

Catheter-directed thrombolysis for the treatment of acute pulmonary embolism refractory to systemic fibrinolysis

Jakub Stępniewski^{1,2,3}, Wojciech Magoń¹, Kamil Jonas^{1,2,3},
Dorota Sobczyk^{2,4}, Piotr Podolec^{1,2}, Grzegorz Kopec^{1,2}

¹ Department of Cardiac and Vascular Diseases, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland

² John Paul II Hospital, Kraków, Poland

³ Department of Medical Education, Jagiellonian University Medical College, Kraków, Poland

⁴ Emergency and Admission Department, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland

The management of acute pulmonary embolism (PE) was defined in recent guidelines; however, a variety of clinical scenarios still remain a diagnostic and therapeutic challenge.^{1,2} Failure of systemic thrombolysis (ST) for acute PE is defined as persistence of hemodynamic instability and right ventricular (RV) dysfunction observed within 36 hours.¹ In some patients, however, symptoms and RV dysfunction persist beyond that time, despite hemodynamic stabilization after ST and continuous anticoagulation. As the treatment of this population is less well established, we share our experience on the use of catheter-directed thrombolysis in such a clinical scenario.

A 36-year-old man with a recent history of lower limb fracture, was admitted to a district hospital due to acute dyspnea. An urgent computed tomography angiography (CTPA) revealed a saddle thrombus in the pulmonary trunk with extensive clots in the right and left pulmonary arteries (PAs) (FIGURE 1A and 1B). Shortly after, the patient's condition deteriorated and he required pharmacological support with dobutamine and norepinephrine. Anticoagulation and ST were initiated immediately. A total dose of 100 mg of alteplase was administered within 2 hours resulting in gradual hemodynamic improvement allowing for discontinuation of catecholamine infusion. In the following days, his clinical status remained stable. He was treated with parenteral heparins followed by a direct oral anticoagulant. Despite the treatment, he complained of shortness of breath on minimal exertion, had persistent tachycardia and hypoxemia. His echocardiography at day 7 after ST displayed permanent RV dysfunction and

increased RV systolic pressure of 86 mm Hg. Biochemical analysis revealed an elevated level of troponin T of 20.43 ng/l that was lower than at baseline (122.2 ng/l; reference range <14 ng/l), continuously increased N-terminal pro-B-type natriuretic peptide (NT-proBNP) of 7062 pg/ml (4422 pg/ml at baseline; reference range <500 pg/ml), and no signs of other significant abnormalities. CTPA showed persistence of clots in both PAs with only a little improvement in arterial patency as compared with the initial CTPA (FIGURE 1C and 1D). No signs of recurrent PE or features of pre-existing chronic thromboembolic pulmonary disease were found. Therefore PE Response Team was requested for advice.³ Due to the presence of clinical, imaging, and laboratory signs of significant RV dysfunction, and incomplete resolution of clots after ST, reperfusion treatment was recommended. Percutaneous ultrasound-assisted low-dose thrombolysis with the use of 2 catheters installed within clots and a total tissue plasminogen activator dose of 10 mg during a 5-hour infusion, as described previously, was safely performed (FIGURE 1E and 1F).^{4,5} Advantage of local delivery of tissue plasminogen activator directly into obstructing clots dictated the use of ultrasound-assisted thrombolysis over ST. The therapy resulted in a rapid improvement of symptoms and RV function, decrease in thrombi burden and systolic PA pressure, and reduction of the NT-proBNP to 216 pg/ml. The patient was discharged home at postoperative day 6 with only mild exercise dyspnea. He was seen at the 6-month follow-up visit reporting no exercise limitation. Echocardiography showed no signs of pulmonary hypertension and the NT-proBNP level was 19 pg/ml.

Correspondence to:

Jakub Stępniewski, MD, PhD,
Department of Cardiac and Vascular
Diseases, Institute of Cardiology,
Jagiellonian University Medical
College, John Paul II Hospital,
ul. Prądnicka 80, 31-202 Kraków,
Poland, phone: +48 12 614 22 87,
email: jakub.stepniewski@gmail.com

