

## Thrombolytic therapy of right heart emboli-in-transit

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**ABSTRACT:** Currently, no consensus exists for the appropriate treatment of echocardiographically diagnosed mobile right heart masses giving rise to a high suspicion of migrant thromboembolism in patients with pulmonary embolism. This may lead to unnecessary delay in the implementation of the most appropriate treatment for these patients.

Several earlier studies have supported the beneficial role of thrombolytic therapy. We report on an additional two patients with mobile right heart thromboemboli, refractory to systemic anticoagulation, who recovered quickly after initiation of thrombolytic therapy.

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The prevalence of pulmonary embolism suggests that the most common cause of right cardiac masses might be emboli-in-transit [1]. These highly mobile structures must, however, be differentiated from myxomas, which rarely present on the right side. Therefore, echocardiographic detection of large serpiginous, coiling right heart masses warrants clinical suspicion of thromboemboli at a critical time in their evolution en route to the pulmonary arteries. Identification of patients with this precarious clinical condition probably necessitates subsequent aggressive therapeutic measurements [1, 2]. Several authors support immediate surgical embolectomy, whereas others advocate conventional therapy using systemic heparin. The efficacy and safety of thrombolytic therapy in this condition remains to be established [1–5]. We report two cases of right heart emboli-in-transit which were successfully treated by early and delayed initiated thrombolytic treatment, respectively. Subsequently, a re-evaluation of current treatment is suggested in individual cases of right heart masses giving rise to a suspicion of thromboembolism.

### Case reports

#### Case 1

A 70 year old man with no previous clinical history was referred to the Emergency Department with rapidly progressive dyspnoea on exertion, of one week duration. Physical examination revealed signs of manifest right heart failure, such as elevated central venous pressure, peripheral oedema and absence of inspiratory crackles. Chest radiography showed no abnormalities. Blood laboratory tests, including arterial blood gases were within normal

ranges. The electrocardiogram (ECG) showed sinus rhythm of 75 beats·min<sup>-1</sup>, left axis deviation of 60°, QS complexes in V1–V2, and negative T-waves in the inferior (II, III, aVF) and precordial leads (V2–V4).

On suspicion of cardiac failure secondary to ischaemia, cardiac ultrasound examination was performed to evaluate right and left ventricular function. This showed an unexpected elongated swirling mass in the right atrium, which was thrown back and forth through the tricuspid valve orifice during the cardiac cycle, with an alarming mobility. During the examination, part of the structure suddenly became visible in the right ventricular outflow tract (fig. 1).



Fig. 1. – Transthoracic echocardiography. Short axis view of the ventricles at the level of the mitral valve in a diastolic frame showing the flattened intraventricular septum (IVS), which is compatible with severe right ventricular pressure overload. The head of the thrombotic mass (THR) is visible in the right ventricular outflow tract. LV: left ventricle.

The right heart dimensions were increased and there was concomitant tricuspid valve regurgitation with signs of pulmonary hypertension, as indicated by a right ventricular peak pressure of 78 mmHg estimated by continuous wave Doppler (normal <35 mmHg).

A lung scan revealed segmental defects in the right middle and left lower lobe, which were compatible with a high probability of pulmonary embolism.

Subsequently, streptokinase was intravenously administered as a bolus of  $250 \times 10^3 \text{ U}\cdot\text{kg}^{-1}$  and continued at a rate of  $100,000 \text{ U}\cdot\text{h}^{-1}$  for 48 h. In response to thrombolytic therapy, the patient showed a quick and complete clinical recovery. Subsequent echocardiography showed normalized right ventricular dimensions and pressure, and complete disappearance of the right heart coiling mass. In addition, a control lung scan demonstrated completely restored perfusion.

### Case 2

A 68 year old man, with a history of chronic obstructive pulmonary disease and a prior inferior myocardial infarction, experienced right-sided chest pain of sudden

onset followed by progressive dyspnoea and massive haemoptysis 1 week after uncomplicated coronary artery bypass surgery. These symptoms were accompanied by haematuria.

Physical examination revealed: respiratory rate of 26 breaths $\cdot\text{min}^{-1}$ ; blood pressure 130/90 mmHg; pulse rate 88 $\cdot\text{min}^{-1}$ ; temperature 37.2°C; and absence of jugular venous distension. Cardiac auscultation was normal. Breath sounds were absent at the posterobasal region of the right-sided lung. Chest radiography showed cardiomegaly and consolidation of the right lower lobe with pleural effusion. The ECG showed sinus rhythm, intermediate electrical heart axis and a complete right bundle branch block.

A ventilation-perfusion scintigram demonstrated a matched impairment of ventilation and perfusion in the right posterobasal segment, which was compatible with a low probability scan for pulmonary embolism. Transthoracic and transoesophageal echocardiography showed a poor left ventricular function, dilated atria, and a mobile free-floating elongated structure in the inferior caval vein, right atrium and ventricle, extending towards the bifurcation of the pulmonary trunk (figs. 2 and 3). Such a mass is highly likely to be a thrombus of the "snake"

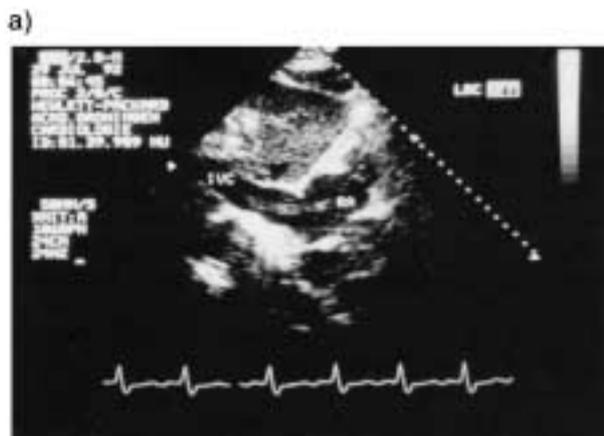


Fig. 2. — Transthoracic echocardiography. a) The subcostal view demonstrates the tail of the "snake thrombus" in the inferior vena cava (IVC) and right atrium (RA). b) The apical four chamber view shows the elongated structure in the RA extending through the tricuspid valve orifice into the right ventricle (RV) during the diastole.



