Surgical Embolectomy of a Floating Right Heart Thrombus and Acute Massive Pulmonary Embolism: Report of a Case

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Right heart thrombus represents a mobilized deep venous thrombosis that is lodged temporarily in the right atrium and ventricle, and is often referred to as “emboli in transit.” Floating right heart thrombus is an uncommon but life-threatening condition, and usually coexists with an already massive pulmonary embolism. The presence of floating right heart thrombus appears to substantially increase the risk of mortality compared to the presence of pulmonary embolism alone. Floating right heart thrombus needs emergency treatment, but there is no clear consensus regarding optimal management, e.g., thrombolytic therapy, anticoagulation therapy, or surgical removal. We present the case of an 80-year-old female with a floating right heart thrombus in conjunction with an acute massive pulmonary embolism, who presented in cardiogenic shock. We successfully carried out surgical embolectomy. The patient’s postoperative course was uneventful, and she remained in good health without recurrence of pulmonary embolism. This success was based on rapid diagnosis by transthoracic echocardiography and computed tomography, prompt decision-making to proceed with surgical intervention and efficient postoperative care. In this case, surgical embolectomy was effective for a floating right heart thrombus with acute massive pulmonary embolism.

Keywords: thrombus, right heart, pulmonary embolism, surgical embolectomy

Introduction

Floating right heart thrombus (FRHTh) is uncommon, and is generally diagnosed when echocardiography is performed in patients with suspected or proven pulmonary embolism (PE). FRHTh is regarded as being in transit from the legs to the pulmonary arteries and thus is a form of venous thromboembolic disease.\textsuperscript{1,2,3} Mortality rates with FRHTh are high, at over 40% of cases.\textsuperscript{3} Moreover, the presence of FRHTh appears to substantially increase the risk of mortality compared to the presence of PE alone.\textsuperscript{4,5} The thrombus is very unstable, and migration towards the pulmonary trunk is often fatal.\textsuperscript{5,6} Thus FRHTh needs emergency treatment; however, there is no clear consensus regarding optimal management, e.g., thrombolytic therapy, anticoagulation therapy, or surgical removal.\textsuperscript{3,4} We herein present the case of a patient who presented with a floating thrombus in the right atrium accompanied by acute massive PE.

Case Report

An 80-year-old female was admitted with sudden dyspnea and chest pain. On admission she presented in cardiogenic shock with a systolic blood systolic pressure of 60 mm Hg, a heart rate of 96 beats/min, and oxygen saturation of 92% while breathing 100% oxygen. The electrocardiogram showed sinus tachycardia and ST-T wave depression in leads V1 to V3. The chest radiogram documented no abnormalities.
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Performance of transthoracic echocardiography (TTE) demonstrated a free-floating, worm-like, homogeneous, right atrial mass of 10 cm in length prolapsing through the tricuspid valve during the cardiac cycle (Fig. 1). The right ventricle (RV) was enlarged and paradoxical septal motion was present, indicating right ventricular pressure overload. The systolic tricuspid valvular gradient was 46 mmHg. Her laboratory data showed normal white blood cell (WBC) and platelet counts. The asparatate aminotransferase (AST) level was elevated to 149 IU/l (normal values: 13–33 IU/l) and the lactate dehydrogenase (LDH) level to 589 IU/l (normal values: 119–229 IU/l). D-dimer was elevated to 20.3 μg/ml (normal values: 0–1 μg/ml). After administration of inotropic drug, computed tomography (CT) demonstrated the presence of thrombi in bilateral pulmonary arteries (Fig. 2A) and deep vein thrombosis (DVT) in both legs (Fig. 2B).

The diagnosis was acute massive PE and thrombus migrating to the right atrium (RA) due to the worm-like morphology of the thrombus and its mobility. There were no clinical signs of infection and no leucocytosis; therefore endocarditis was not likely.

