Case Report
DOI: 10.5455/amaj.2016.02.013

Free floating thrombus in right heart associated with pulmonary embolism: The effect of streptokinase

Khalilova Shabnam, MD[1]
Samira Mammadova, MD[2]
Farida Rustamzade[3]

[1] Department of Neurology,
Central Hospital of Oilworkers,
Baku, Azerbaijan
[2] Department of Anesthesiology and Intensive Care,
Central Hospital of Oilworkers
Baku, Azerbaijan
[3] Azerbaijan Medical University

Correspondence:
Shabnam Khalilova MD,
Department of Neurology,
Central Hospital of Oilworkers,
Baku, Azerbaijan
email: khalilovashebnem@gmail.com
Phone: +994518023582

Floating right heart thrombus in -transit originate from the deep veins of lower limbs, travel to the pulmonary arteries and is uncommon in patients with pulmonary embolism. This condition is a medical emergency due to the high risk of mortality. However, the most appropriate treatment is yet to be determined and there is no clear consensus in the literature on therapeutic management.

We have described two cases of deep vein thrombosis complicated with pulmonary embolism and right heart thrombus, which were treated with streptokinase and had diverse outcomes.

Keywords: pulmonary embolism, floating thrombus, deep vein thrombosis, streptokinase

Introduction

Free floating right heart thrombus-in-transit is a rare condition, which is commonly present with acute pulmonary embolism picture, including dyspnea (91,66%), chest pain (41,66%), syncope during exertion (16,66%), etc [1]. The large use of echocardiography has induced the distinguishing of free floating right heart thrombi [2]. Right heart thrombus is considered to be an extreme therapeutic emergency that can result in fatal pulmonary embolism (PE). The overall mortality rate in patients with right heart thrombus has been reported as 28% and as high as 100% in untreated patients [3]. We report two cases of pulmonary embolism that were found to have free-floating right atrial thrombi on echocardiography. In both patients, PE was confirmed by computer tomography (CT) scan with contrast and thrombolytic therapy initiated with streptokinase. These cases illustrate well the potential life threatening nature of free floating right heart thrombus and suggest that prompt intervention is necessary.

Case Presentation

Case 1. A 65-year-old man was referred to our emergency room because of seven-day history of sudden onset and gradually worsening dyspnea. There was neither chest pain nor syncope. His past medical history was remarkable with left sided lower limb edema. His peripheral oxygen saturations fell to 85% on room air. During physical examination, his blood pressure was 110/70 mmHg with pulse 95 beats per minute. A transthoracic echocardiography was performed immediately and showed moderate enlargement of the right ventricle with systolic impairment and two free-floating thrombi in right atrium sized 27×23 mm and 33×22 mm consequently. This patient also had pulmonary hypertension with peak pulmonary pressure 70 mmHg. CT of the chest with contrast revealed thrombi in the distal branches of right and left pulmonary arteries, right-sided pulmonary effusion and "Hampton hump" sign in superior and posterior basal segments of the right lung which implies lung infarction. Meanwhile, thrombus in right atrium was also confirmed (fig. 1). Patient was admitted to intensive care unit and anticoagulation therapy with heparin was started. Laboratory studies showed D-dimer 2.16 mcg/mL, Troponin I 0.01 ng/mL, Creatine Ki-

