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CASE REPORT

ULTRASOUND-ASSISTED THROMBOLYSIS THERAPY FOR THE TREATMENT OF ACUTE PULMONARY EMBOLISM IN AN INTERMEDIATE-HIGH-RISK PATIENT WITH MULTIPLE COMORBIDITIES: A CASE REPORT

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ABSTRACT

Venous thromboembolism is an event that occurs within two components: deep venous thrombosis and pulmonary embolism. With many risk factors including old age, hospitalization and recent surgeries, malignancies, and the use of oral contraceptives, it occurs to be a relatively common condition. The European Society of Cardiology divides patients with acute pulmonary embolism into high-risk pulmonary embolism, low-risk pulmonary embolism, intermediate-high-risk pulmonary embolism, and intermediate-low-risk pulmonary embolism. This division is made based on three main criteria: hemodynamic stability, right ventricular dysfunction, and elevated troponin levels. Patient management based on risk stratification plays a vital part during the treatment. A 57-year-old woman arrived at the emergency department with syncope, abrupt shortness of breath, and unusual pleuritic chest pain. The patient had hypertension, insulin-dependent diabetes mellitus, and coronary artery disease dating back to 2012, which began with an anterior myocardial infarction treated with a left anterior descending stent. The patient later required additional stents in the left coronary artery and circumflex artery due to restenosis. At the date of approach, a three-vessel coronary artery bypass graft procedure was performed on her a month ago. With this medical history and being assessed as an intermediate-high-risk group, she was considered a candidate for catheter-directed therapy. Ultrasound-assisted thrombolysis therapy was performed with EkoSonic® Endovascular System and the patient was discharged with a total cure. This case serves as an example regarding the issue of a proper approach to patient management.

Keywords: Breast cancer, cardiovascular disease, echocardiography, pulmonary embolism

INTRODUCTION

Venous thromboembolism (VTE) is a thromboembolic event that further divides into deep venous thrombosis (DVT) and pulmonary embolism (PE) (1). VTE occurs when a blood clot is circulating in the bloodstream after breaking off its original site, most commonly a lower extremity (2). VTE is a relatively common condition, having an annual incidence of 1-2 per 1,000 persons in Europe and the United States of America (USA) (3). Although VTE has various risk factors, including old age, hospitalization and recent surgeries, malignancies, and the use of oral contraceptives, it can also occur unprovoked (4). PE is one of the most common complications in cancer patients and the occurrence of VTE in cancer patients has a substantial prognostic significance (5). Cancer increases the risk of PE/VTE because it impacts all the components of Virchow's triad, which consists of blood vessel alterations, circulation stasis, and hypercoagulability state. Apart from the disease itself, some anti-cancer treatments can also increase the risk of PE. Studies have shown that chemotherapy increases the risk of developing PE by six times, and surgery also serves as another significant factor contributing to the increased risk of PE (6).

A study was conducted to compare PE patients with and without malignancy. The study showed that the group that had both PE and malignancy showed compromised vital signs, and they

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were more frequently in the high-risk category compared to the group that only had PE without malignancies. The cancer group also demonstrated higher mortality rates and increased complications to thrombolytic therapy (7).

Pulmonary embolism is the leading and most severe complication of DVT, so PE requires risk stratification according to the severity of the condition to guide treatment (8). The European Society of Cardiology (ESC) guidelines recommend using a combination of imaging, clinical scoring systems, and biomarkers for risk stratification. The ESC divides patients into acute PE into high-risk PE, low-risk PE, intermediate-high-risk PE, and intermediate-low-risk PE (9). The first step is identifying if the patients are high-risk because they have higher mortality rates compared to the others, if the patient is hemodynamically unstable, has right ventricular dysfunction (RVD) and elevated troponin levels, then they are classified as high-risk PE patients (10). Hemodynamic instability is defined as having a systolic blood pressure lower than 90 mmHg without hypovolemia, arrhythmias, or sepsis, and/or the need for vasopressors with end-organ hypoperfusion, so high-risk PE patients are directed immediately to reperfusion therapies and organ support. Low-risk and intermediate-risk (both low and high) PE patients are all hemodynamically stable; however, intermediate-high-risk PE patients have both RVD and elevated troponin levels. On the other hand, intermediate-low-risk PE patients have either RVD or elevated troponin levels, and at last, low-risk PE patients do not present with hemodynamic instability, RVD, and elevated troponin levels (11).

