, deep vein thrombosis (DVT), ischemic stroke, cardiac infarction and arterial embolism is high in COVID-19 patients which have been assessed in various studies (5).It seems that COVID-19-related coagulopathy is a combination of low-grade DIC and pulmonary thrombotic microangiopathy which can have a significant effect on organ dysfunction in patients with severe COVID-19 (6). Numerous complications have been reported following COVID-19 disease. Although the disease often improves after going through various clinical phases, some people have short-term or long-term complications including coagulationdisorders.
This case report presents an unusual instance of extensive left ventricular thrombosis in a patient who had recently recovered from COVID-19, despite being on anticoagulant prophylaxis.

Keywords: Thrombosis; COVID-19; Anti-Coagulant prophylaxis

INTRODUCTION

The appearance of COVID-19 arising from the infection with the new coronavirus rapidly culminated universal. Although many endeavors have been done, it is still associated with a high mortality rate (1).

The COVID-19 pandemic has been associated with various complications, including coagulation disorders that can persist even after recovery from the acute infection. The case highlights the potential for severe thrombotic complications in post-COVID patients and underscores the importance of vigilant follow-up and appropriate anticoagulation management. It also raises questions about the optimal thromboprophylaxis strategies in patients recovering from COVID-19, particularly those with pre-existing cardiovascular risk factors.

CASE SUMMAERIES

The patient was a 59-year-old man with a past medical history of diabetes, hypertension, ischemic heart disease, and hyperlipidemia who was referred to Labbafinejhad

Hospital in Tehran. His chief complaint was chest pain and exacerbated fatigue. He had been diagnosed with COVID-19 and hospitalized for treatment in the past three weeks. The patient was treated with Remdesivir (200 mg IVinf in day one and 100 mg IVinf from the second day for 5 days) and intravenous pulse steroid therapy (methylprednisolone 1 g IV daily for three days followed by 60 mg IV twice daily). He had been discharged from the hospital two weeks before the referral. The patient was re-referred with fatigue and increased dyspnea.

Patient examination showed BP:130/70 mmHg, PR:110, and O_2Sat:83%. In cardiac auscultation, s1 and s2 were normal, and s4 sound was heard. In addition, pulmonary auscultation was without rales on both sides of the lung. The patient's medications included losartan, atorvastatin, metformin, ASA 80mg, and apixaban 2.5mg. The patient had no history of smoking or drug Abuse.

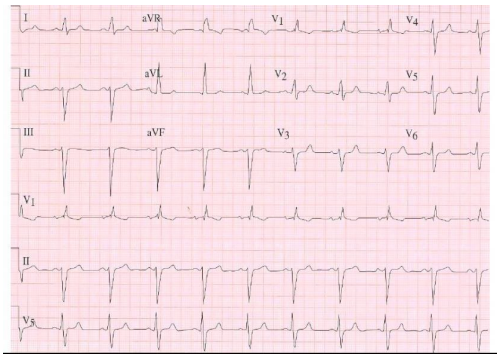

Figure 1. The first EKG at the hospitalization

Electrocardiography and Echocardiography were done following patient admission, the results of which are presented below: EF: 55%, moderate-severe AI, multiple mobile veg in LV attached to apex and apical septal wall, suggestive of wedge or clot (Figure 1 and 2).

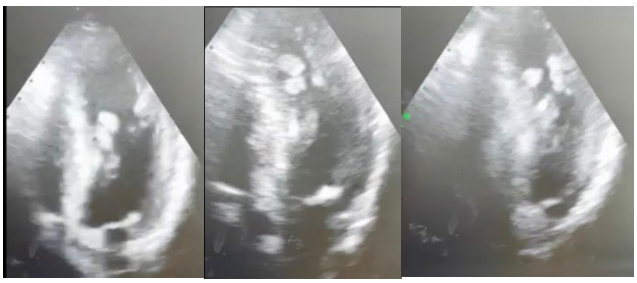

Figure 2. The first Echocardiography at the hospitalization

Heparin infusion (2000U IV stat and then 1500U hourly) was initiated for the patient after the echocardiographic observation. Laboratory Tests were as presented in Table 1.

Table 1. Patient's Laboratory Tests

HB	11.1	**CRP**	8
WBC	9.2	**Calcium**	8.7
Neutrophils	86%	**Urea**	93
Platelets	145	**Creatinine**	1.09
ESR	39	**Uric acid**	4.8
AST	29	**Phosphorus**	6.7
ALT	19	**D dimer**	668
LDH	581	**Troponin**	0.049
Mg	2.2	**INR**	1.8
Alb	2.9	**PTT**	55

Wide QRS was detected in the second EKG (in the second day after admission), for which Amiodarone was administered at a dose of 150 mg intravenously for 10 minutes, followed by an infusion of 1 mg/min for 6 hours and then an infusion of 0.5 mg/min. Concerning the patient's illness and fever, wide-spectrum antibiotics (imipenem, vancomycin, and ciprofloxacin) were prescribed.

PVCs appearing in a pattern of bigeminy was observed in the patient's EKG in the course of treatment (third day). In addition, the patient experienced one episode of sustained ventricular tachycardia (same day) for which he was prescribed intravenous amiodarone (150 mg stat, a 1 mg/min infusion for six hours, followed by an infusion at 0.5 mg/min for 18 hours).