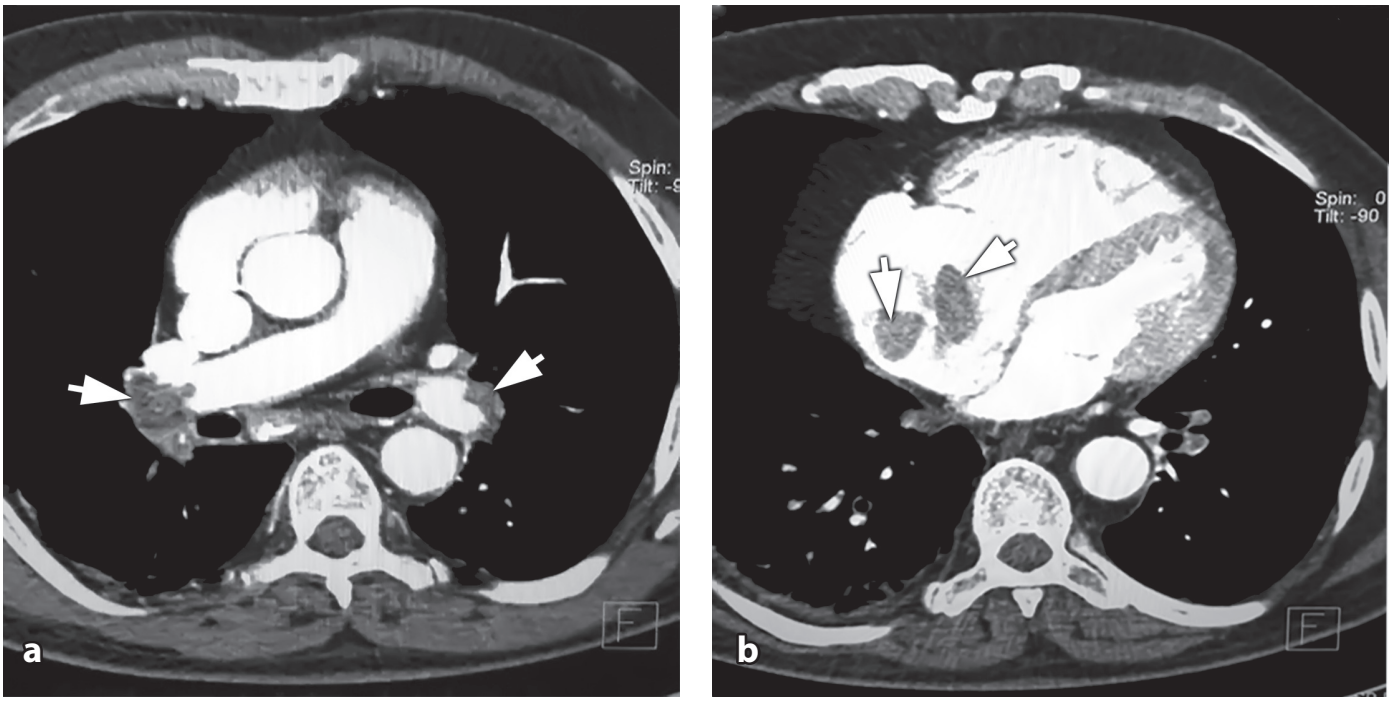

Figure 1.
Pulmonary computed tomography angiogram showing (a) pulmonary embolism and (b) thrombus in right atrium (arrows).

nase - MB 1,7 ng/mL. A Doppler ultrasound of the lower limbs demonstrated right sided deep vein thrombosis of popliteal and femoral veins. After consultation with cardiothoracic surgeon, thrombolytic therapy with Streptokinase was initiated with loading dose 250000 IU in 30 minutes then with 100000 IU per hour as continuous infusion. During the first minutes of infusion, patient suddenly worsened. Patient was sedated, intubated and connected to ventilator. High inotropic support was started. After 24 hour of thrombolytic therapy, repeated echocardiography showed lysis of one of the thrombi inside the right atrium. Streptokinase infusion continued for the next three days. Last echocardiography showed that right heart was free of thrombus and systolic pulmonary artery pressure dropped into the normal values. On the same day sedation was stopped and the patient underwent CT imaging of brain because of the questionable neurological status. CT confirmed small bleeding in brain stem (fig. 2).

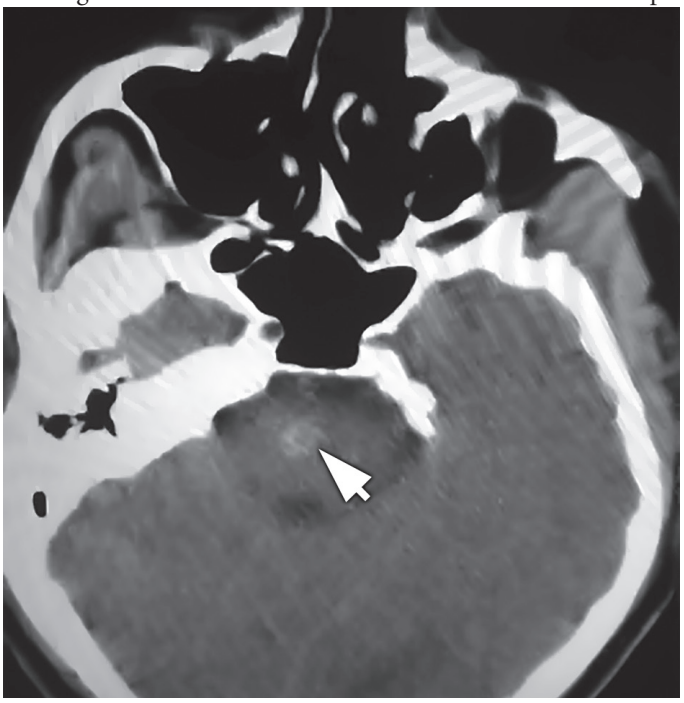

Figure 2.
Computed tomography imaging of brain showing hyperdense region in brainstem associated with hemorrhage.

