



Case Report

Pulmonary Embolism: The Value of the Angiographic Diagnosis

Benjamin Leis, MD,^a Jason Weatherald, MD,^{b,c} Rashpal Basran, MD,^a and Jacobus De Villiers, MD^a

^aDivision of Cardiology, Department of Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

^bDivision of Respiriology, Department of Medicine, University of Calgary, Calgary, Alberta, Canada

^cLibin Cardiovascular Institute of Alberta, Calgary, Alberta, Canada

See editorial by Chandy and Channick, pages 819–820 of this issue.

ABSTRACT

We describe the case of a 55-year-old patient with a history of pulmonary embolism who presented in shock with ST-elevation on his electrocardiogram. He was triaged to the catheterization laboratory where he suffered a cardiac arrest. A pulmonary embolism was diagnosed angiographically, the thrombus was aspirated, and he received systemic thrombolysis. The combination of clot debulking and systemic thrombolysis acted synergistically to improve his right ventricular function by resolving his pulmonary hypertension. Although it is associated with a higher bleeding risk, the combination of clot aspiration with a thrombolytic agent in the treatment of massive pulmonary embolism in young patients might warrant further study.

RÉSUMÉ

Nous décrivons le cas d'un patient âgé de 55 ans ayant des antécédents d'embolie pulmonaire qui s'est présenté en état de choc et dont l'électrocardiogramme a révélé un sus-décalage du segment ST. Au triage, il a été orienté vers le laboratoire de cathétérisme, où il a eu un arrêt cardiaque. Une embolie pulmonaire a été diagnostiquée par angiographie, le thrombus a été aspiré et un traitement thrombolytique général a été administré. La réduction du volume des caillots et la thrombolyse générale ont agi de façon synergique pour améliorer la fonction ventriculaire droite du patient en résorbant son hypertension pulmonaire. Même s'il est associé à un risque d'hémorragie plus élevé, le traitement combiné par aspiration du caillot et administration d'un agent thrombolytique dans un cas d'embolie pulmonaire massive chez un patient jeune mériterait une étude plus approfondie.

We describe the case of a 55-year-old patient who had ST-elevation on his electrocardiogram and was triaged to the catheterization laboratory (CL), where he suffered a cardiac arrest. A pulmonary embolism (PE) was diagnosed angiographically, the thrombus was aspirated, and he received systemic thrombolysis. Although it is associated with a higher bleeding risk, the combination of clot aspiration with a thrombolytic agent in this circumstance might warrant further study.

Case

A 55-year-old man with a history of deep venous thrombosis presented to our academic hospital with right precordial ST-elevation on his electrocardiogram. The patient was hemodynamically unstable with sinus tachycardia at 130 and a

noninvasive blood pressure of 70/50 mm Hg. Because of his hemodynamic instability, ST-elevation, and availability of the CL, he was transferred there rather than computed tomography for definitive diagnosis.

He suffered a cardiac arrest on arrival to the CL, with return of spontaneous circulation after brief cardiopulmonary resuscitation and two 1-mg doses of epinephrine intravenously. Limited bedside echocardiography showed an akinetic right ventricle (RV). The diagnosis of massive PE was an almost certainty, thus alteplase was administered as a 10-mg intravenous bolus. Venous and arterial sheaths were placed, and left heart catheterization showed nonobstructive coronary disease in the right coronary artery and none in the left circulation. The left ventricular function was hyperdynamic (ejection fraction 70%), and the left ventricular end-diastolic pressure was 3 mm Hg. Right heart catheterization showed multiple radio-opaque thrombi occluding the left pulmonary artery (Fig. 1A). After reviewing the images with interventional radiology, the decision was made to deploy the AngioJet (Boston Scientific, Marlborough, MA) for thrombus removal because of the large burden of clot and the patient's ongoing hemodynamic instability (Fig. 1, B and C).

