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Acute Pulmonary Embolismfollowing Polytrauma

Vitharana HS[1*], Somaweera S[2]

[1]Clinical Fellow, Anaesthesia and Critical Care, Gloucestershire Hospitals NHS Foundation Trust, United Kingdom, Former- Senior Registrar in Critical Care, National Hospital, Colombo, Sri Lanka

[2]Consultant Anaesthetist, National Hospital, Colombo, Sri Lanka

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[*]**Corresponding author:** Vitharana HS, Clinical Fellow, Anaesthesia and Critical Care, Gloucestershire Hospitals NHS Foundation Trust, United Kingdom, Former- Senior Registrar in Critical Care, National Hospital, Colombo, Sri Lanka

ABSTRACT/ INTRODUCTON

Pulmonary embolism (PE) is the third most common cardiovascular disease in the world. Trauma is a well-known predisposing factor for PE. Various types of diagnostic methods, scoring systems and specific investigations have been introduced to diagnose PE. With the developed treatment modalities, the life expectancy of patients with PE has been improved. Following case discus, a patient presented with poly trauma who developed pulmonary embolism during ICU stay. Proper identification of the incident and meticulous management lead to a successful recovery.

Keywords: Pulmonary embolism; Cardiovascular disease; Stab injury

CASE HISTORY

A45-year-oldfemale patient suffered stab injury to the face and head and found unconscious. She required intubation and ventilation on admission for reduced conscious level and traumatized upper airway. Non contrast CT brain revealed depressed skull fracture, intracranial haemorrhages and pneumocephalus. She was admitted to intensive care unit for further management. She was managed with multidisciplinary team input including maxillofacial and neurosurgical teams, and underwent surgery under neurosurgical team, for dural repair and wound toilet. DVT stockings and intermittent pneumatic calf compressors were applied. Pharmacological DVT prophylaxis was not started due to traumatic brain injury. Weaning from the ventilator was slow due to a ventilator associated pneumonia, and she was extubated on day five of ICU stay.

On ICU Day 6 she developed acute onset dyspnoea, tachycardia and hypotension, following mobilization. The oxygen saturation was 83% with face mask oxygen. She was tachy cardic and blood pressure was 80/56mmHg. A 12 lead ECG was done and it showed ST depressions in lead I and III. An urgent bed side 2-D Echo showed a filled IVC with minimal variation with respiration. Right atrium and right ventricle were dilated with reduced right ventricular contractility. The left ventricle was hyperkinetic and collapsing. Depending on results of 2D-echo and clinical findings there was a high possibility of pulmonary embolism. A fluid bolus of 250ml was

administered cautiously and inotropes were kept ready. S/C enoxaparin therapeutic dose of 60mg 12 hourly was started with cardiology opinion and with exclusion of neurological contraindications.

Emergency CTPA revealed a saddle thrombus in main pulmonary trunk and there were no pulmonary infarcts. Bilateral lower limb duplex scan was done and DVT of lower limb was excluded. Therapeutic anticoagulation was continued with S/C enoxaparin 60mg 12 hourly.

Warfarin was started with the dose of 5mg daily. After successful recovery patient was discharged from hospital with heamatology follow up.

DISCUSSION

Pulmonary embolism is a consequence of thrombus formation within deep veins of body. It is the third most frequent cardiovascular disease in the world[4] with an annual incidence of 39-115 per 100000 inhabitants [1]. The first presentation may be sudden death. Post traumatic PE is known affect 24% of patients.[4]

The pre-disposing factors can be categorized as medical conditions, drugs, thrombophilia and stasis/endothelial injury[1]. Major trauma, lower limb fracture, joint replacement, spinal injury, previous VTE and metastatic cancer are some of well recognized risk factors. [1,4]

Preferred etiological model for venous thromboembolism is the Virchow's triad, which is vessel wall damage, venous stasis and hypercoagulability[4].Venous thrombi typically develop around intravascular catheters, at site of vascular trauma, and in areas of sluggish blood flow.

Pulmonary embolism results when a thrombus dislodges and trap in pulmonary vasculature. When 30-50% of cross-sectional area of pulmonary circulation is occluded by emboli the pulmonary artery pressure rises, causing increased right ventricular diameter and pressure[1,2].There will be neurohormonal activation causing myocardial inflammation, which in turn increase right ventricular oxygen demand. This will lead to right ventricular ischemia and hypoxic injury. Right ventricular output will reduce causing reduced left ventricular pre-load. As a result, left ventricular filling and cardiac output will reduce causing hypotension. The resulting low coronary perfusion, cause further stress in right ventricle. This spiral of haemodynamic collapse can result in cardiogenic shock and death [1].

