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Acute Pulmonary Embolism - A Clinical Review.

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ABSTRACT

Acute pulmonary embolism is a life threatening condition with a high mortality rate. If diagnosed early, the patient outcome is better. Therefore, early assessment, diagnosis and appropriate treatment are a key to successful management of the patient. This article discusses two (2) cases of acute pulmonary embolism of different aetiologies and also undertakes a review of the literature with regards to classification systems, risk stratification, diagnostic tools and recent advances in management of pulmonary embolism.

Keywords: Acute pulmonary embolism, etiology, deep venous thrombosis, management

INTRODUCTION

Acute pulmonary embolism (PE) is an acute entrapment in pulmonary arteries of dislodged thrombus usually from deep veins of legs, pelvis or arms. It is life threatening and can result in right heart failure low cardiac output and sudden death.[1] Due to its variable initial presentation the true incidence of PE is difficult to estimate.

Dr.John Gibbon[2], designed cardiopulmonary bypass (CPB) after observing a patient of massive pulmonary embolism when he was a Research fellow in Massachusetts General Hospital.

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Studies published by De Monaco et al[3] in 2008 and Wiener et al in 2011 both demonstrated significant increases in the incidence of PE while severity of illness decreased over this period of time. The authors postulated that over diagnosis explains the rapid rise in the incidence of PE because most of the cases are clinically unimportant and non- fatal. Over diagnosis is important to recognise because treatment of PE has its own risks, especially in whom PE is unimportant.

MATERIALS AND METHODS

Material is gathered from Kirklin, Pubmed, Google, Journal of clinical trial results, case reports. Studies from peer-reviewed sources are included.

CASE REVIEW

We report two patients who presented with acute massive pulmonary embolism, the mode of presentation, haemodynamics and etiology of both the cases is different.

Case 1 – 48 year old female patient who is a known case of carcinoma uterus, post-hysterectomy and radiotherapy, presently on chemotherapy admitted for staging laparotomy. On third post-operative day, patient developed shortness of breath with a respiratory rate of 54/min, bradycardia, raised JVP. In view of her previous history and her clinical presentation, acute PE was suspected. She was started on intravenous infusion of UFH, while awaiting diagnosis. Patient underwent emergency CTPA, which confirmed the diagnosis. But, after 24 hours patient succumbed. Thrombolysis was deferred in view of her medical history fearing bleeding.

Case 2 – A 44 year old female patient was referred from a primary center with a history of swelling of the right lower limb for two weeks, with a recent onset of dyspnoea with increasing severity. On admission, suspecting PE, CTPA was done which showed massive embolism extending from main pulmonary artery into lobar branches on the left side with evidence of multiple infarcts in the left lung. Doppler of lower extremities revealed proximal VTE. As the patient presented late in the course of disease, and was also hemodynamically stable she was started on anti-coagulation with unfractionated heparin .But patient continued to deteriorate. Suspecting that she was throwing emboli from lower extremities an IVC filter was planned. As the patient

and attendants were unwilling, the procedure was delayed during which time we lost the patient.

DISCUSSION

The common cause in most cases of pulmonary thromboembolism is detached venous thrombi from the lower limbs and rarely upper limbs, passing through the right heart and entering the pulmonary arteries as a single thrombus or as a fragmented smaller thrombi. Majority lodge in the lower lobes, slightly more in the right than left lung. Shortly after reaching the lungs these emboli get coated with layer of platelets and thrombin. In addition to causing pulmonary arterial obstruction, the platelets on thrombus release vasoactive agents, which elevate pulmonary vascular resistance (RP). Alveolar dead space increases as a result of redistribution of blood flow and V/Q mismatch occurs. Right ventricular (RV) after load increases, RV pressure rises. This results in RV dilatation, ischemia and dysfunction. Increased RP results in reduced RV stroke volume and left ventricular preload. Reduction in preload leads to systemic hypotension and reduction coronary blood flow. This markedly reduces left ventricular stroke volume. If a patent foramen ovale or ASD is present, right to left shunting of blood and severe hypoxemia may occur. Paradoxical embolization may also occur.

Rudolf Virchow[5] has first described the pathophysiology of venous thromboembolism. The Virchow's triad describes the factors contributing to the formation of venous thrombi consisting of hypercoagulability, stasis and endothelial injury. Risk factors for venous thromboembolism are listed in [Table 1].

Table 1: Risk factors for venous thromboembolism.

Table 1. Risk Factors for Venous Thromboembolism (VTE)
Primary
• Idiopathic
• Age > 65 years
• Thrombophilia or inherited hypercoagulable state
• Obesity
• Previous VTE
Secondary/acquired
• Immobility or paralysis
• Trauma especially major limb trauma
• Recent surgery
• Pregnancy
• Oral contraceptive therapy
• Cancer
• Major medical illness (myocardial infarction, stroke, etc.)