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Corresponding author: Dr Benjamin Leis, Division of Cardiology, Department of Medicine, University of Saskatchewan, 103 Hospital Dr, Saskatoon, Saskatchewan S7N 0W8, Canada. Tel.: +1-306-844-1155; fax: +1-306-844-1525.

E-mail: bd1127@mail.usask.ca

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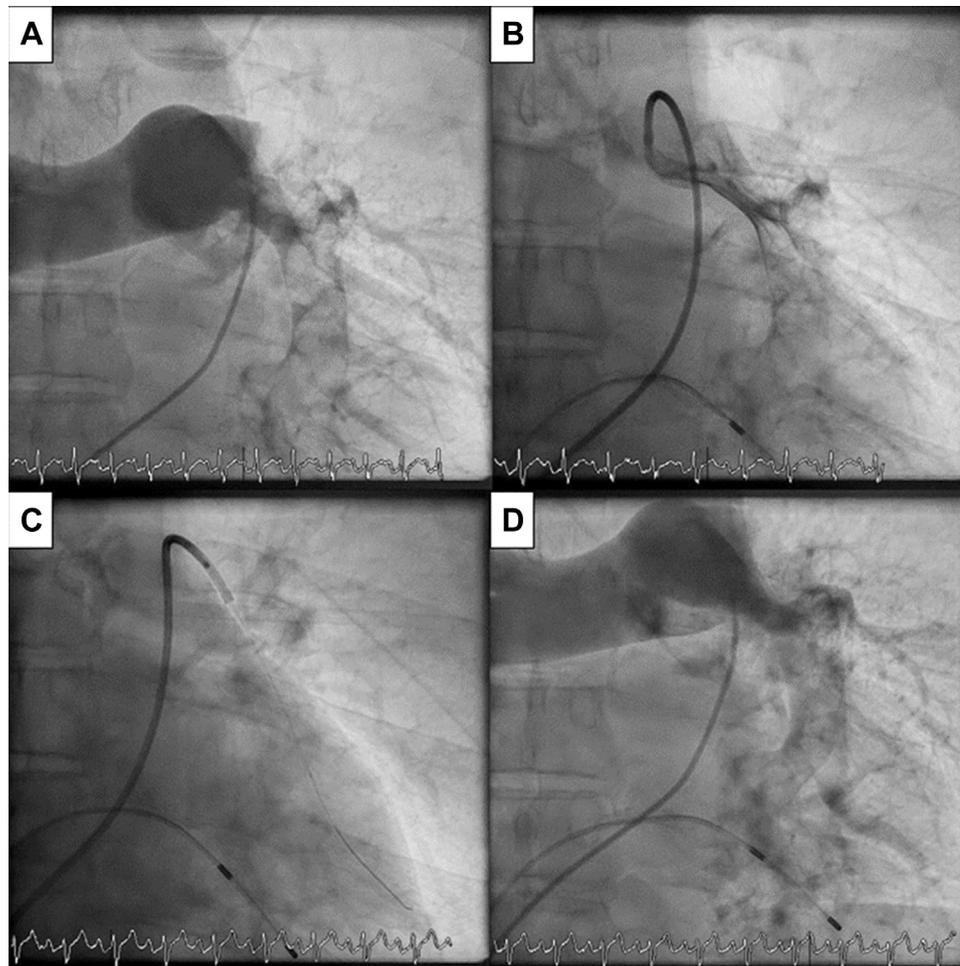


Figure 1. Fluoroscopic images of thrombus aspiration using the AngioJet (Boston Scientific, Marlborough, MA). **(A)** Pulmonary angiogram with pigtail catheter and multiple radio-opaque thrombi in the left pulmonary artery. **(B)** Temporary pacing wire (bottom of image) and AngioJet (Boston Scientific) catheter positioned before aspiration. **(C)** Wire into distal left pulmonary artery before aspiration. **(D)** Reduced thrombus burden after aspiration.

