Saddle pulmonary embolism with normal right ventricular function: a treatment enigma

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We present five cases of saddle pulmonary embolism with normal right ventricular systolic function and discuss the management option of this condition.

Introduction

Management of saddle pulmonary embolism (PE) in haemodynamically stable patients with normal right ventricular (RV) function is unclear.

Thrombolytic agents are indicated in haemodynamically unstable patients with PE.[1] RV dysfunction has also been used as an indication for surgical embolectomy in normotensive patients with PE.[2] Embolized thrombus burden, although intuitively related both to haemodynamic status and RV dysfunction, has not been consistently linked to prognosis. In particular, an unsettled issue is the prognosis and benefit of aggressive treatment including thrombolytic agents in hemodynamically stable patients with large pulmonary embolism in the main pulmonary artery (saddle PE) who have no evidence of RV dysfunction. We discuss the management of five patients with saddle pulmonary embolism who were haemodynamically stable and showed normal RV function.

Case histories

Case 1

A 66-year-old woman undergoing chemotherapy for bilateral breast cancer presented with dyspnoea. On admission, her blood pressure (BP) was 169/99 mmHg, oxygen (O_2) saturation was 93% on room air (RA) with no signs of heart failure. A transthoracic echocardiogram (TTE) showed normal RV size and function with normal left ventricular ejection fraction (LVEF) at 55–60%. Computed tomographic (CT) angiogram scan showed a saddle embolus partially extending in both pulmonary arteries. A Doppler ultrasound of the lower extremities showed a deep vein thrombosis of the left popliteal vein. Unfractionated heparin (UFH) was given and an inferior vena cava (IVC) filter was deployed. The patient was discharged home on warfarin. She died 8 months later due to progression of cancer.

Case 2

A 62-year-old woman who underwent surgery for malignant meningioma presented with chest pain and dyspnoea. On admission her BP was 110/61 mmHg, O_2 saturation of 97% on 4 L of supplemental O_2 and showed no signs of instability. She was diagnosed with lower extremity deep venous thrombosis one week earlier and treated with UFH and warfarin. However, due to an elevated international normalized ratio (INR) of 5.73 and partial thromboplastin time >100, warfarin was withheld. A TTE showed normal RV size and function with an estimated right ventricular systolic pressure (RVSP) at 25 mmHg, and normal LVEF at 55–60%. A 12-lead electrocardiogram (ECG) showed sinus tachycardia at 101 beats per minute (bpm). A CT scan of the lungs showed saddle pulmonary embolus. Low molecular weight heparin (LMWH) was given and an IVC filter was deployed. The patient was discharged home on LMWH. She died 1 month later due to progression of cancer.

Case 3

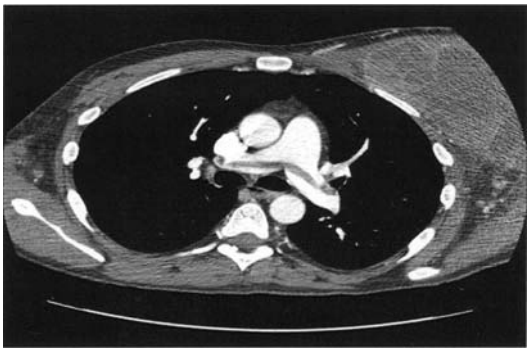
A 37-year-old man who was receiving treatment for metastatic melanoma presented with

dyspnoea. On examination his BP was 100/70 mmHg with no signs of RV failure. A 12-lead ECG showed sinus tachycardia at 107 bpm and a new right bundle branch block. A TTE showed normal RV size and function with an estimated RVSP at 30–40 mmHg and normal LVEF at 60–65%. A CT scan showed saddle embolus extending into the right and left pulmonary artery (Figure 1). Patient received one dose of tissue plasminogen activator (TPA) and was started on LMWH and IVC filter was placed. After a suspected episode of heparin-induced thrombocytopenia, the patient was placed on lepirudin. The patient was eventually discharged home on warfarin. A follow-up CT scan showed progressive reduction in the size of the embolus. The patient died 5 months later due to progression of cancer.

