A Case of COVID-19 Accompanied by ST-Segment Elevation Myocardial Infarction and Phlegmasia Cerulea Dolens

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1. Abstract
Deep vein thrombosis, pulmonary thromboembolism, myocardial infarction, and systemic arterial thrombosis have been reported recently due to Coronavirus 2019 [COVID-19] disease-induced hypercoagulability [6]. Phlegmasia cerulea dolens [FSD] is a serious condition with a 25–40% mortality rate. It is a rare and severe complication of deep vein thrombosis [4]. In this case report, we aim to present the treatment course of an 84-year-old male patient diagnosed with COVID-19 infection and ST-segment elevation myocardial infarction with FSD.

2. Introduction
According to current coagulopathy theories in the Coronavirus 2019 [COVID-19] epidemic, endothelial dysfunction, hypercoagulability, and venous stasis formation play a primary role in advancing the inflammatory process while cytokine storms simultaneously trigger the formation of diffuse thrombosis [1,2]. Phlegmasia cerulea dolens [FSD] is a rare [less than 1%] clinical form of venous gangrene that affects both superficial and deep veins. It is a severe complication of deep vein thrombosis [4]. Females are more likely to be affected. Cyanosis, coldness, pain, and edema in the lower limb are symptoms of severe venous obstruction. Compartment syndrome is caused by a progressive deterioration of the oncotic and hydrostatic pressure balance and fluid accumulation in the interstitial space, which compresses the arterial system and causes ischemia [5].

Diffuse thrombosis, which forms when the coagulation cascade in COVID-19 patients is disrupted, can cause acute coronary syndrome, affecting the arterial system and putting the patient at risk for coronary artery disease [3]. COVID-19 can sometimes manifest as an ST-elevation myocardial infarction [STEMI] as the first symptom. Furthermore, chest pain is inconsistent, and one third of coronary angiographic imaging fails to detect the underlying cause. Secondary causes, such as hypoxia, cytokine storm, coronary artery spasm, and respiratory distress, are attributed to troponin elevation and electrocardiography [ECG] changes in these cases [7,8]. Given the risk of microemboli and endothelial damage in the venous and arterial systems from COVID-19 infection, which is frequently associated with coagulation disorders, an effective dose of antiaggregant and anticoagulant therapy have been included in the treatment algorithm. These treatment strategies are associated with a low mortality rate.

3. Case Presentation
An 84-year-old male patient with a history of diabetes and hypertension was admitted to the emergency department with complaints of new-onset epigastric discomfort and pain, swelling, and bruising in the right lower limb. The patient had also been experiencing a dry cough and a fever that carried on for a week. The patient’s vital signs in the emergency room were as follows: respiratory rate of 17/minute, oxygen saturation of 91% in room air, heart rate of 107 beats/minute, blood pressure of 110/70 mm/ Hg, and a temperature of 37.6 ºC. During physical examination, widespread edema was observed in the right lower limb from the groin to the soles of the feet. Additionally, the skin color was cyanotic and severely painful on palpation. The capillary filling was also prolonged. The patient’s femoral pulse was palpable. However, the distal pulse was not palpable. The 12-lead electrocardiography [ECG] performed on the patient as found to be normal. An ECG and a troponin I repetitions were planned at appropriate intervals for the patient. Doppler ultrasonography revealed thrombus formation in the deep veins, including the femoral and popliteal veins. Normal systolic peak velocity and spectral waveforms were
observed in the femoral and popliteal arteries.

Since the calf was stiff and painful, the patient’s range of motion was limited. Other physical examination findings were evaluated and found to be normal. A plain chest X-ray and thorax computed tomography [CT] were performed due to the fact that the patient’s dry cough and fever had continued over the course of a week. A nasopharyngeal swab was taken for reverse-transcriptase polymerase chain reaction [RT-PCR] test in the patient, as bilateral peripheral ground-glass densities were seen on thorax CT scan (Figure 1). The patient was experiencing ongoing pressure-like pain in the epigastric region, and the 12-lead ECG was repeated; ST-segment elevation was detected in the inferior leads. In laboratory tests, D-dimer levels were 8703 µg/L, and troponinI levels were 390 ng/L. C-reactive protein, ferritin, and procalcitonin levels were also high.

