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Pulmonary Thromboembolism and Splenic Infarction in a Patient with Chronic Atrial Fibrillation: A Case Report

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Abstract

In this report, we present a clinical case of a 64-year-old female patient with permanent atrial fibrillation who was concurrently diagnosed with hemodynamically stable pulmonary thromboembolism and silent splenic infarction at the time of hospitalization. The patient was admitted to the Pulmonary Department with a community-acquired bilateral pneumonia and permanent atrial fibrillation. Chest computed tomography angiography showed direct signs of acute pulmonary thromboembolism. Contrast-enhanced abdominal computed tomography revealed splenic infarction. Statins, antibacterial and non-steroidal anti-inflammatory agents, as well as the direct factor Xa inhibitor (rivaroxaban, 20 mg per day) were prescribed. The patient was discharged 1 month later with improving clinical conditions and laboratory test results.

Splenic infarction is an uncommon complication of the permanent atrial fibrillation. Clinically silent splenic infarction can be diagnosed by contrast-enhanced computed tomography. Contrast-enhanced chest computed tomography provides critical information to confirm the diagnosis of pulmonary thromboembolism in the absence of hemodynamic disorders.

In order to prevent visceral infarcts due to atrial fibrillation, a therapeutic dose of an anticoagulant must be prescribed. Anticoagulants also allow stabilizing the thrombi and preventing the appearance of embolisms in the lung.

Keywords: atrial fibrillation, splenic infarction, pulmonary thromboembolism, contrast-enhanced computed tomography

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Клинический случай тромбоэмболии легочной артерии и инфаркта селезенки у пациента с мерцательной аритмией

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Резюме

У пациентки 64 лет, госпитализированной в пульмонологическое отделение клинической больницы г. Минска с внебольничной двусторонней пневмонией и мерцательной аритмией, при компьютерной томографии органов грудной клетки и брюшной полости с контрастированием были диагностированы тромбоэмболия ветвей легочной артерии и инфаркт селезенки. В стационаре пациентке были назначены статины, антибактериальные и нестероидные противовоспалительные средства, а также ривароксабан – прямой ингибитор фактора Ха в дозе 20 мг в сутки. Через месяц пребывания в стационаре она была выписана с улучшением клинического состояния и результатов лабораторных исследований.
Инфаркт селезенки является редким осложнением мерцательной аритмии. Клинически бессимптомный инфаркт селезенки можно диагностировать только с помощью компьютерной томографии органов брюшной полости с контрастным усилением. При отсутствии гемодинамических нарушений в малом круге кровообращения компьютерная томография органов грудной клетки с контрастным усилением подтверждает диагноз тромбоэмболии легочной артерии.
С целью профилактики висцеральных инфарктов при перманентной форме фибрилляции предсердий необходимо назначить терапевтическую дозу антикоагулянта. Эти препараты стабилизируют уже образовавшиеся тромбы в сосудах большого круга кровообращения и предотвращают появление эмболов в ветвях легочной артерии.
Ключевые слова: мерцательная аритмия, инфаркт селезенки, тромбоэмболия легочной артерии, компьютерная томография с контрастированием

■ INTRODUCTION

Splenic infarction (SI) is a rare condition that occurs due to reduced blood supply to the organ, resulting in tissue ischemia and eventual necrosis [1, 2]. Cardioembolism and

identifiable hypercoagulable states have been considered to be the cause of most splenic infarctions [3].

Splenic infarction is a rare event. An incidence rate of 0.016% of admissions at a single academic medical center over 10 years was documented by Schattner et al. [4]. The authors indicated that cardiogenic emboli as well as atrial fibrillation were the predominant etiological factors of the splenic infarction. The disease may affect patients of all ages. According to Smalls et al. [5] patients 40 years of age and younger are more likely to have underlying hematologic illness while patients over 40 years of age are more likely to suffer splenic infarction due to thromboembolic diseases.

Pulmonary thromboembolism (PTE) is the third leading cause of cardiovascular death. PTE is believed to be caused by thrombi detached from deep veins of lower extremities [6]. However, this concept has recently been challenged by accumulating evidence demonstrating that PE can present without deep venous thrombosis [7].

It the current report, we describe a case with both SI and PTE, which occurred simultaneously in a 64-year-old female patient with a history of atrial fibrillation. According to the literature the concurrent presence of splenic infarction and PTE was presented for the first time in the patient with COVID-19 by Yildiz et al. [8].

■ A CASE REPORT

A 64-year-old Caucasian female patient was admitted to the Pulmonary Department of Minsk city Clinical Hospital 12/26/22 with a diagnosis "Community-acquired bilateral pneumonia, arterial hypertension, permanent atrial fibrillation, complete left bundle branch block, chronic heart failure NYHA III". She didn't have a previous medical history of chronic illnesses and other prior disease states.

