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Case report

Splenic infarct in a COVID-19 patient under anticoagulant therapy with normal D-dimer levels

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ABSTRACT

Background: Many studies have shown that COVID-19 can progress with coagulopathy and multisystemic thrombotic events. We report a patient who presented with abdominal pain after COVID-19 and was found to have splenic infarction (SI) concomitant with acute myocardial infarctus (MI) under anticoagulant treatment.
Case presentation: A 45-year-old man was admitted to the emergency department with left-sided abdominal pain radiating through to his back persisting for one day. He had COVID-19 PCR positivity nine days ago. After seven days of hospitalization due to COVID-19 pneumonia, he had been discharged with low-molecular-weight heparin (LMWH). Abdominal computerized tomography (CT) showed SI. His ECG and laboratory parameters were normal except for 17.2 × 10∧3/μL leukocytosis. The anticoagulant drug dose that he was taking was increased to 2 × 0.6 mL during hospitalization. He described new-onset chest pain during follow-up. Acute anterior MI was detected on ECG. Successful percutaneous coronary angiography was performed by cardiologists. No problems were observed in the follow-up. The patient was discharged on the fifth day of conservative treatment due to splenic infarction.
Conclusion: Thrombosis prophylaxis with prophylactic doses of LMWH in hospitalized COVID-19 patients may not be sufficient to prevent the development of coagulopathy in patients. Abdominal-visceral thromboembolism should be suspected in a COVID-19-positive patient presenting with abdominal pain despite receiving anticoagulant therapy and normal d-dimer levels.

1. Introduction

Coronavirus 2019 disease (COVID-19) is a viral multisystemic disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. A predisposition to arterial and venous thromboembolism associated with impaired coagulopathy has been demonstrated in COVID-19 [1,2]. Thromboembolic events are multisystemic and can be seen most frequently in the lungs but also in the heart, brain, kidney, intestine and spleen [2]. Increased D-dimer levels and low antithrombin levels are among the factors blamed in these situations, but the data obtained could not clearly explain the cause of this coagulation disorder.

Herein, we present a case of SI with acute MI secondary to COVID-19. This highlights the need for diagnostic alertness and high suspicion for significant thromboembolic disease in patients with COVID-19 despite receiving anticoagulant therapy with low d-dimer levels.

2. Case presentation

A 45-year-old male patient was admitted to the emergency department nine days ago with complaints of cough and sore throat. Initial observations showed oxygen saturation of 88% on room air, heart rate of 104 beats per minute, blood pressure of 105/75 mmHg, and temperature 38.5 °C. Naso- and oropharyngeal swabs using the real-time PCR method were positive for SARS-CoV-2 infection. Typical findings consistent with COVID-19 pneumonia were observed on thoracic CT. His body mass index (BMI) was 22.7 kg/m^2. He had no significant medical history. He had no known drug allergies, a history of smoking or drinking alcohol and took no regular medications.

Antiviral (favipiravir), dexamethasone 1 × 1, LMWH (1 × 40 mg enoxaparin) and 3/L oxygen support were given due to SO2: %88. His d-dimer level was 660 μg/L (reference: 0–440).

The patient's oxygen support gradually decreased during follow-up, and antiviral treatment was completed. He had been discharged with LMWH on the seventh day of follow-up with 94% oxygen saturation in

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room air, a heart rate of 78 per minute, a blood pressure of 120/70 mmHg, and a temperature of 36.5 °C. The patient was admitted to the emergency service with a complaint of left upper quadrant and left flank pain that persisted for one day. Physical examination was normal except for mild tenderness without peritonitis findings in the left upper quadrant. No pathological finding was found except 17.2 × 10∧3/μL leukocytosis in the laboratory tests. D-dimer: 310 μg/L (0–440) and troponin: 0.001 values were within the normal limits. His body mass index (BMI) was 22.1 kg/m^2. The patients' COVID-19-positive and post-COVID emergency admission laboratory findings are summarized in Table 1. Abdominal CT showed an approximately 57 × 48 mm noncontrast hypodense area extending from the capsular region to the hilus in the middle of the spleen (Fig. 1). Thorax CT showed subpleural dominant irregular ground-glass opacities and interseptal thickening in both lungs. This view was consistent with a typical COVID-19 image on thorax CT taken approximately 10 days prior (Fig. 2). No additional pathology or progression was observed. Oral intake of the patient was stopped. Intravenous hydration and nonopioid analgesics were started. The anticoagulant treatment (enoxaparin) dose he was taking was increased to 2 × 0,6 mL. The patient described sudden onset chest pain on the first day of follow-up. ST segment elevation in the limb leads (D2, D3, aVF) was seen in the ECG, which is compatible with acute inferior MI. The patient was presented and consulted to cardiologists. No thrombus in the heart was observed in his cardiac echography. Emergency coronary angiography was performed by cardiologists. The patient was followed up in the coronary intensive care unit one day after a stent was successfully placed in the RCA. After a haemodynamically stable course, the patient was transferred to the general surgery clinic. The patient's remaining hospital admission showed regressive abdominal pain requiring insignificant doses of analgesics. Oral intake was gradually increased. Abdominal findings (epigastric and left hypochondrial tenderness) of the patient were regressed in the daily repeated examination. He was discharged with 100 mg acetylsalicylic acid and ticagrelor (90 mg). Genetic hypercoagulant tests included lupus anticoagulan and antiphospholipid syndrome 3, which were examined by the Department of Hematology and Genetics during his follow-up and were found to be negative. The spleen was reported as normal in the control abdominal ultrasonography performed three months later. No intra-abdominal fluid or abscess collection was observed. Since a normal spleen was reported on USG, the patient was not evaluated as asplenic. Meningococcal and Haemophilus influenza vaccines were not considered necessary to him by the department of infectious diseases, and discussion with the department of radiology concluded that follow-up imaging was not indicated. The work has been reported in line with the SCARE 2020 criteria [3].