Venous stasis is a prominent contributing factor for the formation of deep venous thrombosis (DVT)[6]. Thrombus formation occurs in venous valve pockets due to a combination of slow flow and endothelial inflammation caused by blood flow turbulence within these pockets. These factors when combined with prolonged immobility can increase risk of deep venous thrombosis[7].Clots formed in deep veins behind or proximal to knee are more likely to result in fatal PE, than are clots distal to the popliteal vein(Kearon 2003)[7]. This is probably true because thrombi that originate in calf are small and thrombi that embolise from proximal leg veins are usually large.

Pulmonary emboli can be detected in approximately 50% of patients with documented DVT (Hull, 1983; Plate, 1985). symptomatic DVT is seen in 70% of patients with documented and diagnosed PE (Hirsch and Hoak, 1996). The possibility of PE due to causes other than lower limbs has been suggested by Ogren 2005.

The cause of DVT is multifactorial .Molecular factors include inherited or acquired hyper-coagulable states[8]. Risk factors being major surgery, advanced age, prior VTE or family history, cancer major trauma, spinal cord injury, pregnancy, chronic medical illness and oral contraceptives.

Classification:

Pulmonary embolism is classified as minor, major, massive[1] Massive PE may be defined as acute PE with sustained hypotension, pulselessness, or profound bradycardia with symptoms of shock. Major PE is defined as acute PE without systemic hypotension but with either RV dysfunction or elevated troponin I or T indicative of myocardial injury. Minor PE is defined as acute PE without clinical markers that define massive or major PE.RV dysfunction may be defined as RV systolic function on echocardiography[17], RV dilatation RV/LV diameter >1, and EGG changes.

CLINICAL PRESENTATION

Clinical presentation of PE is variable and nonspecific. Classically patients present with atypical chest pain, dyspnoea and haemoptysis. But these symptoms are present in less than 20% of patients as the PIOPED study showed.

The haemodynamic consequences of PE depend not only on the size and number of emboli, but also on the pre-existing cardiac and respiratory status (European Society of cardiology, 2000). Acute PE causes both respiratory and cardiac compromise. Respiratory function is compromised by obstruction of pulmonary arteries, which elevates pulmonary vascular resistance. The increase in alveolar dead space and alteration in ventilation perfusion mismatch impair gas exchange .Cardiac failure results from increased wall stress, cardiac ischemia RV dysfunction. Significant RV damage can produce elevated troponin levels (Meyer 2000).

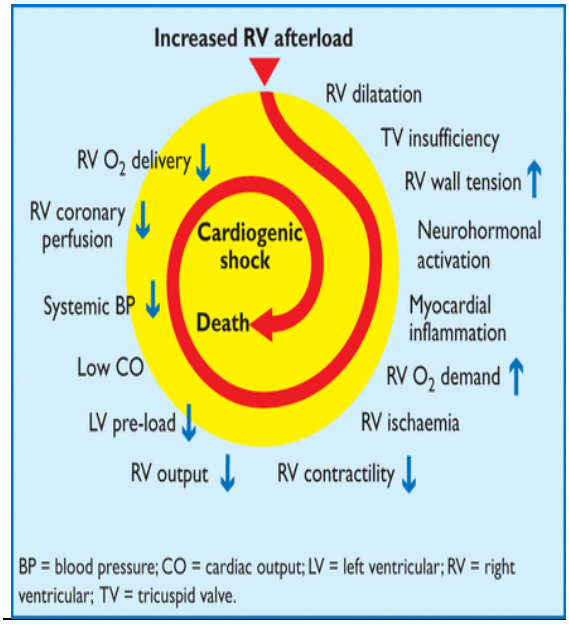

Figure 1: ?????????

DIAGNOSIS

Pulmonary embolism can be a severe and fatal disease but difficult to diagnose as the signs and symptoms are nonspecific. But the investigations used to diagnose PE at times may over diagnose the condition leading to potential harm and unnecessary expense.

Therefore, a few variables have been combined to form prediction rules to predict the probability of PE. The most commonly used are WELLS SCORE and revised GENEVA RULES [Tables 2 & 3].

