Case Report

Management of cardiac tamponade during catheter-directed thrombolysis of saddle pulmonary embolism: A clinical dilemma

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ABSTRACT

Catheter-directed thrombolysis (CDT) for the treatment of acute pulmonary embolism (PE) has gained popularity in recent years, but potential complications during the procedure and their management are not frequently discussed in the literature. In this case report, we describe the clinical dilemma regarding the postoperative anticoagulation management of a 60-year-old male who developed cardiac perforation during a CDT of an acute saddle PE. Early resumption of systemic heparin in such cases may help in clot resolution; however, it can worsen the hemopericardium. On the other hand, delaying restarting heparin may help in healing of the cardiac perforation but can lead to clot propagation. As the chest tube output was minimal initially, anticoagulation was started, which, however, led to disastrous outcome. With limited published medical literature to help guide such a complex situation, it may be prudent to carefully weigh the risks and benefits of resuming systemic heparin versus delaying it for 1–2 days to allow for definitive resolution of the cardiac perforation.

KEY WORDS: Cardiac tamponade, catheter-directed thrombolysis, saddle pulmonary embolism

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INTRODUCTION

Symptomatic saddle pulmonary embolism (PE) is a relatively uncommon (1%–5%) type of acute PE that represents a large clot burden at the bifurcation of the pulmonary artery and has a potential for acute and severe hemodynamic deterioration.[1] Current treatment options include systemic unfractionated heparin and/or thrombolytics, surgical thrombectomy, and catheter-directed thrombolysis (CDT). CDT has gained popularity in recent years for treating acute PE in patients in whom surgery or high-dose systemic thrombolytics are contraindicated. This approach allows for targeted clot removal with considerably lower doses of thrombolytics, in turn leading to reduced rates of associated in-hospital mortality and intracranial hemorrhage.[2-4] However, there is a paucity of information related to the potential complications from CDT and their management in the medical literature. In this case report, we describe the dilemma in the postoperative anticoagulation management of a patient with saddle PE who developed cardiac tamponade during CDT.

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DOI: 10.4103/lungindia.lungindia_383_17

CASE REPORT

A 60-year-old, caucasian male with a body mass index of 32, who sustained multiple musculoskeletal and

How to cite this article: Li H, Jen S, Agarwal S, Rotem E. Management of cardiac tamponade during catheter-directed thrombolysis of saddle pulmonary embolism: A clinical dilemma. Lung India 2018;35:336-8.

vascular injuries from a motor vehicle accident presented 3 weeks later with sudden, worsening dyspnea and sinus tachycardia. Computed tomography angiography (CTA) demonstrated a large saddle PE extending bilaterally into subsequent lobar and segmental pulmonary arteries (PAs) [Figure 1]. Due to a high risk for bleeding, an emergent CDT was to be performed through the right internal jugular vein with an EKOS™ Acoustic Pulse Thrombolysis system (EKOS Corporation, Bothell, WA, USA) by the interventional radiology team. The patient decompensated during catheter manipulation before the insertion of the EKOS catheter, requiring vasopressin and norepinephrine boluses as well as endotracheal intubation. An arteriogram of the main PA revealed contrast extravasation into the pericardium, suggestive of an acute cardiac tamponade [Figure 2]. CDT was abandoned, and heparin therapy was discontinued. A total of 100 cc of dark, sanguineous fluid was drained from the posterior aspect of the pericardium using an 8 French (F) Dawson-Mueller subxiphoid drain (Cook Medical, Bloomington, IN, USA). After the patient was hemodynamically stabilized, he was transferred to the Intensive Care Unit (ICU) where the pericardial drain was exchanged for a 14F percutaneous drainage catheter (Cook Group, Bloomington, IN, USA) and an additional 75 cc of serosanguinous fluid was drained.

As the perforation occurred before the saddle PE could be dissolved and as the pericardial drain output was minimal, the ICU team restarted the patient on intravenous (IV) heparin to prevent clot progression and hopefully achieve some clot dissolution. The patient was evaluated by the cardiothoracic team and was deemed as a poor surgical candidate. Although hemodynamically stable, the patient continued to have persistent serosanguineous pericardial drainage through the night. The next morning, there was a sharp rise in serum creatinine to 4.49 mg/dL and a decrease in urine output to 55 mL in the past 12 h, which was probably due to a combination of inotropic drugs, IV contrast from the CTA as well as the CDT procedure, along with inadequate renal perfusion. The patient was placed on continuous renal replacement therapy, despite which his acidosis and hyperkalemia continued to worsen overnight. On day 2 in the ICU, the patient deteriorated requiring increasing infusions of norepinephrine and vasopressin while the pericardial drainage reduced acutely. Urgent transthoracic echocardiogram showed worsening pericardial effusion with tamponade physiology. Given the low drainage from the pericardium, a clot in the drain was suspected. The drain was replaced with a 14F Wayne pigtail catheter (Cook Medical, Bloomington, IN, USA) with immediate improvement in drainage. However, the patient's coagulation profile became deranged with a platelet count of 47 thousand/mm,[3] international normalized ratio (INR) of 2.2, D-Dimer of >5250 ng/mL, and adjusted partial thromboplastin time of 107.7 s – all suggestive of disseminated intravascular coagulopathy. A subsequent ultrasound showed multiple occlusive thrombi of the patient's femoral and popliteal veins. Furthermore, the patient remained intubated and sedated without response to verbal or painful stimuli. Given the patient's declining condition, the patient's family decided to withdraw care, and the patient passed away on the third postoperative day.

