

# Pulmonary Embolus: The Wrong Thing On The Wrong Journey

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## Abstract

While it may not be possible to obtain the correct figures worldwide, it certainly is a matter of grave concern that a large number of people loose their lives cheaply. While majority emboli are small, the larger ones have an acute course and may lead to cor pulmonale and death. Pulmonary embolus, put simply, is "body friendly blood cells getting together for some sort of a meeting and forming a thrombus; and this thrombus suddenly decides to go out for a roller coaster ride in the vasculature", which for 10% of people turns out to be fatal. Of the people who survive, as many a third will have their thrombi going for a second adventure ride.

## INTRODUCTION

While it may not be possible to obtain the correct figures worldwide, it certainly is a matter of grave concern that a large number of people loose their lives cheaply. While majority emboli are small, the larger ones have an acute course and may lead to cor pulmonale and death. Pulmonary embolus, put simply, is "body friendly blood cells getting together for some sort of a meeting and forming a thrombus; and this thrombus suddenly decides to go out for a roller coaster ride in the vasculature", which for 10% of people turns out to be fatal. Of the people who survive, as many a third will have their thrombi going for a second adventure ride.

## DEFINITION

Pulmonary embolism put simply is the lodging of a thrombus in the pulmonary artery or its branches. Before considering the various aspects, it will be useful to deal with the differences between a clot and a thrombus. A clot is a cast, which sets in the vessel, post mortem due to the settling of blood constituents. A thrombus (which potentially can embolize), on the other hand is the deposition of the platelets and red cells in the vessel ante mortem. Currant jelly appearance of a clot is due to settling of red cells in the dependent parts of the vessel, while chicken fat comprises of

the serum so separated. Lines of Zahn are the concentric entanglements of platelets in a thrombus. Clots do not adhere to the vessel wall, while thrombi do. It is such thrombi, which when become friable get dislodged and travel in the vasculature.

## EPIDEMIOLOGIC OVERVIEW

Pulmonary arteries (main or its peripheral branches) almost always get occluded following the lodging of embolus coming from the deep venous system of the lower extremities. An overwhelming majority (more than 95%) of the emboli have their origin in deep veins of lower extremities and peri pelvic plexus. The incidence of PE is highly variable with respect to whether general population or hospitalized population is being looked at. Autopsy findings suggest the incidence to be about 30% in patients with burns or major trauma. About two thirds of autopsies of patients who were hospitalized have shown to have PE. Prolonged hospitalization or convalescence is certainly a major cause of PE. Other causes include long hours of driving, hyper coagulable states, burns etc. One in ten victims loses his life to PE and of the surviving patients, a third get PE again.

## PATHOGENESIS

As is evident a vast majority of emboli have their origin in the deep venous system, in the form of a thrombus, it is

important to understand why in the first place, such a thrombus forms. Arterial thrombi are composed of platelets with some fibrin and a few red cells. Relative paucity of red cells renders them pale. Pulmonary emboli arise from venous thrombi. Venous thrombi are composed of red cells, white cells and platelets woven together by a large amount of fibrin (however tumor thrombi, amniotic fluid, air, fat, marrow, and many iv materials may also cause PE, but they do so in a minority of cases). These are typically red in color. We will restrict our discussion only to the venous thrombi. The process leading to thrombus formation can be understood by considering Virchow's triad. To put simply the following three factors aid in the forming of a thrombus:

**STASIS:** For any reason, if the normal flow of the blood is so influenced that it becomes sluggish, red cells & platelets get an occasion to weave up a thrombus. Such conditions include prolonged immobility like long hours of uninterrupted travel in airplanes, cars, buses, immobility due to convalescence etc. Hence it is always recommended to take periodic breaks while traveling long distances and make sure to move body parts if status post op or bedridden due to any other cause.

**ENDOTHELIAL DAMAGE:** Any nature of endothelial insult can further predispose to thrombus formation. Such an injury causes the activation of complement cascade and trigger thrombus formation. Artherosclerosis, arteritis, phlebitis are such contributors.