There are several treatment options for PE and they all depend on the risk group of the patient. Patients with high-risk status require urgent reperfusion therapy or catheter-directed treatments (CDTs) (12). CDTs are also considered in patients developing hemodynamic instability regardless of the risk group. However, proceeding with anticoagulant treatment alone is usually enough for most patients with low-risk or intermediate-low-risk PE. According to the ESC Guidelines, advanced therapies (systemic lysis, catheter lysis, or surgical/catheter thrombectomy) should be considered in those with intermediate-high-risk PE and deteriorating clinical signs (9, 13).

This case report aims to present a patient with acute PE accompanied by multiple comorbidities, serving as an example of CDT.

CASE REPORT

A 57-year-old woman arrived at the emergency department with syncope, unusual pleuritic chest pain, and abrupt shortness of breath. The patient's medical history includes hypertension, insulin-dependent diabetes mellitus, and coronary artery disease dating back to 2012, which began with an anterior myocardial infarction treated with a left anterior descending stent. The patient later required additional stents in the left coronary artery and circumflex artery due to restenosis. Subsequently, a three-vessel coronary artery bypass graft procedure was performed on February 9, 2024, to address the restenosis. The patient also had a decreased left ventricle ejection fraction (LVEF) of 40-45%. Additionally, the patient had a history of breast cancer for which she was treated with bilateral mastectomy, followed by chemotherapy with cyclophosphamide and tamoxifen. However, chemotherapy was completed before the onset of PE. The patient was taking multiple medications including aspirin, clopidogrel, fenofibrate, perindopril, amlodipine, indapamide, metoprolol, sitagliptin and insulin glargine.

Upon arrival at the emergency department, the patient exhibited acute distress, tachycardia with a heart rate of 108 beats per minute (bpm), a blood pressure of 120/75 mmHg, and moderate hypoxemia with oxygen saturation of 91%. An electrocardiogram showed sinus tachycardia and a pattern consistent with S1Q3T3 pattern, which suggested acute PE. Physical examination revealed decreased breath sounds on the right side, as well as murmurs of mitral and tricuspid regurgitation, both graded as 3/6. The bilateral lower extremity venous system was examined using color Doppler ultrasonography. Because DVT was the initial diagnosis, the Valsalva maneuver was not performed. Widespread acute-to-subacute thrombotic alterations were detected by the imaging, which included filling deficiencies in the lumens of both common femoral veins, the bilateral superficial femoral veins along their whole course, both popliteal veins, and the visible proximal portions of the crural veins.

The right great saphenous vein terminated as a stump in its proximal portion, according to further observations; surgery was started at this level. Bilateral saphenopopliteal junctions and the left great saphenous vein were found to be patent.

Echocardiography revealed LVEF of 45%, moderate mitral and tricuspid regurgitation, a reduced tricuspid annular plane systolic excursion of 1.3 cm reflecting mild RVD, and elevated systolic pulmonary artery pressure (sPAP) of 45 mmHg, indicating pulmonary hypertension. Laboratory findings showed an elevated D-dimer at 11.1 µg/L consistent with thromboembolism, and positive troponin suggesting myocardial strain. Pulmonary computed tomographic angiography showed the right main pulmonary artery and the lobar and segmental branches of the upper, middle, and lower lobes of the right lung showed an embolism-like filling defect on multidetector computer tomography (Figure 1). In the right pleural space, a 10 mm pleural effusion was observed. A triangle-shaped region of consolidation with elevated density and ground-glass opacity was seen in the lateral portion of the right lung's middle lobe (Figure 2). This region was compatible with a pulmonary infarction secondary to embolism since it had a base connected to the pleura and showed air bronchograms. Areas of increased density next to the effusion were found in the posterobasal portion of the right lung's lower lobe. These regions were diagnosed as atelectasis because they lacked distinct borders and showed air bronchograms.

Both lungs showed a mosaic attenuation pattern, which could indicate a small airway or small vessel illness. Bilaterally, only minor emphysematous alterations were observed. The thoracic aortic and coronary vascular walls displayed calcified atheroma

plaques, while the major mediastinal vascular structures displayed normal calibration. There was no pathological wall thickening in the thoracic esophagus, and the trachea and both major bronchi were patent. There was no evidence of a left pleural or pericardial effusion. No anomalies were found in the study's upper abdominal portions. The bone structures that could be seen showed no signs of lytic or destructive lesions.