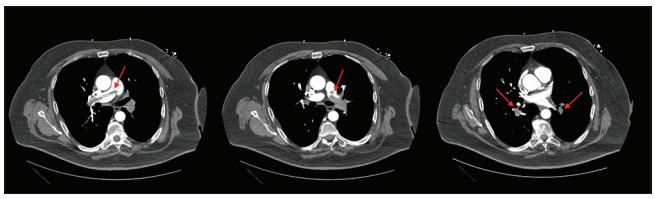

Figure 1: Contrast-enhanced computed tomography angiogram demonstrates a large saddle pulmonary embolism extending bilaterally into subsequent lobar and segmental pulmonary arteries

DISCUSSION

The use of CDT has recently gained popularity as the first-line treatment for patients with PE who are at risk for bleeding. In a 2009 review of the literature, the collective success rate for CDT was >82.2%; the minor complication risk (requiring at most an overnight observation) was <11.3%; and the major complication risk (requiring major therapy, prolonged hospitalization, or resulting in death) was <4.3%.[3]

This case highlights the clinical challenges associated with an accidental cardiac perforation during CDT. Interventional radiologists should be aware of risk factors for cardiac perforations such as old age, female sex, vessel abnormalities, and prior cardiac interventions.[5] Although our patient did not have any of these risk factors, the emergent nature of the procedure may have contributed to this complication.

Effective management comprises prompt recognition of the perforation, pericardiocentesis, intubation, vasopressors, and atropine.[6,7] Should pericardiocentesis prove ineffective, a surgery would be required.[8] However,

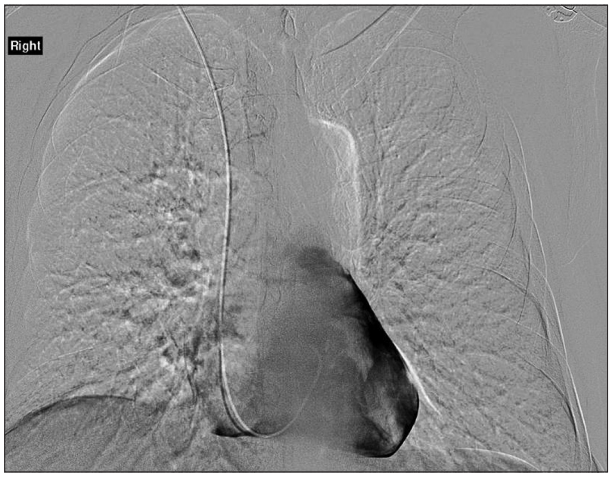

Figure 2: An arteriogram of the main pulmonary artery revealed contrast extravasation into the pericardium

because of the extremely high risk for bleeding, surgery is generally contraindicated. Furthermore, such situations bring with them the ever-present dilemma about whether systemic anticoagulation should be resumed and if so, then when? On the one hand, heparin infusion can aid in the resolution of PE and improve right heart and pulmonary function, whereas, on the other hand, it can place the patient at a higher risk of continued bleeding at the perforation site. In addition, renal dysfunction and other causes of coagulopathy may synergistically worsen the heparin effect. In the limited number of case studies with concurrent acute PE and hemorrhagic cardiac tamponade, clinicians have taken varying courses of action. In one case in which metastatic cancer caused cardiac tamponade and acute PE, the patient received an inferior vena cava filter, and heparin infusion was started 4 days after the removal of the pericardiocentesis drain.[9] In another case, a patient with a traumatic hemopericardium and acute PE was monitored for 2 days before starting the heparin infusion at a target PTT of 1.5–2.3 times the normal range. This patient also received warfarin for a target INR of 2.0–3.0.[10] Reversal agents such as protamine sulfate and Vitamin K or prothrombin complex concentrates are commonly used to reverse the effects of heparin and warfarin, respectively, in cardiac perforation cases;[6,11] however, this approach is not feasible in patients with unresolved saddle PE as it would worsen the hypercoagulable state and cause more clot progression.

CONCLUSION

Although CDT remains an effective treatment for acute PE in patients with a high risk of bleeding, there is a potential for life-threatening complications. Awareness of the risk factors for cardiac perforations as well as quick recognition and prompt pericardiocentesis will yield the most optimal outcome. If systemic anticoagulation is warranted for continued clot dissolution, the risks and benefits of resuming systemic heparin versus delaying it for 1–2 days to allow for definitive resolution of the cardiac perforation must be considered.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgment

We would like to acknowledge Dr. Hoang Anh Vo and Dr. Gregory Bowers for their clinical insight into this case.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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