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ИНФАРКТ СЕЛЕЗЕНКИ И ИНФАРКТ МИОКАРДА У БОЛЬНОГО COVID-19 НА АНТИКОАГУЛЯНТНОЙ ТЕРАПИИ С НОРМАЛЬНЫМ УРОВНЕМ D-ДИМЕРА

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Splenic Infarction and Myocardial Infarction in A Patient with COVID-19 on Anticoagulant Therapy with Normal D-Dimer Levels

Резюме

Многие исследования показали, что COVID-19 может прогрессировать с коагулопатией и мультисистемными тромботическими патологиями. В данной статье представлен случай пациента, у которого через 9 дней после лабораторно подтвержденной коронавирусной пневмонии на фоне антикоагулянтной терапии был, при повторной госпитализации, диагностирован инфаркт селезенки в сочетании с последующим острым инфарктом миокарда. Предупреждение тромбоза профилактическими дозами низкомолекулярного гепарина у госпитализированных пациентов с COVID-19 может оказаться недостаточным для предотвращения развития коагулопатии. Следует заподозрить у COVID-19-положительного пациента с болью в животе абдоминально-висцеральную тромбоэмболию, несмотря на антикоагулянтную терапию и нормальный уровень D-димера.

Ключевые слова: *инфаркт селезенки, COVID-19, острый инфаркт миокарда, коагулопатия, тромбоз*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Many studies have shown that COVID-19 can progress with coagulopathy and multisystem thrombotic pathologies. This article presents the case of a patient who, 9 days after laboratory-confirmed coronavirus pneumonia against the background of anticoagulant therapy, was diagnosed with splenic infarction in combination with acute myocardial infarction during subsequent hospitalization. Prevention of thrombosis with prophylactic doses of low molecular weight heparin in hospitalized patients with COVID-19 may not be sufficient to prevent the development of coagulopathy.

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Abdominal visceral thromboembolism should be suspected in a COVID-19 positive patient with abdominal pain despite anticoagulant therapy and normal D-dimer levels.

Key words: *splenic infarction, COVID-19, acute myocardial infarction, coagulopathy, thrombosis*

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The authors declare no conflict of interests

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SI — splenic infarction, MI — myocardial infarction, CT — computer tomography, LMH — low molecular heparin, VTE — venous thromboembolism

Introduction

COVID-19 is a viral multisystem disease caused by SARS-CoV-2 respiratory virus [1]. During COVID-19, susceptibility to arterial and venous thromboembolism caused by impaired coagulation was observed [2]. Thromboembolic complications are multisystem and most often involve lungs, heart, brain, kidney, bowels, and spleen [2]. Increased D-dimer and low antithrombin levels are one of the factors associated with a higher risk of thromboembolism; however, the available data failed to clearly explain the cause of such clotting disorder.

This article describes splenic infarction (SI) with acute myocardial infarction (MI) after COVID-19 infection; the aim is to emphasise the need for diagnostic vigilance concerning severe thromboembolic complications in COVID-19 patients, irrespective of anticoagulant therapy and low D-dimer levels.

Case Study

On March 17, 2023, a 45-year-old male was admitted to the admissions room of Simferopol State Clinical Hospital No. 7 with cough and sore throat. The medical history was unremarkable. The patient did not have any drug allergies; he did not smoke, did not consume alcohol or medicinal products. Body mass index (BMI): 22.7. Upon admission, blood oxygenation: 88 %, heart rate (HR): 104 bpm, blood pressure (BP): 105/75 mm Hg, body temperature: 38.5 °C. Chest CT demonstrated typical signs of COVID-19 pneumonia. Nasopharynx PCR swabs came back positive for SARS-CoV-2 infection. D-dimer level was 460 ng/mL DDU (normal value: < 243). Prescribed therapy: antivirals (favipiravir), dexamethasone 6 mg, anticoagulating agent (enoxaparin 40 mg) and oxygen support, which was gradually reduced along with improved oxygenation.