After thrombus aspiration (Fig. 1D) and infusion of 500 mL normal saline, the patient's norepinephrine requirements were weaned from 0.6 $\mu\text{g}/\text{kg}/\text{min}$ to 0.05 $\mu\text{g}/\text{kg}/\text{min}$ to maintain a mean arterial pressure > 60 mm Hg. During that time span, his pulmonary artery systolic pressure decreased from 80 mm Hg initially to 55 mm Hg (mean pulmonary artery systolic pressure 44 mm Hg). He stabilized and was transferred to the intensive care unit to finish the thrombolytic infusion. Over the next 24 hours, he required 4 units of packed red blood cells for bleeding around catheter sites. He was extubated on day 4 and transferred to the medicine ward on day 6 of his admission. An echocardiogram at discharge showed a normal-sized RV with mild hypokinesia and a right ventricular systolic pressure of 37 mm Hg. The patient followed up with his family doctor weeks later, ambulating with no complaint of dyspnea on exertion.

Discussion

It is well known that patients with acute PE can present with precordial ST-elevation on their electrocardiogram,¹ but an anterior ST-elevation myocardial infarction can present similarly. Our patient's hemodynamic instability contributed to a decision

to be transferred under the care of the CL team, which consistently has better access to emergent echocardiography, expertise in shock, and the resources to manage such patients.

We acknowledge that the administration of a thrombolytic agent, before any catheter-based therapies, might have been the only life-saving therapy required. However, small randomized trials in the context of catheter-directed therapy have shown significant immediate RV hemodynamic improvement in the context of submassive PE.² We maintain that his hemodynamics only significantly and rapidly improved after mechanical thrombus removal; whether this would have occurred anyway over time, albeit more slowly, is still possible.

We acknowledge that the AngioJet (Boston Scientific) is not the first-line catheter-directed treatment for acute PE.³ Nonetheless, we postulate that clot debulking improved peripheral delivery of thrombolysis; we recognize there is a higher bleeding risk associated with this approach. Indeed, most patients who receive catheter-directed therapy who are believed to be candidates for thrombolytic treatment receive a prolonged infusion or reduced dose to mitigate bleeding risk.³

Because of the suggestion of a "golden hour"⁴ of survival in massive PE, the emergent deployment of therapy is paramount

to improve survival. Echocardiography, assuming it is readily available and interpreted by experts, can help suggest the etiology of shock, but there are cases when it is misleading, like preexisting pulmonary hypertension with a superimposed process (ie, sepsis, small PE, or myocardial infarct). Furthermore, the computed tomography suite is generally ill-equipped to deal with an unstable patient because of staff and resource limitations. The growing body of evidence supporting the benefit of catheter-directed therapy in PE has led to the inclusion of interventional cardiology in many PE response team,⁵ a shift we are considering at our centre.

In conclusion, in acute shock with ST-elevation on the electrocardiogram and suspected massive PE, emergent angiographic diagnosis and catheter-directed therapies deployed by the interventional cardiologist contributed to an excellent outcome for our patient. Although it is associated with a higher bleeding risk, the combination of clot aspiration with a thrombolytic agent in the treatment of acute massive PE diagnosed in young patients might warrant further study.

Disclosures

The authors have no conflicts of interest to disclose.

References

1. Wang K, Asinger RW, Marriott HJL. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med* 2003;349:2128-35.
2. Sista AK, Kearon C. Catheter-directed thrombolysis for pulmonary embolism: where do we stand? *JACC Cardiovasc Interv* 2015;8:1393-5.
3. Kuo, et al. Catheter-directed therapy for the treatment of massive pulmonary embolism: systematic review and meta-analysis of modern techniques. *J Vasc Interv Radiol* 2009;20:1431-40.
4. Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 2002;121:877-905.
5. Jaber WA, Fong PP, Weisz G, et al. Acute pulmonary embolism: with an emphasis on an interventional approach. *J Am Coll Cardiol* 2016;67:991-1002.