**HYPERCOAGULABLE STATE:** The following gives an overview of the various conditions, which cause the blood to be hypercoagulable.

Deficiency of Protein C or Protein S is one of the many reasons the blood goes disobedient. Autosomal Dominant in inheritance, this state prevents adequate inactivation of Factors Va and VIIIa.

Deficiency of anti thrombin III is also autosomal dominant. Anti thrombin III (ATIII) is responsible for keeping a check on thrombin and activated forms of Factor IX and X. When ATIII is inadequate quantitatively or qualitatively, the thrombus formation is promoted.

Disseminated cancers, paraneoplastic conditions, blood dyscrasias (polycythemia, thrombocytosis etc.) also cause hypercoagulability.

The use of Estrogens is also associated with Hypercoagulable State due to lowered ATIII concentrations

and increased concentrations of coagulating factors.

Physiological states like pregnancy, smoking and advanced ages are also predisposing factors.

When finally the stage is all set for the thrombus to form, red cells, platelets and white cells get enmeshed with fibrin. Thrombus so formed gets dislodged en masse or more commonly a part of it and sets for the most undesirable journey. This embolus meanders along the venous system and goes to the right side of the heart. Finally, right ventricle shows the embolus its final (read potentially fatal) destination and pulmonary artery is occupied. The size of this embolus and the site of occlusion determine the extent of damage caused by PE.

While formation of the embolus is the event, we are mainly concerned here, it is important to appreciate that thrombi are more likely to be cleared by the fibrinolytic activity. Other fates of thrombi are recanalization and propagation.

**CLINICAL COURSE**

The course is primarily influenced by the size of the embolus and the site it occludes. Prior cardiopulmonary status also dictates the clinical course. Fortunately most emboli are not large enough to occlude the bifurcation of Pulmonary artery and may cause limited compromise of the lung tissue.

Clinically, the incidence of symptoms and signs of angiographically proven PE suggests the following distribution:

**Figure 1**

Table: Incidence of Symptoms and Signs of angiographically proven PE (3)

Symptoms	Incidence (%)	Signs	Incidence (%)
Chest Pain	88	Resp. rate >16	92
Dyspnea	84	Resp. Crackles	58
Apprehension	59	Accentuated S2P	53
Cough	53	Pulse > 100 per min.	44
Hemoptysis	30	Temperature>37.8 C	43
Sweating	27	Phlebitis	32

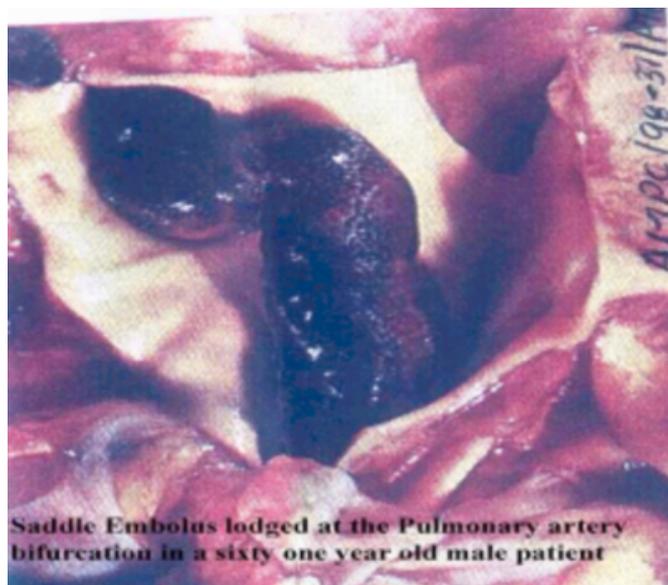
Thrombi that are not very large, travel through the pulmonary artery and are lodged in the peripheral vessels (which is a more common event than the emboli actually completely occluding the pulmonary artery). This embolus fortunately in a large majority of cases is clinically not very significant; but in unfortunate few, pulmonary infarction occurs. PE will cause infarction in the setting where pulmonary circulation is compromised or there is attendant cardiopulmonary lesion. Generally, the bronchial arteries take care of the lung parenchyma. In such a setting infarction is quite unlikely but hemorrhage may occur. Repeatedly,