Rapid antigen test for COVID-19 was negative. The patient has not been previously vaccinated against coronavirus infection.

The patient was admitted with shortness of breath on exertion, a history of high-grade fever (40 °C), dry cough shortness of breath and weakness that last 3 days.

Patient's physical exam at admission revealed a low-grade fever (37.2 °C); oxygen saturation of 95% on room air; a respiratory rate of 18/min; blood pressure of 140/80 mmHg and irregular pulse of 78 beats per minute. Muffling of the heart sounds, as well normal vesicular breath sounds were confirmed by auscultation. Abdominal wall was firm, smooth, elastic, and painless. Organs accessible through palpation were of the adequate size with no tenderness.

Electrocardiography (Fig. 1) showed atrial fibrillation with rapid ventricular response (ventricular rate 125 per min). Intermittent left bundle branch block.

Echocardiogram showed no enlargement of the cardiac cavities; contractile function of the heart was characterized as satisfactory. Pulmonary artery systolic pressure was 38 mm Hg with mean pressure of 25 mm Hg. According to ESC guidelines on pulmonary hypertension [9], pulmonary hypertension is defined by a mean pulmonary artery pressure >20 mmHg at rest and pulmonary artery systolic pressure – 15–30 mmHg.

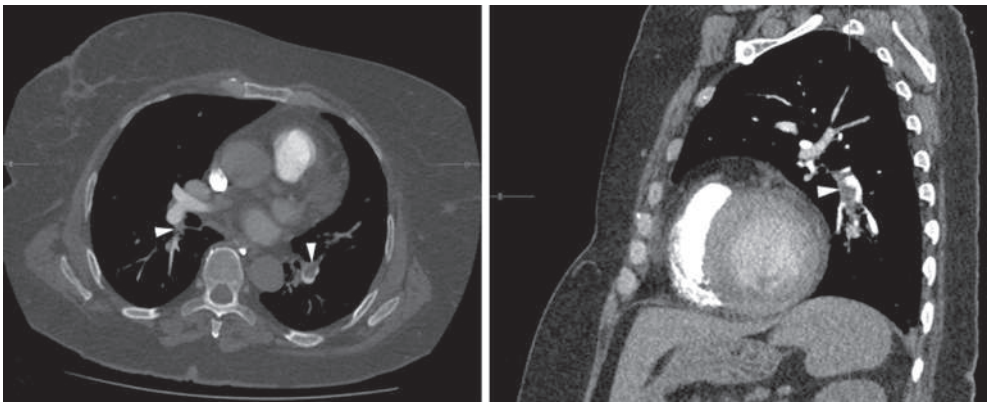
Chest CT revealed bilateral pneumonia. CT angiography showed fragmentary contrast defects in the segmental and small branches of the pulmonary arteries of the lower sections of both lungs, which are considered as direct signs of acute PTE (Fig. 2).

Fig. 1. ECG showing atrial fibrillation with a lack of visible P waves preceding irregular QRS complexes. The ventricular rate is frequently fast

Computed tomography of the abdomen and pelvis with intravenous contrast demonstrated splenic infarction (Fig. 3). Spleen showed signs of splenomegaly. In its middle third, a peripheral, hypodense area with attenuation coefficients ranging from +15 to +30 Hounsfield units, 55.5×45.5×65.4 mm in size with multiple air bubbles was visualized.

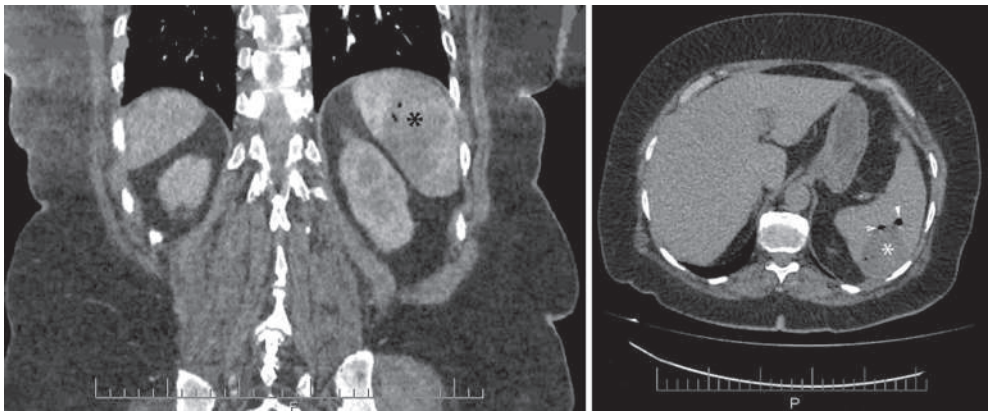
Abdominal ultrasonography revealed hepatosplenomegaly, as well as diffuse changes in the liver and pancreas. In the middle third of the spleen, an oval-shaped area of mixed echogenicity with relatively clear uneven contours, 58×38 mm in size, was found.

