CLINICAL IMAGE

Pulmonary embolism in a patient with mild factor VII deficiency after administration of recombinant activated factor VII during a urological procedure

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A 58-year-old white man with a history of mild factor VII deficiency and superficial urinary bladder cancer, treated with bacillus Calmette–Guérin therapy and transurethral tumor resection, was transferred from a urology department to our hospital due to pulmonary embolism (PE). In the past, the patient underwent several transurethral mapping biopsies of the urinary bladder according to postoperative management protocol. Recombinant factor VII (rVIIa) as a prophylaxis of hemorrhage was used during each procedure. No history of hemorrhagic or thrombotic complications was revealed.

Recently, the patient underwent urgent surgery at the urology department because of massive extraperitoneal bleeding after the latest transurethral procedure, performed a week earlier. During the surgery, rVIIa was administered again. The surgery was successful, without any local complications. On the fifth day after the procedure, the patient fainted. Because of persistent dyspnea and hypoxia, PE was suspected. Computed tomography angiography (FIGURE 1) was performed, revealing massive PE.

On admission to our hospital, the patient was in cardiogenic shock. Oxygen saturation was 85% despite oxygen therapy, and blood pressure was 80/60 mm Hg during adrenaline infusion. The patient's Pulmonary Embolism Severity Index (PESI) score was 148 points, which corresponds to high-risk PE (PESI class V).[1] The D-dimer level exceeded 34 690 ng/ml, the high-sensitivity troponin level was 0.131 ng/ml, activated partial thromboplastin time index was 0.95 (reference range, 0.88–1.2), prothrombin time was 38 s (reference range, 9.4–13.4 s), and international normalized ratio was 2.9. Dobutamine infusion and antibiotic therapy were initiated. A transthoracic echocardiography (TTE) showed significant right ventricular overload (right ventricular outflow tract, 40 mm; right ventricular inflow tract [RVIT], 52 mm; tricuspid annular plane systolic excursion [TAPSE], 11 mm; severe tricuspid regurgitation jet, 3.2 m/s). Because of high risk, the patient was excluded from a surgery or an invasive procedure by a cardiothoracic surgeon. Alteplase, followed by heparin infusion, was administered, which in this case was a life-saving treatment and the only possibility left. Two hours later, a massive bleeding from the urethral catheter occurred. The hemoglobin level decreased from 12.5 g/dl to 7.6 g/dl, and the patient was excluded from urgent surgery by a general surgeon. The heparin infusion was terminated. Then, 3 units of red blood cells were transfused and the heparin infusion was restarted. The patient's condition stabilized after 3 days. The anticoagulant therapy was switched to enoxaparin, while dobutamine and adrenaline infusions were gradually tapered off and discontinued. A control TTE revealed significant improvement in right ventricular function (RVIT, 40 mm; TAPSE, 24 mm; mild tricuspid regurgitation jet, 2.7 m/s), and the patient was discharged 10 days later. He was prescribed enoxaparin (2 × 0.6 ml subcutaneously) for 3 months, torasemide (1 × 5 mg), perindopril (1 × 5 mg), and iron supplementation. A TTE performed after 3 months showed normal right ventricular function (RVIT, 31 mm; TAPSE, 21 mm; mild tricuspid regurgitation), and no symptoms of chronic thromboembolic pulmonary hypertension were observed.

The current guidelines recommend rVIIa supplementation in patients with factor VII deficiency

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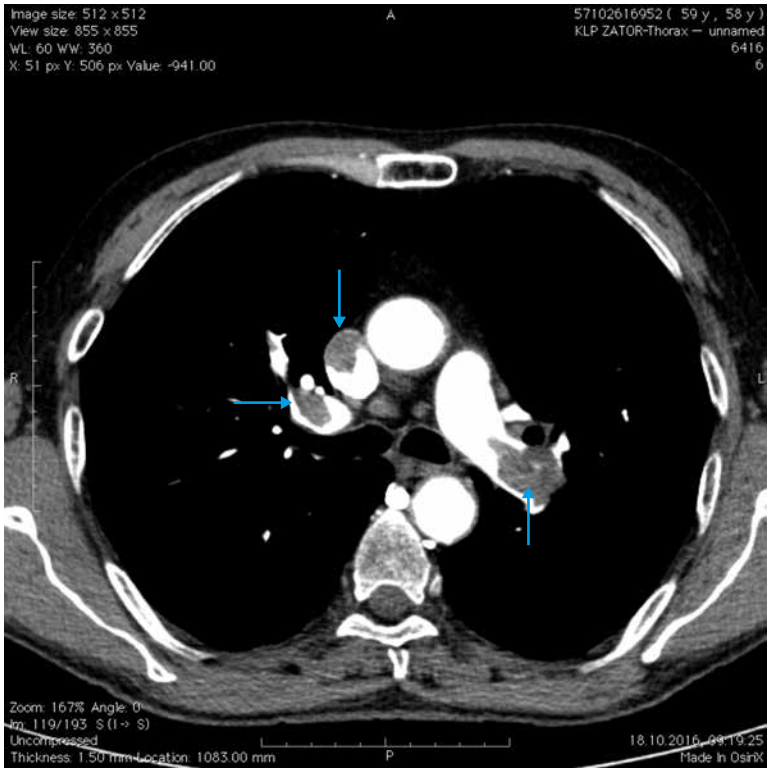

FIGURE 1 Computed tomography angiography of pulmonary arteries, revealing massive pulmonary embolism

to avoid hemorrhagic complications.[2,3] Importantly, the history of rVIIa supplementation without thrombotic complications does not exclude the possibility of thrombotic complications in the future. Several such cases have been reported by Girolami et al.[4] Our case was complex and untypical in that the patient had cancer and suffered from massive bleeding that required urgent surgery. Both these factors significantly increase the thrombotic readiness and the risk of PE. In our opinion, rVIIa administration might have been one of the many causes of PE.[5] Therefore, in such extreme cases, the use of rVIIa must be weighed against the expected benefits.

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