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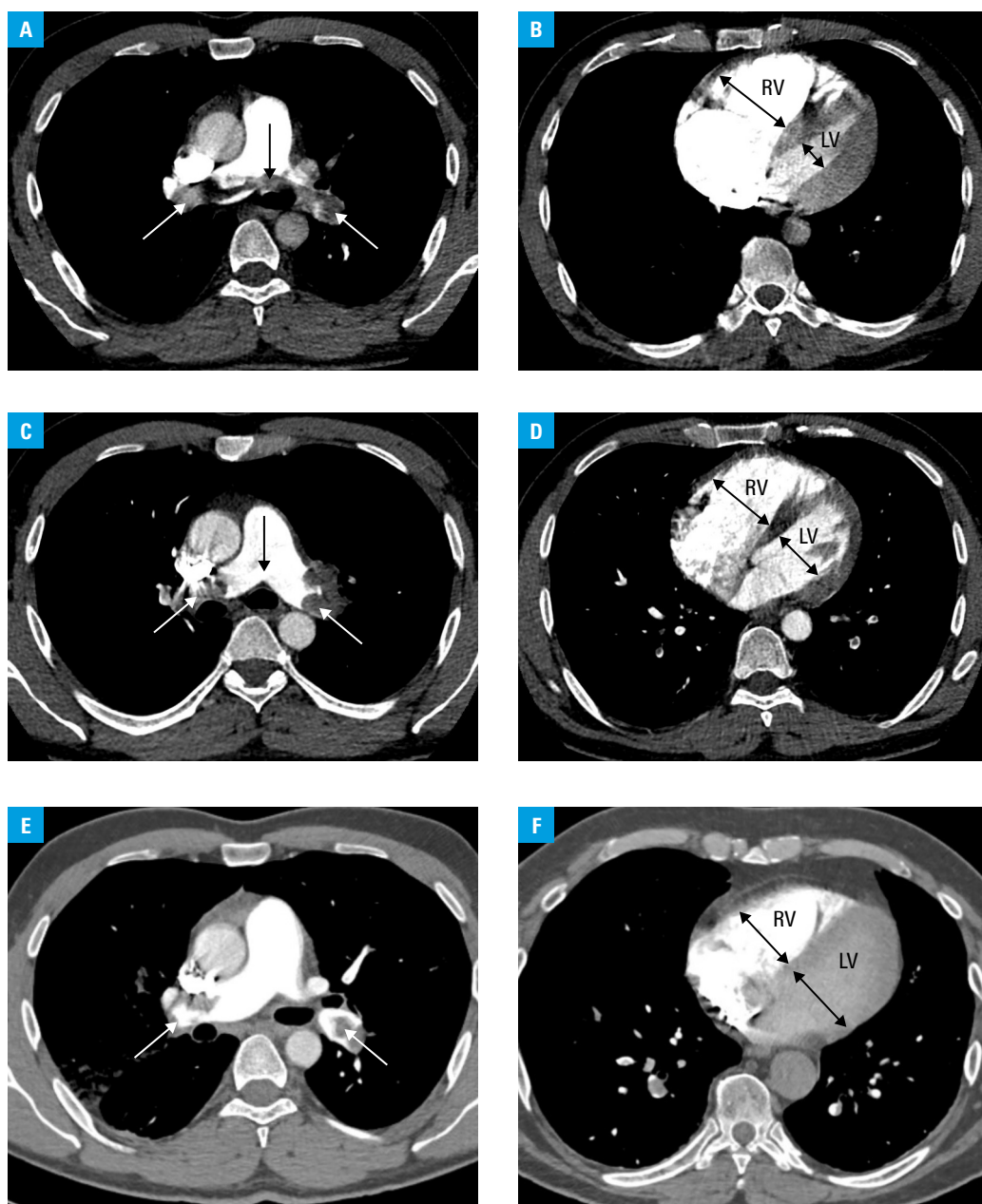


FIGURE 1 Computed tomography pulmonary angiography in a patient with high-risk acute pulmonary embolism showing saddle thrombus (black arrow) overriding the pulmonary trunk bifurcation with extensive clots in the right and left pulmonary arteries (PAs) (white arrow) (A) and dilatation of the right ventricle (RV) with the RV to left ventricle (LV) ratio of 2.2 (B), who was initially treated with systemic thrombolysis (ST) resulting in hemodynamic improvement, but persistence of dyspnea, occlusive clots in the proximal left and right PAs (white arrow) with sole resolution of the saddle thrombus (black arrow) (C) and RV dilatation (RV/LV ratio, 1.5) (D), and who eventually underwent percutaneous ultrasound-assisted low-dose local thrombolysis with the use of 2 catheters installed within clots, which led to withdrawal of symptoms, restoration of both PAs's patency (white arrow) (E) and reduction of the RV dilatation (RV/LV ratio, 0.9) (F).

In this report, we showed that catheter-directed thrombolysis may bring benefit to patients with persistent symptoms after incomplete ST. Vigilance to such clinical situations and awareness of potential therapies are warranted as no routine management has been established yet.

ARTICLE INFORMATION

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CONFLICT OF INTEREST None declared.

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