Table 2: Wells Score

Variable	Points
Clinical Signs and symptoms of deep venous thrombosis(DVD) (Leg swelling and tenderness)	3
Alternate diagnosis less likely than pulmonary embolism	3
Heart rate more than 100 bpm	1.5
Immobilisation or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy	1
Calculation of Clinical Probability	***Total***
Low	<2
Intermediate	2 to 6
High	>6

Table 3: Revised Geneva Score

Risk Factors	Points
Age >65 years	1
Previous DVT or PE	3
Surgery or fracture of lower limbs within the past month	2
Active Malignancy	2
Symptoms	
Unilateral lower limb pain	3
Haemoptysis	2
Clinical or physical exam signs	
Heart rate 75 to 94 BPM	3
Hear rate >95BPM	5
Lowe limb pain on deep venous palpation and unilateral oedema	4
Clinical Probability	Total
Low	0 to 3
Intermediate	4 to 10
High	>10

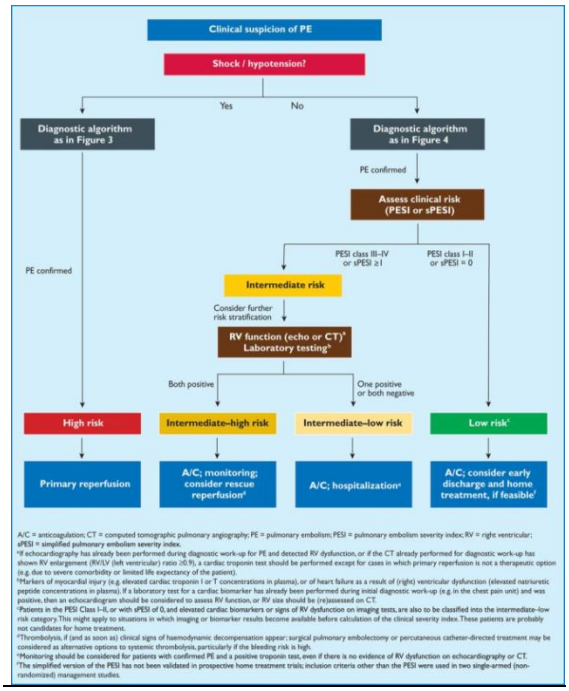

Figure 2: ?????????

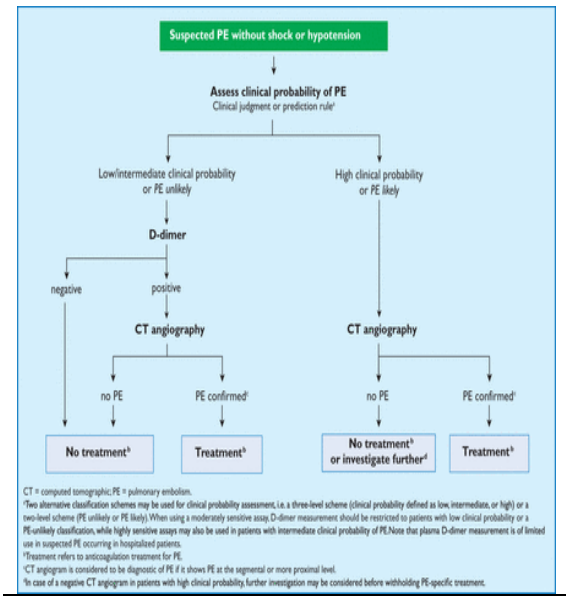

Figure 4: ??????????

CT SCAN:

Table 4: Imaging rationality

Clinical Situation	Basis for Imaging Action (Reference)
Immediate CT	
Hemodynamically unstable, with suspected PE*	Risks of inaction outweigh risks of CT
High pretest probability of PE	Incidence of PE 19%-28% even with a D-dimer level <500 ng/mL (7, 74)
Defer CT until after D-dimer result	
Intermediate pretest probability	Low incidence of PE (<1.1%) if D-dimer level <500 ng/mL (41-43)
Low pretest probability and PERC > 0	
No CT or D-dimer test	
Low pretest probability and PERC = 0	Incidence of PE <1% (47)
Begin with lower-extremity venous ultrasonography	
Patients with symptoms of DVT and PE	Similar treatment will be pursued without exposing the patient to the risks of radiation or intravenous contrast

CT = computed tomography; DVT = deep venous thrombosis; PE = pulmonary embolism; PERC = Pulmonary Embolism Rule-Out Criteria.
* Hemodynamic instability may make transport for imaging problematic. Supportive measures or empirical anticoagulation until imaging can be obtained may be required.

Arterial blood gas analysis:
It may show respiratory alkalosis and hypoxaemia.

Echo cardiogram:
Though not diagnostic of PE, it can be helpful in determining the risk of mortality or adverse outcomes. It helps in diagnosing RV dysfunction and failure. It may also be used to visualise patent foramen ovale in cases of paradoxical systemic embolism and also any thrombi. The response to treatment can be evaluated by the improvement of RV function.

Ventilation and perfusion scan:
It is less expensive and less invasive than pulmonary angiography. A perfusion defect without a corresponding ventilation defect has a high probability of PE. A perfusion defect with matched ventilation has a low probability of PE. Matched perfusion defects corresponding to those on chest X-ray as in COPD have an intermediate probability.