Table 1
The patients' COVID positive and post-COVID emergency admission laboratory findings.

Parameters	COVID positive	During admission ER	Reference range
White blood cell	3.9	17.2	4-10 × 10∧3/μL
Neutrophil	2,1	14,8	1,2–8 × 10∧3/μL
Lymphocyte	0,8	2,0	0,5–5 × 10∧3/μL
Platelet	235	212	100–400 × 109/L
Haemoglobin	14,4	14,7	11,5–16,5 g/dL
Urea	21	24	17-43 mg/dL
Creatinin	0,81	0,72	0,51–0,95 mg/dL
Sodium	137	136	135-145 mmol/L
Potassium	3,9	4,0	3,5–5.1 mmol/L
Alanine aminotransferase	25	27	0-40 U/L
Aspartate aminotransferase	28	29	0-40 U/L
Glucose	86	88	74-106 mg/dL
Amilase	74	75	0–96 U/L
Troponin I	0.001	0.001	
Estimated glomerular filtration rate	>60	>60	>60 mL/min
Prothrombin time	13	14	11–16,5 s
INR	1,03	1,04	0,8-1,2
D-dimer	660	310	0-440 μg/L

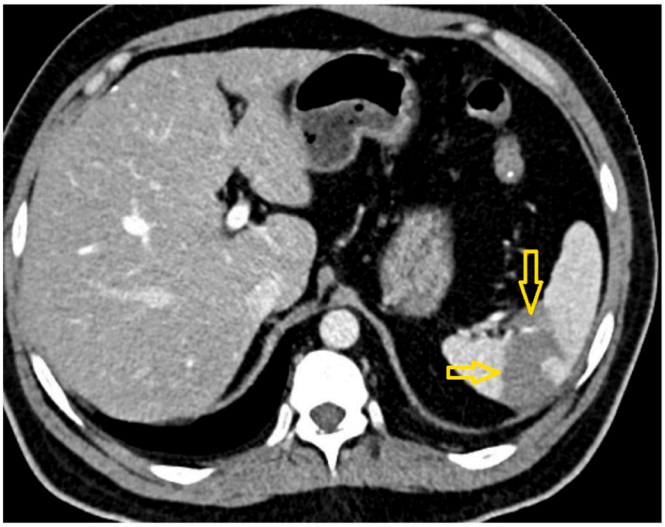

Fig. 1. Abdominal CT showed an approximately 57 × 48 mm noncontrast hypodense area extending from the capsular region to the hilus in the middle of the spleen.

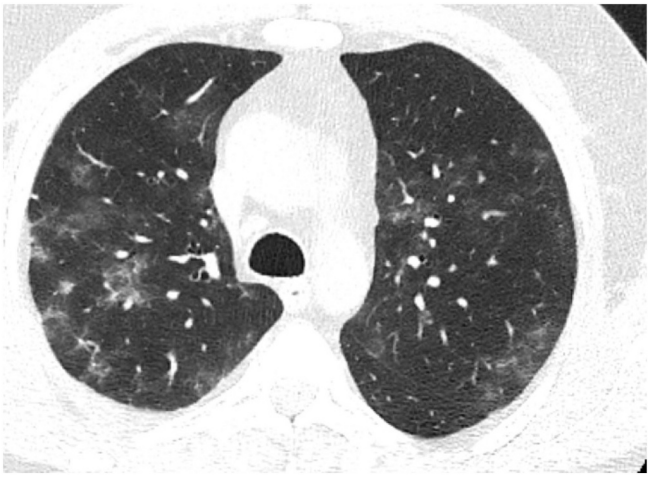

Fig. 2. Thorax CT showed subpleural dominant irregular ground-glass opacities and interseptal thickening in both lungs, which was consistent with typical COVID-19 pneumonia.