Case 2. A 42-year-old man with past medical history of PE was admitted to emergency department with shortness of breathing, palpitations and syncope. He had a history of Diabetes Mellitus type 1. Patient started receiving anticoagulation therapy with warfarin one month prior to admission in our hospital. His physical examination was remarkable for tachycardia 110 bpm and tachypnea 32 rpm with a blood pressure 90/60 mmHg. Echocardiography showed mobile large thrombus in right atrium and relatively enlarged right ventricle with systolic dysfunction. It was not possible to measure pulmonary pressure. Laboratory studies showed an elevated D-dimer 121.5 ng/mL, INR 1.4 and troponin I 0.07 ng/mL. After initial stabilization and invasive monitoring, urgent CT of the chest was performed and revealed filling defects in both the right and left pulmonary arteries. Meanwhile, there was hypodensity inside the vena cava superior and filling defect in the right atrium. All the radiological findings were in favor of thrombi (fig. 3).

The patient suddenly developed cardiac arrest while diagnostic work-up and cardiothoracic surgeon consultation was going

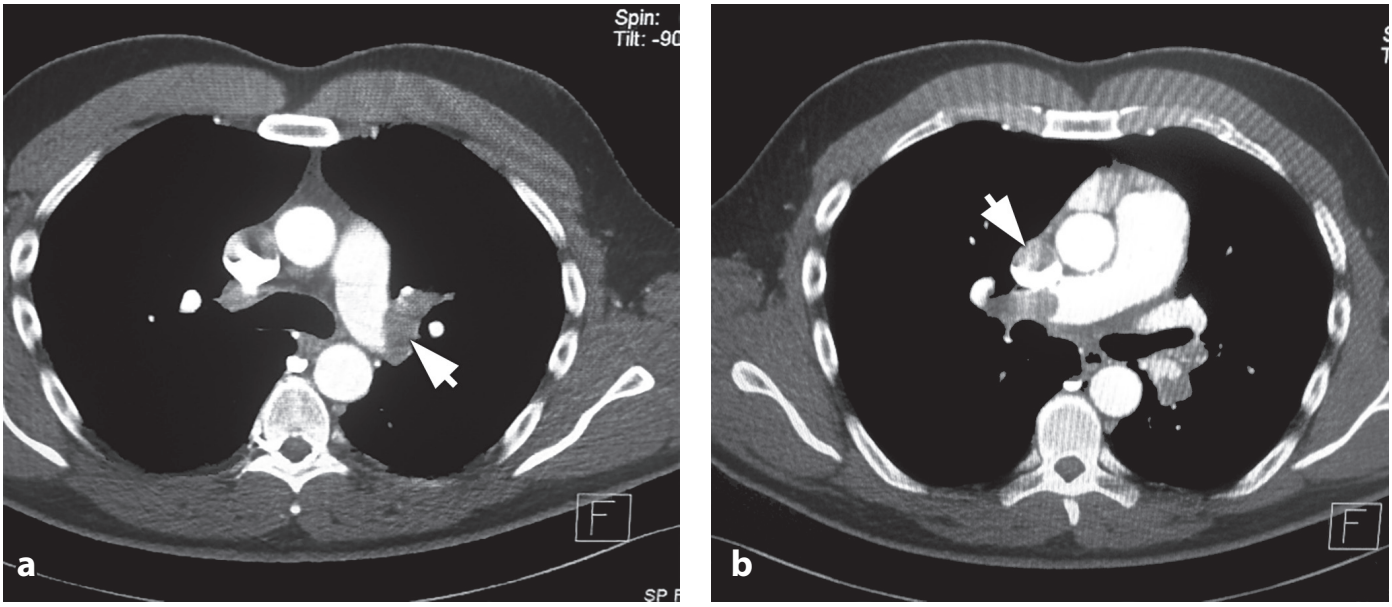

Figure 3.
Pulmonary computed tomography angiogram showing filing defects in (a) left pulmonary artery and in (b) inferior vena cava (arrows).

on. During CPR streptokinase 1.5 million IU intravenous bolus dose was given as a step of despair to save patient. Despite of one hour of vigorous resuscitation the patient did not survive.