Appropriate doses of acetylsalicylic acid P2Y12 inhibitor and low molecular weight heparin were rapidly loaded for the patient. The patient was taken to the angiography laboratory for primary percutaneous coronary intervention. While no pathology was observed in the left coronary system in the angiographic imaging of the patient, an acute thrombosed lesion was detected in the right coronary artery [RCA] proximal segment. An appropriately sized drug-eluting stent was directly implanted in the RCA proximal segment, and TIMI-3 flow was provided (Figure 2). In the same session, a temporary filter was placed at the infrarenal level of the inferior vena cava through the 6 French size sheath placed on the left femoral vein (Figure 3). The patient was placed in the prone position under the scope, and ultrasound-guided popliteal vein access was obtained using Seldinger’s technique. A guidewire was placed in the right popliteal vein after the lumen was confirmed via contrast injection. With the help of a special catheter at the popliteal vein level, the patient’s existing thrombus load of the was initially mechanically fragmented with the pharmacomechanical thrombectomy method (Figure 4).

Meanwhile, negative aspiration was used to prevent the fragmented thrombi from becoming proximal. Subsequently, 10 milligrams of tissue plasminogen activator was administered to the popliteal vein. After completing the procedure, the patient was transferred to the intensive care unit, and anticoagulant and antiaggregant treatment was continued. In intensive care follow-ups, the patient’s hemodynamics were stable, and he was no longer experiencing epigastric pain. Distal pulses were detected in the right lower limb, skin color returned to normal, and pain and edema regressed (Figure 5 and 6). A multidisciplinary approach was taken with the infectious diseases department in order to treat COVID-19 of the patient, whose RT-PCR test was positive. Warfarin and dual antiplatelet therapy were intended for use for 3-6 months as a medical treatment, the diameter difference between his legs disappeared, and the cough and fever were no longer present. After reaching the therapeutic INR value, the patient was discharged following a complete recovery.
Figure 5: Pharmacomechanical thrombectomy procedure performed through the right popliteal vein under fluoroscopy while the patient is prone.

Figure 6: A) Widespread edema and cyanosis in the right lower limb before the procedure. B) Regression in edema and cyanosis after the procedure.

4. Discussion

In this case report, we observed that COVID-19 affects the arteriovenous system in patients by causing hypercoagulation. Cytokine storm, triggered by proinflammatory processes, causes coagulopathy and causes severe complications by forming thrombus in many organ systems [6]. Along with the extreme increase in secondary cardiovascular system pathologies due to the COVID-19 pandemic, there is a decrease in hospital admissions due to the fear of getting infected with the coronavirus. A significant increase in mortality is observed with the delay in access to the hospital and accompanying additional complications. It is important to consider acute coronary syndromes in the differential diagnosis of patients with COVID-19 when high-sensitivity troponins are positive [3]. Taking a simple 12-lead ECG can prevent us from missing a diagnosis requiring urgent cardiovascular intervention. In this case, we detected an acute cardiac pathology by taking a 12-lead ECG in a patient who applied to our emergency department due to non-cardiac symptoms. It has been stated that decreased organ perfusion, abnormal complement activity, proinflammatory processes, low platelet, and high D-dimer levels cause hypercoagulation and increased mortality and morbidity in the COVID-19 epidemic. FSD is a rare complication in which mortality and morbidity are significantly reduced by rapid diagnosis and treatment of the pathological condition accompanied by acute massive venous thrombus. It has been revealed that predisposing risk factors such as cancer, pregnancy, trauma, surgical operations, venous catheter interventions, genetic predisposition, and infections increase the incidence of deep vein thrombosis [9]. FSD should be considered in the differential diagnosis of these patient groups who present clinically with sudden and severe leg pain, cyanosis, compartment syndrome, venous gangrene, increased leg diameter difference, progressive pain, and swelling [10,11]. With rapid diagnosis and treatment, heparinization, use of thrombolytic, and, if necessary, mechanical thrombectomy methods, the risk of death can be reduced by 60% and amputation by 50% [12].

5. Conclusion

The COVID-19 pandemic involves the cardiovascular system, with extensive thrombi accompanied by a coagulation disorder [13]. Positive effects of rapid diagnosis and treatment on mortality and morbidity have been reported in diseases with extensive thrombus in the vascular system, such as acute coronary syndromes and FSD secondary to COVID-19 [14]. In this case report, we aim to contribute to the patients' treatment in the co-existence of FSD and ST-segment elevation myocardial infarction, which is rarely seen in the literature.

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