Case 4

A 65-year-old man who was undergoing chemotherapy for glioblastoma multiforme presented with dyspnoea. On examination, his BP was 127/97 mmHg, O_2 saturation was 98% on room air and appeared stable. A 12-lead ECG was normal. A TTE showed normal RV size and function, with mildly increased RVSP at 30–40 mmHg and a normal LVEF at 55–60%. A CT scan showed saddle embolus and multiple smaller peripheral emboli. The patient was started on LMWH and was discharged home. Patient died 3 months later due to progression of brain tumor.

Figure 1
Saddle pulmonary embolus



Case 5

A 71-year-old man with a recent compression fracture due to multiple myeloma presented with chest pain and dyspnoea. He had been diagnosed with deep vein thrombosis 2 months earlier and had been receiving warfarin. The INR was 1.12 when he presented to the hospital. On admission his BP was 135/81 mmHg and showed no signs of heart failure. An arterial blood gas showed a pH of 7.46, PO2 of 56 mmHg and a PCO2 of 31 mmHg. A TTE showed normal RV size and function with mild pulmonary hypertension (RVSP at 30–40 mmHg) and normal LVEF at 60–65%. CT scan showed a large saddle embolus extending into right and left pulmonary artery. The patient was started on UFH and TPA. TPA was discontinued due to abrupt onset of chest pain during its administration. A repeat CT scan after 2 days showed a decrease in size of the embolus. The patient was later discharged home on warfarin. The patient is still alive 7 years after the episode of saddle PE.

Discussion

PE is a leading cause of death among hospitalized patients.[3] Mortality in PE is related to RV dysfunction and hemodynamic instability. Thromboembolic obstruction of the PA and vasospasm due to hypoxia increases the pulmonary vascular resistance and leads to RV pressure overload. This in turn causes RV dilatation, hypokinesis or even failure.[4] Decrease in RV output reduces left atrial filling, which in turn compromises LV preload. In addition, RV enlargement causes leftward shift of interventricular septum during diastole leading to additional impairment in left ventricular filling and further decrease in cardiac output.[4]

The largest PE, termed saddle pulmonary embolism is defined as thromboemboli at the bifurcation of the pulmonary trunk with or without extension into the left and/or right main pulmonary arteries.[5,6] While saddle PE may usually cause RV dysfunction, up to 45% of stable patients with saddle PE have normal RV function.[6] Even in the absence of RV dysfunction, saddle PE is associated with a higher one year mortality, when compared to large non-saddle PE.[6]

RV dysfunction is a major determinant of prognosis in both hemodynamically stable and

unstable patients with PE.[7–9] Short-term mortality is high in normotensive patients with RV dysfunction than in patients with normal RV function.[7] Furthermore, one-year mortality in patients with PE and RV strain at presentation was 13% and 1.3% in those without RV strain.[8] In another study there was two-fold increase in mortality in patients with right ventricular hypokinesia.[9]

The treatments for saddle PE beyond anticoagulants may include thrombolytic therapy (systemic and intra-luminal via catheter) and surgical embolectomy (open and percutaneous).[6] Thrombolytics are recommended in patients with PE and hemodynamic instability.[1] The data for thrombolytic therapy even in this subset of patients are sparse, with only one randomized trial of eight patients in cardiogenic shock showing mortality benefit with streptokinase.[10] In patients with PE, thrombolytics improve RV function.[11] In a case of saddle PE, thrombolytics has been reported to improve and normalize RV function.[12] Whether this will result in mortality benefit is unknown, as there are no randomized trials of thrombolytics in saddle PE. The limitations of published studies on saddle PE are small sample size and inconsistent management strategies. The total number of patients included in the studies of saddle PE has ranged from 17 to 22 patients.[6,13,14]

Although thrombolytics was used in two patients in our series, long-term prognosis could not be assessed as four of them died within a few months due to progression of cancer. Whether thrombolytics or pharmacological reduction of pulmonary hypertension in patients with saddle PE would improve late mortality needs further exploration.

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