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Case Report

How we dealt with the double whammy! Acute pulmonary embolism with abdominal aortic clot and renal infarcts[☆]

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ABSTRACT

Pulmonary embolism (PE) is the third most common cause of cardiovascular mortality in the United States, and the submassive PE accounts for 20%-25% of all acute PE. In the last decade, endovascular therapy with catheter-directed thrombolysis (CDT) intervention has shown great success in the treatment of submassive PE. There is limited data regarding using these devices to treat patients with concomitant abdominal aortic and renal vessel clots. Herein, we present a case of a 23-year-old male who presented with submassive PE associated with abdominal aortic thrombosis and renal infarcts. The patient was successfully treated with CDT with complete resolution of pulmonary and bilateral renal artery clots.

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Introduction

Pulmonary embolism (PE) is the third leading cause of cardiovascular mortality in the United States [1]. It has variable clinical presentations and can be a mild, moderate, and sometimes fatal disease [2]. Pulmonary embolisms are classified into low risk, sub-massive, and massive groups based on hemodynamic stability, cardiac biomarkers and right ventricular (RV) strain [3]. Catheter-directed thrombolysis (CDT) is one of the latest treatment options, and it is thought to be safer than systemic thrombolysis with better efficacy than anticoagulation alone. There is limited data regarding using

these devices in the treatment of renal vessel clots. In this report, we present the case of a young male who presented with submassive PE along with abdominal aortic thrombosis and renal infarcts, and was successfully treated with CDT.

Case presentation

A 23-year-old male with a past medical history of morbid obesity and obstructive sleep apnea presented to our facility with sudden onset of shortness of breath that started earlier on the day of presentation. The patient also reported that he had 2

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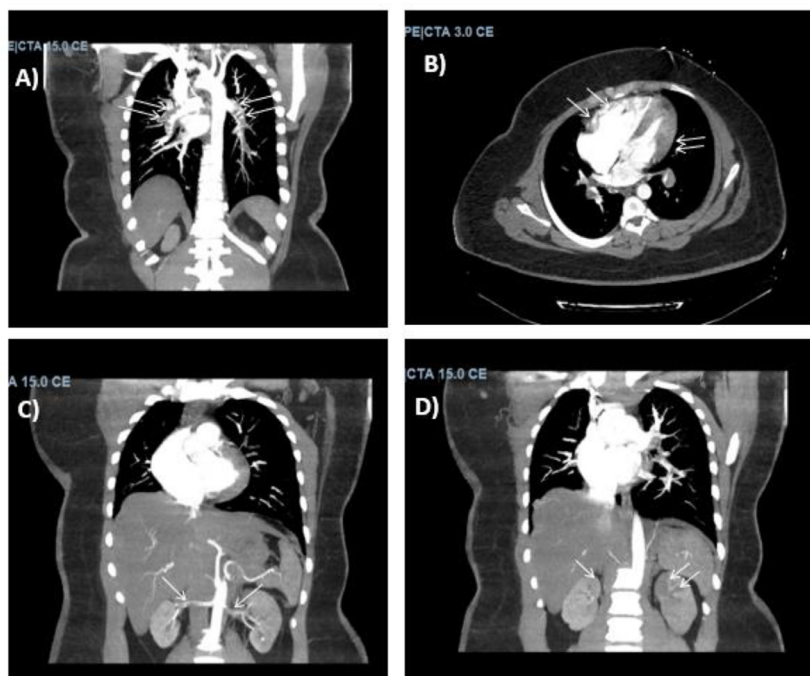


Fig. 1. – CT angiography of chest/abdomen showing extensive bilateral pulmonary emboli with high clot burden (A) and findings suggesting right heart strain with increased RV/LV ratio (B) in addition to thrombus seen within the abdominal aorta extending into the renal arteries bilaterally (C) with bilateral renal infarcts (D).

syncopal events since the last night associated with brief periods of dyspnea before losing consciousness.

He denies any chest pain, fever, history of smoking, malignancies, recent surgeries or travels. Initial vital signs showed a temperature of 36.4°C, heart rate of 118/min, blood pressure 110/66, respiratory rate of 20/min, and hypoxia with SpO₂ of 85% on room air.

The patient was subsequently placed on a 4L nasal cannula with improvement in SpO₂ to 98%. Physical exam revealed fair air entry to the lungs bilaterally with no rales or rhonchi. He was also noted to have right lower limb pitting edema.

Electrocardiogram (EKG) showed sinus tachycardia without signs of right heart strain, and troponin was 298. CT angiography of chest/abdomen revealed extensive bilateral pulmonary emboli with high clot burden and findings suggesting right heart strain in addition to thrombus seen within the abdominal aorta extending into the renal arteries bilaterally with bilateral renal infarcts (Fig. 1).

As the patient was hemodynamically stable with elevated cardiac biomarkers and CT findings suggesting RV strain, he meets the diagnostic criteria for high-risk submassive PE. Treatment options were discussed with the patient, including systemic thrombolytic therapy vs catheter-directed therapy. Given the significant risks of systemic thrombolytic therapy, including intracranial hemorrhage, the catheter-directed approach was pursued. On the other hand, the presence of bilateral renal thrombi was another challenge, so we had 2 suggestions: either administer localized tissue plasminogen activator (tPA) for pulmonary embolism and then reevaluate the renal arteries thrombi later or perform an urgent renal artery intervention.

