

# Saddle Pulmonary Embolus Successfully Treated With Unfractionated Heparin

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## Abstract

Antemortem diagnosis of a saddle thromboembolus at the bifurcation of the main pulmonary artery (MPA) is rare<sup>1</sup>. Such proximally located thrombi are regarded as unstable, and may fragment spontaneously or following treatment, leading to obstruction of distal pulmonary arteries. We describe a case of a saddle embolus of the MPA successfully treated with intravenous unfractionated heparin (UFH), followed by oral anticoagulation for three months with complete resolution of the thrombus.

## CASE REPORT

A 42 year old male presented to the Emergency Department with sudden onset of left sided chest pain and transient syncope with spontaneous recovery. He had previously undergone right Achilles tendon repair 3 weeks prior to admission with no peri-operative deep vein thrombosis prophylaxis.

On admission the patient was pain free, afebrile with a normal pulse and blood pressure. He had no respiratory distress with arterial blood saturations of 99% on air. Both systemic examination and twelve-lead ECG were normal. Apart from a mildly elevated troponin his blood tests including a thrombophilia screen and homocysteine levels were all normal.

In view of his recent orthopaedic surgery and immobilisation, the diagnosis of acute pulmonary embolism (PE) was suspected; so both transthoracic echo (TTE) and CT pulmonary angiogram (CTPA) were obtained. TTE revealed normal left and right ventricular size and function, with no evidence of pulmonary artery hypertension or thrombus. CTPA confirmed a large saddle thromboembolus at the bifurcation of the MPA, crossing the left and right pulmonary arteries (Figure 1).

## Figure 1

Figure 1: CT pulmonary angiogram (axial slice) revealing thrombus (arrowed) in the main pulmonary artery at the saddle extending across the origin of both right and left pulmonary arteries.



As the patient was hemodynamically stable, without signs of right ventricular pressure overload, we elected to treat him with intravenous UFH, administered at 1000 U/hour for 6 days maintaining the APTT to 2-2.5 times the upper limit of normal. He was commenced on warfarin therapy from day 2 (targeted to maintain INR of 2.5-3). Heparin was discontinued once we had achieved a therapeutic INR for 2 days. The patient remained asymptomatic and was discharged the following week. Prior to discharge the patient underwent pulmonary function tests and a repeat TTE, both

of which were normal. Repeat CTPA imaging 3 months later showed complete resolution of the saddle PE with no residual thrombus apparent (Figure 2).

### Figure 2

Figure 2: Repeat CTPA at 3 months confirming complete resolution of the thrombus.



### DISCUSSION

Saddle (or acute massive) PE can be a potentially fatal occurrence. This condition was generally recognised only at necropsy but as diagnostic imaging modalities like CTPA have become widely available, the diagnosis has been increasingly made ante-mortem. In a retrospective analysis of 289 patients with CT-confirmed PE, saddle thromboembolism was present in 2.5% of cases<sub>2</sub>. Such proximal thrombi may fragment spontaneously or secondarily following treatment (eg. thrombolysis) potentially causing multiple occlusions of distal pulmonary arteries, which may adversely affect the hemodynamic status of the patient. Generally patients who present with a saddle PE tend to either be haemodynamically compromised (hypotensive or tachycardic) or have evidence of right ventricular strain or pressure overload on TTE. In these circumstances the recognised therapeutic options include urgent surgical pulmonary embolectomy<sub>3</sub> or thrombolysis<sub>4</sub>

with their inherent risks. Percutaneous intervention using either capture devices or a rotational pigtail catheter has also been tried in a few cases with success<sub>5</sub>. However, the management of large saddle PE without haemodynamic compromise remains controversial with no uniform treatment consensus. As our patient exhibited no signs or symptoms of haemodynamic instability following admission, we opted successfully for a less aggressive approach using intravenous UFH, a recognised treatment option for peripheral PE, followed by oral warfarin with no adverse outcome.

### CONCLUSION

Patients with massive saddle pulmonary embolus may present without haemodynamic compromise, respiratory distress or obvious RV pressure overload. Systemic anticoagulation using intravenous UFH followed by oral warfarin therapy with repeat imaging to monitor the status of the thrombus, remains a reasonable treatment strategy for such patients.

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