Fig. 3. — Transoesophageal echocardiography. Transverse section through the ascending aorta (ASC AO), main pulmonary artery (MPA), right pulmonary artery (RPA) and left pulmonary artery (LPA). a) demonstrates the head of the thrombotic mass (THR) in the MPA; b) shows one day later the tail of the THR which remains visible in the LPA after complete embolization to the pulmonary arteries.

type, which may originate from venous thrombosis of the lower extremities or secondary to a Grawitz tumour. This condition, however, was excluded by a computed tomography of the abdomen.

Further physical examination and laboratory tests did not suggest other malignancy. The patient was treated with full dose intravenous heparin and oral anticoagulants. Deep venous thrombosis of the left femoral vein was confirmed by Doppler ultrasound examination.

When thrombectomy was being considered as a possible therapeutic option in a yet stable but threatening condition, a second echocardiogram was performed 24 h after admission to determine the actual location of the mass at that time. This elucidated complete disappearance of the right heart structure. However, part of the mass was still visible in the left pulmonary artery (fig. 3b). Pulmonary angiography now showed bilateral segmental perfusion defects in both lower lobes. Subsequently, intrapulmonary urokinase was administered as a bolus of 2,000 U·kg<sup>-1</sup>, followed by 2,000 U·kg<sup>-1</sup>·h for 48 h. The patient had a rapid and uneventful clinical recovery and repeated pulmonary angiography demonstrated improved perfusion of the right and left lower lobe.

### Discussion

Anticoagulative therapy for pulmonary embolism enables the endogenous fibrinolytic system to lyse the thrombus and prevent its recurrence. In addition to anticoagulation, thrombolytic therapy has been studied in patients with clinical signs and symptoms indicative of pulmonary embolism fulfilling one or more of the following criteria: 1) a confirmatory pulmonary angiogram showing vascular obstruction greater than 50% [6]; 2) a discrete ventilation-perfusion mismatch at the lung scan [7]; 3) a mean pulmonary artery pressure  $\geq 20$  mmHg [8]; or 4) a massively enlarged right ventricle with reduced contractility and paradoxical movement of the interventricular septum and/or right ventricular pressure over 40 mmHg [9]. Reviewing these and other studies, indicates that the administration of a thrombolytic agent was beneficial and sometimes a lifesaving alternative to thrombectomy [9]. However, one should always take into account the risk of major bleeding associated with the use of thrombolytic compounds. Consequently, thrombectomy may be indicated in massive pulmonary embolism shortly after surgery, trauma or birth. Unfortunately, perioperative mortality and morbidity used to be high [3, 10].

Transthoracic and transoesophageal echocardiography offers the physician the opportunity to diagnose right heart thromboembolism in patients with pulmonary embolism. In that case, a new therapeutic dilemma emerges. Since occlusion of the pulmonary trunk may occur after dislocation of such thrombi, which is usually fatal, one must consider adjunctive therapy. Long, thin and mobile (type A thrombi) appear to be especially risky structures. In contrast, type B thrombi are immobile, often consist of organized material and are usually firmly adherent to the wall [11, 12].

Few papers report on the efficacy and safety of thrombolytic therapy in right heart thromboembolism [1, 4, 5, 13]. Fragmentation of a right heart mass leading to clinical deterioration has never been described following thrombolytic therapy. Moreover, it was shown that after right heart catheterization, it was feasible to mechanically fragment and disperse a proximal pulmonary embolus, which substantially improved the condition of the patients [14]. Simultaneous mechanical clot fragmentation and pharmacological thrombolysis in acute massive pulmonary embolism has also been demonstrated to be efficacious in patients who were in a state of imminent cardiovascular collapse [15].

In Case 1, severe pulmonary hypertension due to multiple pulmonary emboli and the right heart migrant thrombotic mass, was judged to require additional therapeutic intervention supplementary to anticoagulation. Logistical problems prohibited thrombectomy, so that thrombolytic therapy was chosen. In Case 2, the right intracavitary thrombotic mass dislodged into the pulmonary trunk whilst the patient was adequately anticoagulated. For this precarious clinical condition, we again chose thrombolytic therapy despite the short postoperative status. In this patient, we retained the option of mechanical clot fragmentation. Since both our patients were critically ill and showing a type A mobile thrombus, whereas the operative risk was considerably high, we felt aggressive thrombolytic therapy was justifiable.

In our opinion, these case reports suggest a new first-line treatment in qualifying patients with right heart emboli-in-transit. Adjunctive thrombolytic therapy may lead to a quick clinical recovery and should, therefore, be considered provided there is an absence of contraindications. Surgical pulmonary embolectomy may be regarded as a treatment of last resort in patients with firm contraindications for thrombolytic treatment after failure of mechanical clot fragmentation.

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