3. Discussion

Thromboembolic events are most common in the lungs and have been reported to be less common in the heart, brain, kidney, gastrointestinal system and spleen in COVID-19-positive patients [1]. These thromboembolic events can be seen simultaneously in COVID-19-positive patients, as in our case. SI is a rare condition in which many predisposing factors, such as obesity, malignancy, cardioembolic events, vasculitis, autoimmune disorders, atrial fibrillation, endocarditis

history, red blood cell abnormalities, and hypercoagulopathy, are involved in its etiology [4]. Rarely, it can be seen in COVID-19-positive patients. Ninety-two percent of cases of SI coexisting with COVID are seen in men in the literature, and the average age has been reported to be sixth decade [4].

Thromboembolic events are usually observed in these patients two weeks after the diagnosis of COVID-19. Hypertension has been reported as the most common comorbidity in patients. However, it has been reported that it can also be seen in patients without comorbidities, as in our case. Most patients diagnosed with SI present with pain in the left hypochondrium or left flank. SI can present with a wide perspective ranging from an asymptomatic clinic to acute abdomen or hypovolemic shock [5]. In the definitive diagnosis of the disease, imaging methods are most frequently utilized by CT.

Although our patient's result was normal, increased d-dimer levels were also remarkable in these patients [6]. D-dimer is an increased fibrin degradation product in many thrombotic events, and its increased levels in patients with COVID-19 pneumonia have been reported to be associated with a higher risk of venous thromboembolism (VTE), disease severity, and increased mortality rates [7,8]. High D-dimer levels have low specificity for VTE since they can be seen as increased in many different conditions (pregnancy, sepsis, malignancy, etc.). Although it has low specificity, normal D-dimer levels in a patient can rule out VTE [9]. Studies conducted on the etiopathogenesis of thromboembolic events in patients positive for COVID-19 blamed the resulting endothelial damage, coagulopathy secondary to sepsis, virus-induced antiphospholipid syndrome and systemic inflammatory response syndrome [2]. Although increased platelet and d-dimer levels and low antithrombin levels are among the factors that are blamed in these cases with the data obtained, the cause of this coagulation disorder could not be clarified clearly.

Conservative medical follow-up is usually sufficient with anticoagulant therapy. However, due to splenic bleeding, aneurysm, spontaneous rupture and splenic abscess, these patients may need to undergo surgical operation. Patients with a positive test for COVID-19 have an increased risk of pulmonary complications and mortality secondary to surgical operations performed in the early period [10]. In addition, the risk of infection may be high in patients after a single splenectomy operation. Therefore, patients should be monitored closely and should be followed up conservatively as much as possible.

The risk of thromboembolism is higher in patients with a chronic disease, obesity, high D-dimer level and positive COVID-19 [7–9]. One of the most important features that distinguish our case from other patients with SI is that our patient did not have any predisposing factors that would increase thromboembolism and d-dimer levels, and he was followed under anticoagulant therapy from discharge to the diagnosis of SI. Although LMWH dose was increased and treated, MI developed in the patient and was successfully treated with emergency coronary angiography. Therefore, prophylactic anticoagulant therapy has an important role in preventing the development of arterial and venous embolism in the postdischarge period even in the absence of high-risk factors, obesity, high D-dimer levels or inactivity in patients with COVID-19 pneumonia [8,11].

In conclusion, guidelines support the use of prophylactic doses of anticoagulants in all hospitalized COVID-19 patients, but thrombosis prophylaxis with LMWH may not be sufficient to prevent the development of coagulopathy in patients who have been hospitalized due to COVID-19 pneumonia. Prospective studies involving patients without risk factors for this condition will help guide the best prophylaxis and treatment after discharge.

Informed consent

Written informed consent was obtained from the patient who participated in this study.

Financial disclosure

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Consent

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version. Additionally, there are no conflicts of interest in connection with this paper, and the material described is not under publication or consideration for publication elsewhere.

Author contribution

SDA designed, prepared and wrote the article. SDA and GA collected the patient's data, participated in the medical treatment of the patient. All authors have read and approved the last article.

Registration of research studies

Not applicable.

Guarantor

Semra Demirli Atici, MD.

Declaration of competing interest

No conflict of interest was declared by all the authors.

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