High-risk pulmonary embolism in the course of COVID-19 infection: safety and efficacy of systemic thrombolysis

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High-risk pulmonary embolism in the course of COVID-19 infection: safety and efficacy of systemic thrombolysis
Zatorowość płucna wysokiego ryzyka w przebiegu infekcji COVID-19 — bezpieczeństwo i skuteczność systemowej trombolizy

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Abstract
Coronavirus disease 2019 (COVID-19) is an established risk factor for venous thrombosis. Treatment of pulmonary embolism (PE) of this etiology is standard, but the course of systemic thrombolysis is relatively poorly studied. The authors present the case of an 86-year-old woman with high-risk PE related to COVID-19 infection, who was treated with thrombolytic therapy with a very good clinical effect confirmed in additional studies.
Key words: pulmonary embolism, COVID-19, thrombolysis

Introduction
Pulmonary embolism (PE) is a common cause of death from cardiovascular causes. In addition to the classic risk factors predisposing to the development of thrombosis, coronavirus disease 2019 (COVID-19) infection has recently become an important cause. Sever clinical presentation of COVID-19 related PE also requires reperfusion therapy. There is limited experience in systemic PE in COVID-19.
Case report

An 86-year-old woman with hypertension, obesity, and degenerative disc disease, was urgently admitted to Emergency Department after syncope. The patient complained of weakness and pain in the upper abdomen. On admission, symptoms of cardiogenic shock were observed- pale skin covered with sweat, hypotension of 80/50 mm Hg. An electrocardiogram showed atrial fibrillation with a ventricular rate of 95/min, right bundle branch block (RBBB), and S1Q3T3 sign (Figure 1A). Performed SARS-CoV-2 antigen test was negative. An arterial blood gas test showed hyperoxia, hypercapnia, and elevated lactate levels. Transthoracic echocardiography revealed significant dilatation of the right ventricle, positive McConnell’s sign, shortened pulmonary artery acceleration time, and significant tricuspid regurgitation (Figure 2). Due to the suspicion of PE, an urgent computed tomography pulmonary angiography was performed, which confirmed a massive embolism and a lung infarction. An embolic material was shown in the division of the right pulmonary artery, the right upper lobar artery and its segmental branches, the intermediate artery and the middle and lower lobe and their branches, left pulmonary artery, lobar arteries, and also a dilated pulmonary trunk (Figure 3). Because of syncope computed tomography of the head was performed. There were no significant deviations. The patient in the emergency department received unfractionated heparin at a dose of 5000 IU and was transferred to the Intensive Cardiac Care Unit. Due to the persistent shock symptoms, it was decided to qualify for thrombolytic treatment. The patient received 100 mg of tissue plasminogen activator (alteplaza). Due to the significant overload of the right ventricle, an infusion of dobutamine was started. After the alteplaza infusion, she received a 48-hour infusion of unfractionated heparin. Six hours after the applied thrombolytic treatment, the patient’s condition improved spectacularly. The shock symptoms subsided. Echocardiography showed normal right ventricle function and electrocardiogram showed resolution of RBBB and S1Q3T3 (Figure 4 and 1B). The next day after admission, a polymerase-chain-reaction test was performed which showed COVID-19 infection. Antibiotic therapy was initiated due to an increase of inflammatory parameters. The patient was referred for further treatment in isolation and then was discharged home.
Figure 1. Electrocardiogram performed on patient: A. Before thrombolysis; B. After thrombolysis
Figure 2. Echocardiographic examination at admission: **A.** Extension of right ventricular outflow track dimension; **B.** Extension of right ventricular inflow track dimension; **C.** Shortened pulmonary artery acceleration time; **D.** Significant tricuspidal regurgitation
**Figure 3.** Computed tomography pulmonary angiography showed massive embolic material

**Figure 4.** Echocardiographic examination after systemic thrombolysis: **A.** Normal right ventricular outflow track dimension; **B.** Normal right ventricular inflow track dimension
Discussion

The literature describes numerous cases associated with COVID-19 infection and an increased risk of venous thromboembolism (VTE), including PE [1, 2]. Coagulopathy in COVID-19 differs from that in sepsis and disseminated intravascular coagulation, where the consumption of platelets and coagulation factors is not common [3]. The clotting system is related to the immune system, including cells and cytokines involved in the inflammatory process. The interaction may lead to the formation of immuno-clotting. Although this effect is initially favorable, it may eventually lead to pronounced thrombosis with organ dysfunction [4]. Endothelial defect, caused by circulating inflammatory cytokines and/or direct viral invasion, also plays a very important role in the course of VTE [5]. This may explain why the infection is more severe in diseases that are primarily endothelial dysfunction (e.g., diabetes or hypertension) [4] and why treatments aimed at endothelial regeneration may be useful in the treatment of patients with COVID-19 [5].

According to the European Society of Cardiology guidelines, in the conservative treatment of PE with cardiogenic shock or hypotension, anticoagulation therapy is used, also thrombolysis plays an important role. However, there are no clear guidelines for the treatment of patients with COVID-19 and PE. There are reports suggesting that the use of thrombolytic therapy in some patients with massive PE without cardiogenic shock may have a positive clinical effect [6]. Our description shows that the implementation of aggressive therapy in PE during COVID-19 resulted in improvement of the patient's condition and the relief of symptoms, without the side effects of thrombolysis.

References