Upon discharge on March 23, 2023, the patient was recommended to take low molecular heparin (LMH); blood oxygenation was 94 %, HR — 78 bpm, HR — 120/70 mm Hg, body temperature — 36.5 °C. Two days letter, the patient sought medical assistance in the admissions ward with pain in the left hypochondrium and left side of the body, which persisted for a day. Physical examination was unremarkable, except for mild tenderness in the left hypochondrium with no signs of peritonitis. Complete blood count revealed leukocytosis: 17.2×10^9/L. D-dimer of 150 ng/mL (< 243) and troponin of 0.001 (0–0.29) were within the normal range. Abdominal CT showed an unenhanced hypodense area of approx. 57 × 48 mm, extending from splenic capsule to splenic hilum in the middle of spleen (Fig. 1).

Chest CT showed prevailing subpleural ground-glass opacity and interseptal thickening in both lungs. The pattern corresponded to a typical chest CT pattern of COVID-19 obtained 9 days before (Fig. 2).

The patient stopped taking LMH. Intravenous hydration and non-opioid analgesics were initiated. Anticoagulant (enoxaparin) dose was increased to 80 mg. On day one of his readmission, the patient complained of chest pain. ECG demonstrated elevated ST segment in limb leads (II, III, aVF), evidencing acute lower MI. The patient was referred to cardiologists for consultation. EchoCG did not show any clots in the heart. Cardiovascular surgeons performed urgent coronary angiography. The patient was under observation in ICU after successful stenting of the subclavian artery. Once his haemodynamics were stable, the patient was transferred to a general surgical ward. During hospitalisation, abdominal pain reduced with the use of low-dose painkillers. Oral anticoagulants were gradually increased. The patient was discharged and recommended taking acetylsalicylic acid 100 mg and ticagrelor 90 mg twice daily. Genetic hypercoagulation testing, including lupus anticoagulant

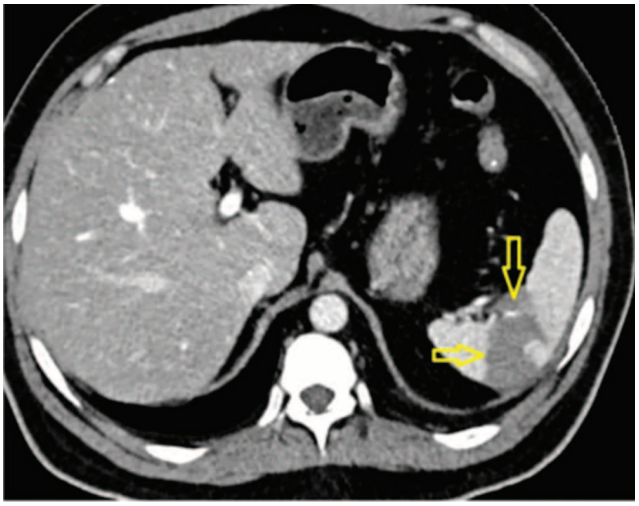

Figure 1. *Abdominal CT. Arrows indicate the hypodense area.*

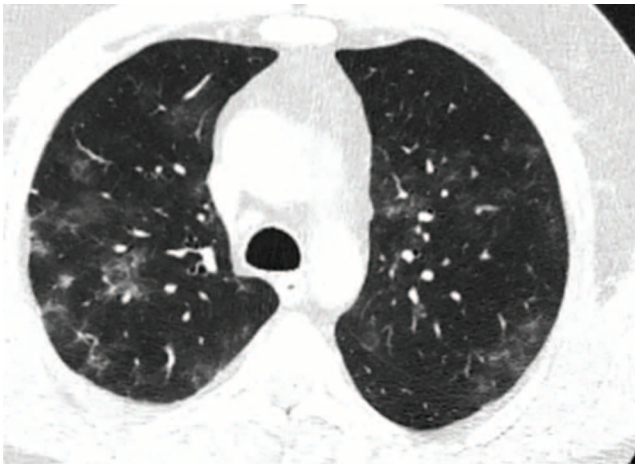

Figure 2. *Chest CT. Ground-glass opacities and interseptal thickening.*

and antiphospholipid syndrome: negative. A follow-up ultrasound examination 3 months later did not show any pathological changes in the spleen, therefore, splenectomy was not required. The infectious disease ward did not deem it necessary to vaccinate the patient agianst meningococcal and haemophilus influenza; also, the X-ray department concluded that repeated chest imaging would not be necessary.