It was decided to use the EkoSonic® Endovascular System [(EKOS) EKOS Corporation; Bothell, WA, USA] for ultrasound-assisted, catheter-directed thrombolysis because of the intermediate-high-risk of PE and the elevated risk of bleeding from recent coronary artery bypass surgery. Before the EKOS procedure, a pulmonary angiography was conducted (Figure 3). During the EKOS procedure, to execute selective pulmonary angiography, a 6F JR4 catheter was inserted into the right major pulmonary artery. Massive thrombi in the right pulmonary artery's middle, lower, and upper lobe branches were visible on the angiography. A hydrophilic wire was then used to insert an EKOS catheter into the right major pulmonary artery (Figure 4). After giving a 5 mg bolus of tissue plasminogen activator (tPA), 30 mg of tPA was continuously infused over the course of 24 hours. The catheter system was successfully removed following the completion of the thrombolytic therapy. Activated partial thromboplastin time monitoring was used to maintain therapeutic anticoagulation after the patient had an intravenous heparin bolus (5,000 units) and an infusion. The patient was followed up as uneventful.

The patient's clinical condition significantly improved after receiving treatment with EKOS. Following treatment, the patient's vital signs were stable, with a heart rate of 75 bpm, blood pressure of 120/80 mmHg, and an oxygen saturation level of 95%. The resolution of pulmonary hypertension was suggested by follow-up echocardiography, which showed a stable LVEF at 45%, a decrease in tricuspid regurgitation, and a drop in sPAP to 30 mmHg. A 24-hour follow-up pulmonary angiography verified that the right-sided pulmonary thrombus had resolved (Figure 5). After being declared clinically stable,

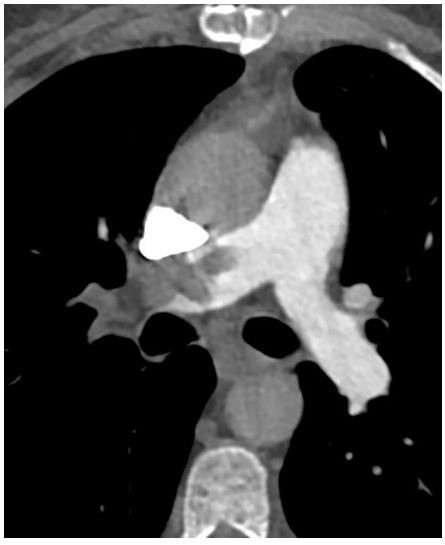

Figure 1: Computed tomography scan (superior).

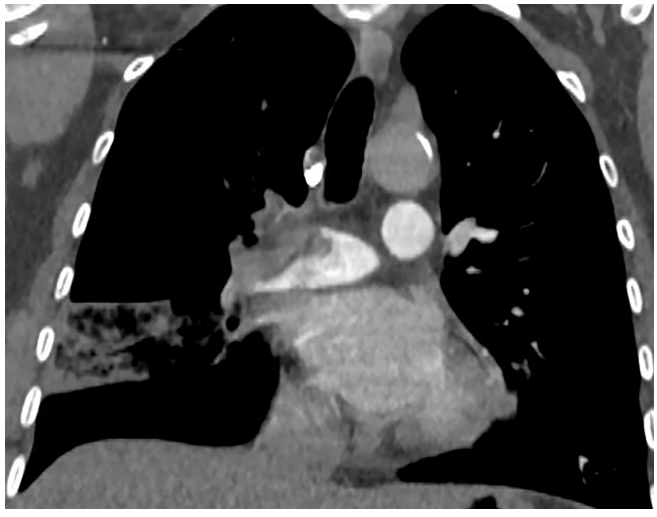

Figure 2: Computed tomography scan (frontal).