Laboratory tests showed abnormal erythrocyte sedimentation rate of 56 mm/hr (reference range – 0–15 mm/hr); lymphocyte count of 2.676×10^9/l (reference range



a **b**

Fig. 2. Segmental pulmonary artery branches thrombosis (arrowhead) on the axial (a) and frontal (b) reformatting images of chest CT with contrast



a **b**

Fig. 3. Infarction in the spleen (*) with air bubbles (arrowhead) on the axial (a) and frontal (b) reformatting images of abdominal/pelvic CT with contrast

1.4–4×10^9/l); C-reactive protein level of 230.5 mg/l (reference range 0–5 mg/L); fibrinogen A of 7.41 g/l (reference range 2.0–3.9 g/l) and D-dimer concentration of 3909 ng/mL (reference range <250 ng/mL); procalcitonin at 7.18 ng/ml (reference range 0–0.046 ng/ml). The patient's hemoglobin, creatinine and liver function test results were within normal limits.

Statins, antibacterial and non-steroidal anti-inflammatory agents, as well as the direct factor Xa inhibitor (rivaroxaban, 20 mg per day) were prescribed to the patient during her stay at the hospital.

The patient was discharged 1 month later with improving clinical conditions and laboratory test results (ESR – 25 mm/h, fibrinogen A – 3.33 g/l, D-dimer – 511 ng/ml, CRP – 14.2 g/l, procalcitonin – 0.06 ng/ml).

■ DISCUSSION

The described case contains a description of a rare combination of simultaneous thrombosis of the branches of the splenic artery and the pulmonary artery. The similar combination was recently described by Yildiz et al. [8]. The authors believe that this is the first clinical report of the COVID-19 case with both splenic infarction and PTE in the literature. Sinha and Mohakud [10] published a report of the case of traumatic splenic infarction with evidence of pulmonary embolism.

In our case, there were no objective data indicating SARS-CoV-2 or blunt abdominal trauma.

We were able to make a diagnosis of PTE through the direct visualization of intraluminal emboli of the segmental and sub-segmental branches of pulmonary arteries. The diagnosis was supported by pulmonary arterial hypertension on echocardiography.

The diagnosis was confirmed by high plasma D-dimer and fibrinogen levels (3909 ng/ml and 7.41 g/l respectively). According Kearon et al. [11], the pulmonary

embolism is ruled out by a D-dimer level of less than 1000 ng per milliliter. A normal D-dimer level makes acute PTE unlikely [12].

Elevated plasma fibrinogen levels are also associated with increased risk of PTE in combination with deep veins thrombosis (DVT) but not with DVT alone [13]. D-dimer/Fibrinogen ratio >103 is highly specific for the presence of acute PTE [14].

Cardiogenic emboli are the predominant etiology of visceral infarctions [15]. The most common source of peripheral embolism and the main cause for SI in elderly subjects is atrial fibrillation [16].

Splenic infarctions are a rare disorder. Usually they remain clinically silent and are not likely to be diagnosed antemortem [17]. In our case the patient with atrial fibrillation did not have the most common clinical features, including left-sided abdominal pain, and diagnosis of splenic infarction was made by enhanced-contrast computed tomography. Moreover increased levels of inflammatory markers (C-reactive protein, fibrinogen, erythrocyte sedimentation rate, and procalcitonin) which were revealed by laboratory tests support the diagnosis of the splenic infarction according to Wand et al. [16].

Increased inflammatory markers may be also associated with a splenic abscess. The presence of gas within the lesion detected by computed tomography in our case is characteristic although an uncommon sign of splenic abscesses [18].

In order to prevent further visceral infarcts due to atrial fibrillation, the patient was prescribed with a therapeutic dose of an anticoagulant. Anticoagulation is also the mainstay of treatment of acute PTE [12]. The patient was discharged, 31 days later with improving laboratory test results and clinical condition.

■ CONCLUSION

Splenic infarction can be the initial and the only one presentation of AF. The disease may be asymptomatic and accompanied by an increase in inflammatory markers. The diagnosis can be made based on CT scans of the abdomen with contrast enhancement. A CT scan of the chest with contrast can provide critical information to confirm the diagnosis of pulmonary thromboembolism in the absence of hemodynamic disorders.

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