Regarding persistent arrhythmias and large wedges (27mm) in the heart, the patient received a consultation for cardiac surgery to remove wedges through surgery and intravenous infusion of heparin (900 -1300unit/hour to maintain PTT between 60 to 90 seconds)

The patient was sent for cardiac surgery After 5 days. Another echocardiography was done for the patient while preparing him for the surgery. The patient was returned to the ward due to the reduction in the size of cardiac masses (size:14mm) and became stable after three weeks of intravenous infusion of heparin. Then, he received oral

Apixaban (5mgr/ BID) for up to two months and then completely recovered.

DISCUSSION

The emergence of COVID-19 arising from the infection with the new coronavirus rapidly culminated in a pandemic. Despite many efforts, it is still associated with a high mortality rate (1). Even though the disease is ameliorated after passing various clinical phases, different short-term and long-term complications in different organs have been reported because of the disease. In this regard, some of the most frequently reported complications include lung fibrosis, secondary bacterial or fungal infections, gastrointestinal complications, as well as cardiac (myocarditis and pericarditis), and vascular complications such as embolism of pulmonary arteries (2).

There is increasing evidence of COVID-19-related coagulation disorders that expose patients to thrombotic complications. These complications are among the causes of the disease's poor prognosis and are associated with a high mortality rate (3). Viral infection complications in patients afflicted with the severe phase of the disease lead to the activation of various systemic inflammatory and coagulation responses which are vital for the host's immune system. Stimulation of the host's inflammatory responses will also lead to increased production of pre-inflammatory cytokines which has pleiotropic effects such as activation of the coagulation cascade, and if not controlled, can result in coagulopathy (4).

Coagulation disorder is common among COVID-19 patients, particularly those afflicted with the severe form of the disease. Manifestations of thrombotic complications such as pulmonary embolism, deep vein thrombosis (DVT), ischemic stroke, cardiac infarction, and arterial embolism is high in COVID-19 patients which have been assessed in various studies (5). The primary clinical presentation of coagulopathy in COVID-19 is the failure of organs, while hemorrhagic complications and drainages are less common.

Variations of hemostatic markers in laboratory analyses such as D-Dimer, fibrin, and Fibrinogen Degradation Products (FDP) show that coagulopathy basis probably lies in fibrin products. In the final phase of COVID-19, there is an increase in levels of markers related to fibrin (FDP, D-Dimer) in all mortality cases. It indicates the activation of coagulation cascade or secondary hyperfibrinolysis in these patients.

It seems that COVID-19-related coagulopathy is a combination of low-grade DIC and pulmonary thrombotic microangiopathy which can have a significant effect on organ dysfunction in patients with severe COVID-19 (6). In a study conducted to evaluate the relationship between coagulopathy and COVID-19 in Shanghai, COVID-19 patients were divided into two relatively mild (n=277) and severe (n=26) groups. According to the results, the mean coagulation parameters were higher in patients with the severe form of the disease, compared to patients in the mild group (7).

Attention to the risk of venous thromboembolism (VTE) in COVID-19 patients is also of paramount importance. Prolonged immobility due to illness, dehydration, acute inflammatory response, cardiovascular risk factors (e.g., hypertension, obesity, and diabetes) or cardiovascular diseases (e.g., coronary artery disease, history of ischemic attack or peripheral artery disease), and a previous history of VTE are among the most common factors that can potentially increase VTE risk in COVID-19 patients. Moreover, damage to or activation of endothelial dysfunction through virus binding to ACE2 receptors can increase VTE risk (8). Patients with severe COVID-19 may experience increased blood density due to the release of large amounts of inflammatory mediators, immunoglobulins, and hormones. Furthermore, mechanical ventilation, a central venous catheter (CVC), and surgery can instigate vascular endothelial dysfunction. Association of all factors can lead to DVT manifestation or PE-related mortality due to clot movement. Therefore, drug thromboprophylaxis is mandatory in COVID-19 patients in case of VTE risk (9).

Presented reports of PE in COVID-19 patients were indicative of high disproportionate manifestation of vascular thromboembolism and arterial embolism in

patients. Manifestation of thromboembolism complication is between 5-15% in patients afflicted with the severe form of COVID-19 (10). Primary cohort studies have shown that the manifestation of thromboembolism complication is between 35-45% in COVID-19 patients (11).

CONCLUSION

COVID-19 can lead to significant coagulation disorders, including thrombotic complications that may persist even after recovery from the acute infection. This case highlights the importance of vigilant monitoring and appropriate anticoagulation management in post-COVID patients, especially those with pre-existing cardiovascular risk factors. Early diagnosis of coagulation abnormalities, identification of high-risk patients, optimization of thromboprophylaxis regimens, and consideration of anti-inflammatory properties of anticoagulants are crucial aspects in managing these patients. Ongoing research and clinical awareness are needed to better understand and manage the complex coagulopathy associated with COVID-19, potentially reducing mortality and improving long-term outcomes for recovered patients.

Ethical Considerations

The study protocol was in compliance with the Declaration of Helsinki (1989 revision). The participant had given consent to publish the clinical results of the study.

Consent for Publication

Written informed consent was obtained from the patient to publish this report following the journal's patient consent policy.

Acknowledgment

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