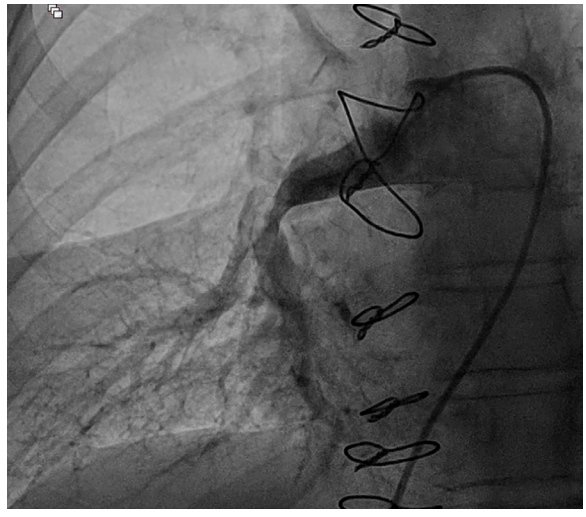

Figure 3: Pulmonary angiography before the EkoSonic® Endovascular System procedure (EKOS Corporation; Bothell, WA, USA).

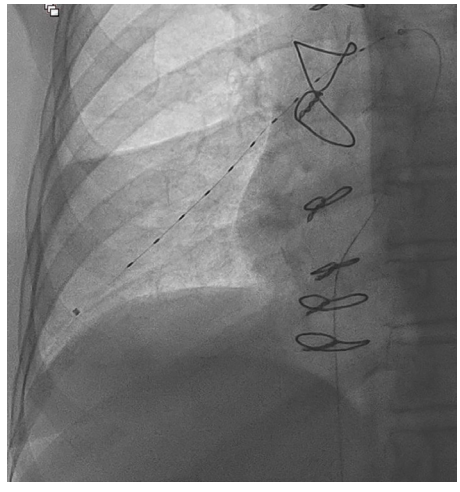

Figure 4: Insertion of EkoSonic® Endovascular System catheter in pulmonary angiography (EKOS Corporation; Bothell, WA, USA).

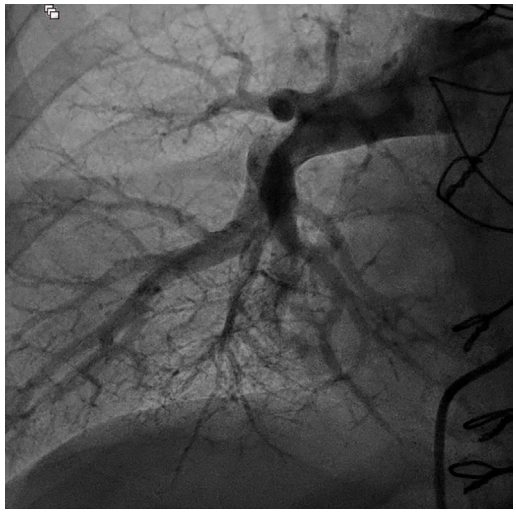

Figure 5: Pulmonary angiography after the procedure.

the patient was discharged on 6,000 units of enoxaparin (low molecular weight heparin), prescribed twice a day.

The patient was transitioned to oral rivaroxaban 20 mg once a day for continued anticoagulation at the 15-day follow-up.

DISCUSSION

Regarding the management of acute PE, a risk evaluation based on early mortality rates is a must. Hemodynamic instability, troponin levels, RVD, and clinical PE severity must be evaluated. Based on this evaluation, patients are categorized under four groups: high-risk PE, intermediate-high-risk PE, intermediate-low-risk PE, and low-risk PE (9). Besides providing ventilation and oxygen therapy, treatment options have changed drastically over time (14). For low-risk patients, anticoagulant therapy is observed to be satisfactory, and for high-risk patients, systemic thrombolytic therapy is advised, if there are no contraindications. In case of contraindications with high-risk groups, surgical treatment and CDTs must be evaluated. For intermediate-risk groups, therapy options need to be decided after patients' personal risk assessment that is done by clinicians' expertise (9). For intermediate-high-risk groups, it is vital to bear in mind that the situation may convert into high-risk; anticoagulant therapy, thrombolysis, and thrombectomy (surgical or catheter-directed) must be evaluated based on the individual (9, 15).

Based on the risk evaluation, our patient was categorized between the intermediate-high to high-risk group. With a recent history of bypass surgery, our patient was a match for CDT and was discharged with a total cure. This case serves as an example regarding the issue of a proper approach to patient management.

Ethics

Ethics Committee Approval: *N/A*

Informed Consent: *Informed consent was obtained from the patient.*

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Footnotes

Conflict of Interest: *The authors declared no conflict of interest.*

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