Discussion

Mobile right heart thrombi are quite uncommon and there are two morphological types of thrombi: type A and type B. there might be peripheral venous clots which accidentally lodge in the right heart on their way to the lungs (type A) or they may develop within the right heart chambers (type B). Type A thrombi are worm like shape and are extremely mobile. Type B thrombi are less mobile, attach to the right atrial or ventricular wall and are morphologically similar to left heart thrombi [4]. Our patient had mobile thrombi moving to and from into right ventricle which resembles type A thrombi. These are very dangerous and can critically worsen the hemodynamic. For this reason, free floating thrombi are an extreme therapeutic emergency and any delay to diagnosis and treatment could be lethal. From this point of view, transthoracic echocardiography is essential for diagnosis and must be performed systematically as soon as PE is suspected. This is an essential investigation that can be performed at bedside to directly visualize the thrombi, asses and monitor right ventricle function, and help making treatment decision. [5]. Despite, this is one of the therapeutic emergencies, but there is no clear consensus on how to manage it. Three options are possible: 1) single intravenous bolus heparin, with further supporting dose, while keeping the activated partial thromboplastin time at 2-2,5 times normal; 2) surgical embolectomy with exploration of pulmonary arteries and right atrium; 3) thrombolysis. Anticoagulation with heparin is reserved for haemodynamically stable patients who are not candidates for surgery/thrombolysis. In our patients, we had a therapeutic dilemma: surgery or thrombolysis. Some studies reported that there was no expressive contrast between these therapeutic modalities in terms of in-hospital mortality. However, recent data indicate better outcomes with thrombolysis [5]. Cardiac surgery is preferred for very large RHT, tricuspid occlusion, associated paradoxical embolism via patent foramen ovale transit, thrombolytic failure or contraindications to thrombolytic therapy. In addition the theoretical advantages of thrombolysis are numerous. It accelerates lysis of thrombi and pulmonary reperfusion, minimizes pulmonary hypertension and raises right ventricular function. Moreover, it may destroy the clot at three locations at the same time: intracardiac, pulmonary and venous [5]. In our first case, thrombolysis worked very well, however there was side effect, i.e. intracranial bleeding. Besides the risk of major bleeding, thrombolytic therapy may be associated with a hypothesized risk of clot fragmentation and migration, complete pulmonary embolization or recurrent PE following partial dissolution of the venous thrombus. The occurrence of such an event in a hemodynamically unstable patient can lead to a catastrophic clinical course with severe hemodynamic compromise. However, there are numerous survivors who underwent thrombolytic therapy during cardiac arrest caused by PE [6]. Our second patient was diagnosed to have thrombi in right ventricle and proximal parts of pulmonary arteries. Such condition implies urgent surgical intervention. Unfortunately, patient had arrest during necessary diagnostic work-up. Bolus streptokinase infusion was tried as a step of despair, but failed. Nevertheless, we would like to emphasize necessity of early and aggressive use of thrombolytic therapy in patients suspected to have PE according to clinical findings. Further clinical trials are needed to elucidate all pros and cons of usage of streptokinase in similar cases.

Conclusion

Free floating thrombi in the right heart are rare and usually indicate travelling clots from the legs to the lungs. Echocardiography is main diagnostic tool in identifying the problem. However the option of optimal therapy for patients with PE with mobile right heart thrombi is still open to debate, but thrombolysis is readily available and effective.

References

1. Mollazadeh R, Ostovan MA, Abdi Ardekani AR. Right cardiac thrombus in transit among patients with pulmonary thromboemboli. Clin Cardiol. 2009 Jun;32(6):E27-31.
2. Nkoke C, Faucher O, Camus L, Flork L. Free Floating Right Heart Thrombus Associated with Acute Pulmonary Embolism: An Unsettled Therapeutic Difficulty. Case Rep Cardiol. 2015;2015:364780.
3. Rose PS, Punjabi NM, Pearse DB. Treatment of right heart thromboemboli. Chest. 2002 Mar;121(3):806-14.
4. The European Cooperative Study on the clinical significance of right heart thrombi. European Working Group on Echocardiography. Eur Heart J. 1989 Dec;10(12):1046-59.
5. Naeem K. Floating thrombus in the right heart associated with pulmonary embolism: The role of echocardiography. Pak J Med Sci. 2015 Jan-Feb;31(1):233-5.
6. O'Connor G, Fitzpatrick G, El-Gammal A, Gilligan P. Double Bolus Thrombolysis for Suspected Massive Pulmonary Embolism during Cardiac Arrest. Case Rep Emerg Med. 2015;2015:367295.