Because the kidneys' perfusion was still preserved, the decision was made to perform pulmonary thrombolysis and then repeat the angiogram the following day and decide about the renal intervention. We also had 2 suggestions if the renal clots were not dissolved: either administering localized tPA for the renal clots or using an extraction device for aspiration.

Directed thrombolytic infusion with ultrasonic enhancement using the EKOS catheter was performed for the pulmonary embolism. A total of 24 mg tPA was used: 2 mg per catheter per hour for 6 hours and then 1 mg per hour per catheter for another 12 hours. Fortunately, the repeat angiogram showed resolution of all the thrombi, including renal thrombi, with no intervention needed, and a significant improvement in the patient's symptoms was noted (Fig. 2).

To search for the source of emboli, US LE venous Duplex was performed and revealed right popliteal vein nonocclusive thrombosis. Also, to rule out paradoxical thrombus with PE and arterial thrombosis, TEE was done, which showed small PFO with predominantly left to right flow, negative bubble study and no pulmonary hypertension. The patient was discharged to home on DOAC therapy with outpatient follow-up, and a full hypercoagulability workup was sent, which came back unremarkable.

Discussion

Pulmonary embolism (PE) is one of the common causes of cardiovascular mortality [1]. The clinical presentation of PE is variable, ranging from asymptomatic and mild to severe and critical disease. Patients with PE present with nonspecific

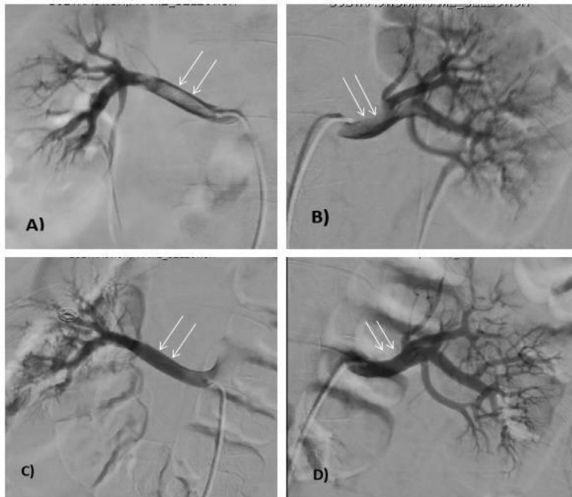


Fig. 2. – (A, B) Renal angiogram upon admission showing bilateral renal artery clots. (C, D) Repeat angiogram in the following day showing resolution of the clots.

symptoms and signs such as dyspnea, chest pain, tachycardia, hypoxemia, and even shock [2].

Based on hemodynamic stability, cardiac biomarkers and RV strain, pulmonary embolisms are classified into low risk, intermediate (submassive), and high risk (massive) groups, while the intermediate (submassive) group is further subdivided into intermediate-low risk and intermediate-high PEs [3].

The submassive PE group includes hemodynamically stable patients with significant RV strain and/or cardiac injury [4]. This group accounts for 20%-25% of all pulmonary embolisms, and these patients are at high risk of progression to hemodynamic instability [5]. Since our patient was hemodynamically stable with significant RV strain and elevated cardiac biomarkers, he was diagnosed with submassive PE.

Treatment of submassive PE targets the reduction of RV afterload and decreases RV strain. Endovascular therapy with CDT and mechanical thrombectomy has shown great success in treating submassive PE, in the last decade [6]. Catheter-directed intervention with directed thrombolysis or mechanical thrombectomy is increasingly used. It is assumed that it confers a lower risk of bleeding than systemic thrombolysis, and it has more efficacy than anticoagulation alone with low rates of procedural complications [7].

CDT involves the infusion of a thrombolytic agent intravascularly directly into the pulmonary artery adjacent to the clot burden through a percutaneous transcatheter. It rapidly decreases pulmonary artery pressures, reducing the right ventricle strain [8]. The current guidelines recommend using catheter-directed lytics in patients with intermediate-high-risk PE and relative contraindications to thrombolytic therapy or patients who failed or have absolute contraindications to thrombolytic therapy [9].

In our case, we pursued the CDT for PE due to the significant risks of bleeding with systemic thrombolysis; however, the presence of renal artery clots was an additional challenge. The renal angiogram showed adequate perfusion to both kidneys, so we decided to administer localized tPA for PE and then

reevaluate the bilateral renal artery thrombi later without performing an urgent renal artery intervention.

Mechanical thrombectomy and intra-arterial thrombolysis have been used to treat renal artery thrombosis [10]. The first case treated with intra-arterial thrombolysis was reported in 1981 [11].

We were planning to either use an extraction device for aspiration or administer localized tPA for the renal clots if they were not dissolved by the following day. Fortunately, the repeat angiogram showed resolution of all the thrombi in the renal arteries, and no further intervention was needed.

As of today, no trials with enough power have been performed to evaluate the mortality benefits of this endovascular catheter-directed intervention for submassive PE or renal artery thrombosis; therefore, treatment decisions are still affected by the location of the thrombus, operator experience, and individual risk of bleeding [12]. More randomized trials with enough power are needed before CDT can be routinely used to treat submassive pulmonary embolism and/or renal artery clots.

Conclusion

CDT has been increasingly used to treat submassive PE as it is assumed to be safer than systemic thrombolysis with uncommon complications. Currently, treatment decisions are affected by the individual risk of bleeding, thrombus location, and operator experience. Aspiration devices can also be considered an alternative therapy for retrieving renal artery clots. More research is needed to prove the efficacy and safety of this approach.

Patient consent

As this is a case report, informed consent for publication has been obtained from the patient.

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