An emergent operation was conducted using cardiopulmonary bypass (CPB). After median sternotomy, a dilated hypokinetic RV was seen at the onset of surgery. CPB was established with bicaval drainage and ascending aortic perfusion. Cold crystalloid cardioplegic solution was injected into the aortic root, and the aorta was then cross-clamped. Firstly, a right atriotomy was performed, and the thrombus was readily extracted (Fig. 3-A). Secondly, thrombi in the bilateral pulmonary arteries were extracted under direct vision using a forceps inserted through a longitudinal incision in the main pulmonary artery (PA) (Fig. 3-B). Weaning from CPB was uneventful, requiring 72 min of CPB time and 30 min of aortic cross-clamp time. Finally, an inferior vena cava (IVC) filter was deployed. Histological examination confirmed thrombus and cultures were negative. The patient was initially treated 6 hrs after surgery with intravenous heparin, which was continued until warfarin was fully effective, with a target international normalized ratio of between 2 and 2.5. The patient’s postoperative course was uneventful and no recurrence of PE was observed.

Discussion

In patients with FRHTh, the incidence of PE is 97% and reported mortality is over 44%. Torbicki et al. reported results of the International Cooperative Pulmonary Embolism Registry (ICOPER) study and reported that patients having PE with FRHTh had shorter duration of symptoms, lower systemic arterial pressure, higher heart rate, more frequent right ventricular hypokinesis, and greater frequency of congestive heart failure. The presence of FRHTh appears to...
Intraoperative photograph shows extraction of a floating thrombus in the right atrium (A), and pulmonary arterial thrombi under direct vision through a longitudinal incision made in the main pulmonary artery (B).

substantially increase the risk of mortality compared to the presence of PE alone. Despite this, the optimal management of FRHTH remains unclear despite the availability of several different modalities of treatment including anticoagulation with heparin, thrombolysis, catheter embolectomy, and surgical embolectomy. FRHTH is an extreme therapeutic emergency, and any delay to treatment could be lethal.

Surgical embolectomy has its own set of potential complications including an inherent delay of at least hours, general anesthesia, CPB, and the inability to remove coexisting PE beyond the central pulmonary arteries. One of the major advantages of the surgical approach is the ability to simultaneously repair a patent foramen ovale (PFO), thus reducing the risk of a subsequent paradoxical embolism. The site of surgery for severe PE with or without concomitant FRHTH is discussed in the literature. In contrast, thrombolytic therapy can be administered quickly and results in the simultaneous thrombolysis of cardiac and pulmonary arterial thromboemboli as well as thrombus in the femoral venous circulation. Thus, thrombolysis may be initially advocated. However, there have been several cases of sudden death reported with thrombolytic therapy, which may be due to thrombus fragmentation. Moreover, fragmented thrombi could cause chronic pulmonary hypertension.

In our opinion, and according to several authors, surgical embolectomy with exploration of the right atrium (RA) appears to be the most efficient treatment. When compared to medical treatment of massive PE, particularly in the last decade, surgical embolectomy was found to have lower mortality rates, a lower number of hemorrhagic events and recurrent thrombosis. We present a case of successful surgical treatment of a patient with a FRHTH and acute massive PE complicated with hemodynamic collapse and RV dysfunction. This success was based on rapid diagnosis by CT and TTE, prompt decision-making to proceed with surgical intervention and efficient postoperative care. The use of CPB would facilitate careful unloading and resuscitation of the RV and would be efficient in oxygenation of the lung.

We did not extract distal thrombi in the branches of the PA by means of Fogarty catheter extraction or manual compression of the lungs. Both methods may not be well controlled and may cause mechanical injuries to the pulmonary arterial wall, especially in the segmental branches, and may provoke parenchymal and endobronchial bleeding. Hemodynamic state may be sufficiently improved by extraction of thrombi in the central PA. After surgical embolectomy, an IVC filter was deployed. The role of IVC filters has been a controversial issue. Kucher et al. reported that IVC filter placement in cases of massive PE has been shown to reduce recurrent PE and 90 d mortality. Moreover, ICOPER data as well as several recent studies favor its use. In our opinion recurrent PE remains a significant threat after massive PE and warrants IVC filter placement. Postoperative anticoagulation therapy with heparin and warfarin was important in preventing recurrence of PE.

**Conclusion**

Optimal treatment still has to be defined; this is difficult because of the relative rarity of FRHTH and its associated high mortality rate. However, advances in surgical techniques, coupled with a multidisciplinary
approach to diagnosis and perioperative care, have significantly decreased the mortality associated with surgical embolectomy.

We successfully carried out surgical embolectomy in an elderly female patient with FRHTh and acute massive PE. Success was achieved by rapid diagnosis and decision to surgically intervene. In our opinion, surgical embolectomy, when possible, remains the best available treatment.

Disclosure Statement

The author declares that he has no conflict of interest.

References