Discussion

Most often thromboembolic complications involve lungs and are rarer in the heart, brain, kidney, GIT, and spleen of COVID-19 patients [1]. Such thromboembolic complications can be observed in COVID-19 patients, as see in this case study. SI is a rare condition, the aetiology of which involves a number of predisposing factors, such as obesity, malignancies, cardioembolic complications, vasculitis, autoimmune disorders, atrial fibrillation, a history of endocarditis, RBC abnormalities, and hypercoagulation [3]. It is rare in COVID-19 patients. In literature, 92 % of SI cases seen in COVID-19 patients are diagnosed in males, while the mean age is 60 years [3].

Thromboembolic events are usually observed two weeks after COVID-19 diagnosis. Arterial hypertension is reported as the most common comorbidity in such patients. However, SI is diagnosed in patients without any comorbidities, as seen in this case study. A majority of patients with this condition complain of pain in their left hypochondrium or left side of the body. Symptoms are versatile: from asymptomatic condition to acute abdomen or hematogenic shock [4]. Usually, final diagnosis uses CT imaging.

Such patients can also have elevated D-dimer levels; however, in this case study, the patient had normal examination results [5]. D-dimer is a degradation product of fibrin in a number of thrombotic complications; it was reported that elevated D-dimer levels in patients with COVID-19 pneumonia are associated with a higher risk of venous thromboembolism (VTE), disease severity and mortality [6]. High D-dimer levels have low specificity in VTE, since their levels can be elevated in a number of conditions (pregnancy, sepsis, malignancies, etc.). Despite low specificity, a normal D-dimer level may not be indicative of the absence of a VTE event [7]. Studies of aetiopathogenesis of thromboembolic complications in patients with a positive COVID-19 test explain resulting endothelial damage, blood-clotting disorder caused by severe viral sepsis, virus-induced antiphospholipid syndrome and systemic inflammatory response syndrome [2]. Although elevated D-dimer and low antithrombin levels are among factors associated with complications, the cause of this clotting disorder cannot be explained clearly.

Traditional medical follow-up with the use of anticoagulation therapy is usually enough for the management of patients with SI. However, due to splenic bleeding, aneurysm, spontaneous rupture and splenic abscess, such patients can require surgery. Also, a risk of infection can be high in post-splenectomy patients. Patients with positive COVID-19 test are at a higher risk of pulmonary complications and mortality as a result of early surgeries [8]. Thus, patients should be observed as close as possible.

A risk of thromboembolism is higher in patients with chronic conditions, obesity, high D-dimer levels and positive COVID-19 test [7]. One peculiarity distinguishing this case study among other patients with SI is that the patient did not have any predisposing factors, which could aggravate thromboembolism and elevate D-dimer

levels, while after SI diagnosis the patient was taking anticoagulant therapy. Despite higher LMH doses, on day one of readmission the patient developed MI, which was successfully managed with urgent coronary angiography. Thus, preventive anticoagulation therapy is essential for prevention of arterial and venous embolism after discharge, even in the absence of high risk factors, obesity, higher D-dimer levels and limited mobility of patients with COVID-19 pneumonia [9].

Conclusion

After discharge, all COVID-19 patients are recommended taking preventive doses of anticoagulants; however, thrombosis prevention with LMH can be insufficient in prevention of blood-clotting disorders in patients hospitalised with COVID-19-associated pneumonia. Prospective studies in patients who do not have any risk factors of this condition will help to develop an optimal post-discharge prevention and management.

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