such small emboli may come to lodge in the peripheral vessels and this causes the peripheral areas of lung parenchyma to bathe with a shower (generally that of the lower lobes). It is important to appreciate the dual blood supply of the lung (bronchial arteries and pulmonary artery). Such an arrangement may prevent eventual necrosis of lung parenchyma. Hemorrhagic infarction with ischemic necrosis is the likely fate of the affected portion. Infarction typically appears in a wedge form with the base at the lung periphery and the apex towards the hilum.

The fate of a large embolus is much different. A large embolus hardly ever gives lung any time to adapt to the stress. More often than not, a saddle embolus (defined so because a large embolus comes from the venous vasculature and sits astride the bifurcation of pulmonary artery) turns out to be fatal. Lungs humbled by a large embolus do not enjoy the privilege of being showered by emboli gradually and PE in this comes as a blow. Right heart tries its best to pump out the blood, but in vain. Eventually cor pulmonale ensues and all this happens in a short time with no tell tale histologic features in the lung. Lungs appear normal histologically and sudden death ensues.

### **Figure 2**

Figure 1: Saddle embolus lodged at the pulmonary artery bifurcation as seen on autopsy of a sixty one year old male patient. Reproduced with permission, from Dr. Dilip Jindal, MS, New Delhi; Personal Collection (5)



### **DIAGNOSIS**

A large number of lives are lost due to delayed diagnosis and the acute course of large embolus. Unfortunately, it is not always possible to make the correct diagnosis and intervene

as the course is very acute and PE is often a finding in autopsy results. In patients identified to be at risk, abrupt onset of dyspnea, apprehension, chest pain, sweating and hemoptysis should strongly indicate PE.

The most prudent diagnostic tool for a provisional diagnosis appears to be the history. However, history only contributes in making a diagnosis; a thorough work up is required for a final diagnosis. Knowledge about patient having undergone some surgery or being immobilized on some account is certainly desirable. Any family history of hypercoagulable states, history of long distance travels, cancers, pregnancy, use of contraceptive pills etc. contribute in arriving at a conclusion. Radiologic techniques remain the mainstays of diagnoses. For the diagnosis of DVT, venography is the gold standard. Lung scans are generally the first step towards the diagnoses and may show V/P mismatch (with adequate ventilation and very little perfusion). Sensitivity of lung scan is as high as 90% if two perfusion defects are identified. A normal scan excludes the diagnoses while a high probability deserves an immediate treatment. For the low probability or indeterminate scans, Doppler study and/or arteriogram may be required.

### **PREVENTION AND TREATMENT**

When a patient is identified to be at risk, appropriate antithrombotic measures should be instituted.

Administration of heparin remains the mainstay of treatment, anticoagulation is desirable in appropriate patients.

For the prevention of PE, the risk factors need to be identified. As obesity & smoking are identified risk factors, weight loss and smoking cessation are recommended. An obese patient or a lady who is at risk of DVT should consider contraceptive methods other than Estrogen containing pills. It is important to emphasize role of frequent moving of immobilized patient. Early ambulation helps avert development of PE. Long journeys need to be interrupted to avoid stasis. If at increased risk, prophylactic low dose heparin is administered before surgery. Elastic stockings & isometric exercises are also recommended post op. Insertion of venous filter may be considered in appropriate candidates. However, PE continues to be a significant cause of mortality and it is desirable that an early diagnoses and emergent management be done. If hypercoagulable states or other risk factors are identified, it is extremely important to prevent blood cells to get rebellious and make thrombi which I due course can dictate cor pulmonale and death; which in many cases can be achieved with adequate prophylactic measures.

(DISCLAIMER: The authors do not intend to recommend any self-medication. The references made to medical or surgical interventions or any alterations in lifestyle are a part of this paper and should be resorted to only under supervision. Authors are not responsible for any damages whatsoever that might arise out of any injudicious step taken by the reader).

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