Pulmonary angiography:
It was the gold standard for diagnosing PE. But the cost, requirement of qualified personnel to do the procedure, the risks involved in the procedure and the availability of less expensive, non-invasive methods made this procedure obsolete.
CTPA:
It is being extensively used in the diagnosis of PE. The condition is seen as a filling defect. Radiation is a drawback of this investigation.

MR Angiography:
It is being used in situations where CTPA is contraindicated as in contrast allergies and in pregnant women.

D Dimers:
This is not a specific test for PE, but a normal or negative D dimer in a low probability situation excludes PE.

Cardiac Troponins:
Presence of cardiac enzymes along with PE was associated with adverse outcome as their presence indicate RV stress and failure. Along with H-FABP, and BNP these indicate RV strain and dysfunction.

Venous duplex ultra sound:
Along with compressibility this investigation is used to diagnose DVT in peripheral limbs. In a non-diagnostic V/Q scan a negative venous duplex rule out PE.As such a venous ultra sound is recommended in situations where there is low probability of PE.

TREATMENT

Anticoagulation is the mainstay of treatment for acute PE, the objectives being prevention of thrombus extension and recurrence of VTE. Rapid anticoagulation can be achieved by using parenteral anti-coagulants as UFH, LMWH, or fondaparinux. In cases of high probability and while awaiting confirmation this therapy may be considered in the absence of contraindications.

Thrombolysis:
Achieves clot lysis more rapidly than anticoagulation. It reduces pulmonary vascular resistance, improves pulmonary perfusion, haemodynamics and gas exchange. Rapid clot reduction also reduces the risk of chronic pulmonary hypertension.

Pulmonary embolectomy:
It is used to relieve acute obstruction in emergency situations when thrombolysis is contra indicated or not practical. Mortality with this procedure is high and can be associated with complications like ARDS, mediastinitis, ARF, and severe neurological sequelae.
Massive PE patient usually presents in emergency block in a decompensate state. High clinical

suspicion is warranted. After stabilisation and confirmation of diagnosis thrombolysis either systemic or catheter directed and/or mechanical thrombectomy percutaneously or surgical embolectomy are preferred.

Intravenous thrombolysis has been shown to reduce risk of death and recurrent PE significantly but GI bleeds and intracranial or retroperitoneal bleeds are frequent than warranted. Unfractionated heparin should be used for anti-coagulation.

Catheter directed thrombolysis and mechanical thrombectomy has been recommended in patients with proximal PE with absolute contraindications to systemic thrombolysis or surgical embolectomy, or those who failed thrombolytic therapy. Another device used to treat massive PE is the Ekosonic ultrasound assisted thrombolysis infusion device. A recent publication has shown that ultrasound assisted thrombolysis resulted in improvement of clinical symptoms and RV dysfunction without major bleeding complications.

The main stay of treatment in for acute PE in nonmassive i.e., minor and major PE is systemic anticoagulation. The standard regimen is a course of LMWH, or Fondaparinux for 5 days with VKA started on third day of anticoagulation to maintain a INR between 2 and 3. UFH has to be given as a continuous infusion, and bleeding complications are also more. Compared to LMWH the incidence of HIT is high with UFH.

Direct thrombin inhibitors as hirudun, argatroban, melagatran, ximelagatran are not first line of treatment in PE, but are being investigated.

Inferior venecava filters:

These filters function to prevent large emboli from lower extremities from reaching lungs. Candidates for IVC filter placement are those who have DVT and have contraindications to anticoagulation, recurrence despite adequate anticoagulation, inability to achieve or maintain therapeutic anti coagulation. Many trails have been done like PREPIC, DENALI, but there is no uniform consensus on usage of IVC filters in PE. Use is determined by case to case basis.

Long-term treatment:

The aim of long term treatment is to prevent fatal and nonfatal recurrence of VTE.VKA's are given to maintain INR at 2-3. They remain the treatment of choice for long term. The duration depends on the risk of recurrent VTE after discontinuation, patient's demographics, and the risk of bleeding.

CONCLUSION

Pulmonary embolism is a common and fatal disease. The two cases discussed above emphasize the fact. Early and correct diagnosis is important. Diagnosis is difficult as the presentations are varied and nonspecific. A high degree of clinical suspicion is necessary to come to an early and accurate diagnosis. At the same time over diagnosis is also harmful subjecting the patients to unnecessary investigations, which in themselves are harmful. The American college of physicians has issued the best practice advice for evaluation of patients with pulmonary embolism. Institution of appropriate treatment saves many lives. The corner stone of therapy remains anticoagulation with Heparin. Surgical intervention is reserved in those who are unstable and anticoagulation and/or thrombolysis is contraindicated. Newer strategies, drugs and devices to treat PE continue to emerge.

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