The heamodynamic instability associated with PE is categorized as[1]

1. Cardiac arrest
2. Obstructive shock (systolic blood pressure(SBP) less than 90mmHg or vasopressors required to achieve SBP of 90mmHg, and end organ hypoperfusion
3. Persistent hypotension- SBP <90mmHg or SBP drop >40mmHg lasting more than 15 minutes

When pulmonary embolism is suspected it can be diagnosed by clinical presentation, assessment of clinical probability, CTPA, D-dimer, echocardiography, V/Q scan, MRI, Pulmonary angiography and ECG findings [1].

The common clinical characteristics are dyspnoea, pleuritic type chest pain, acute onset cough, tachypnoea, fever, syncope, haemoptysis and signs of DVT [1,3,4]. But these clinical symptoms are non-specific and always need to be combined with other diagnostic tests. Out of these our patient had acute dyspnoea and syncope.

To assess the clinical probability there are several scoring systems. From these Wells[1,3] and Geneva scoring systems are commonly used. [1]

In Wells scoring system seven criteria are considered with one mark for each. Total of >2marks are considered as high risk for PE. Our patient had three out of seven and those were heart rate more than 100/min, surgery or immobilization within past four weeks and alternative diagnosis less likely than PE. Therefore,she was identified as in high-risk category.

The imaging techniques which are used to detect pulmonary embolism are CTPA, V/Q scan, MRI and pulmonary angiography. Bed side echocardiogram plays a major role. CTPA is the investigation of choice for patients with suspected PE. [1]

According to the guidelines patients with shock or hypotension with suspected PE should undergo CTPA. As the facility was readily available our patient was sent for CTPA. It is the best method of diagnosing pulmonary embolism which allows proper and adequatevisualization of the pulmonary arteries down tosegmental level[1]. Pulmonary embolus appears as partial or complete intraluminal filling defect. CTPA is readily available in most centres and may provide alternative diagnosis if PE is excluded[1]. The drawbacks of CTPA are radiation exposure, iodine contrast exposure, and unknown relevance of diagnosing subsegmental PE. [1]

The characteristic echo findings are enlarged and dilated RV, flattened intraventricular septum, distended IVC, collapsed LV, decreased TAPSE and decreased peak systolic velocity of tricuspid annulus. Sometimes a mobile thrombus may be seen in right heart. [1]Other than this 2D echo will also help to rule out other causes of shock as pericardial tamponade, acute valvular dysfunction, LVdysfunction,aortic dissection orhypovolaemia[1] which we didn't see in our patient.

ECG will show RV strain, T inversion in V1-V4,QR pattern in V1, S1Q3T3, incomplete or complete right bundle branch block, sinus tachycardia, atrial arrhythmia and sometimes atrial fibrillation. ECG findings are non-specific.

Compression venous ultrasonography will help to find the origin of the clot[3]. In suspected PE the four-point ultrasonography can be used to detect deep vein thrombosis (groin and popliteal fossa). We performed this and excluded DVT in our patient.

D-dimer test is used in haemodynamically stable patients with intermediate probability of PE. It is highly sensitive >95% and low specific. A value of <500ng/ml exclude PE. Other than in PE this test can be positive in recent surgery, trauma, infection, liver disease, pregnancy and in some cancers. This test didn't have a value in our patient as she had history of trauma.

After confirming the diagnosis of PE, the prognosis can be assessed by PESI score, echocardiography, laboratory tests and biomarkers and combined modalities and scores[1].

PESI score is the Pulmonary Embolism Severity Index which is composed of six criteria[1]. Out of these our patient had tachycardia, systolic blood pressure <100mmHg and arterial oxygen saturation of <90%. As patients who gained >1mark are classified under high risk, our patient felt into high-risk category, with risk of mortality of 10.9% within 30 days.

When considering treatment of PE, the options are haemodynamic and respiratory support, thrombolytic treatment, anticoagulation, surgical embolectomy, percutaneous catheter-directed treatment and IVC filters. [1]ECMO may be considered in patients with refractory cardiac collapse.

When managing PE, patients who are haemodynamically unstable should be aggressively treated.

For respiratory support supplemental high-concentration oxygen should be administered with a target of >94%. In hypoxaemia or respiratory failure intubation and mechanical ventilation may be necessary.

When,SBP<90 mmHg initial fluid bolus of 500ml can be tried[1]but aggressive fluid resuscitation will give no benefit and may even worsen the right ventricular function.

Vasopressors may be given parallel with surgical and pharmacological interventions. Norepinephrine and dobutamine described. [1]

Primary reperfusion is recommended in patients with shock or hypotension to provide clot dissolution and to improve right ventricular function, pulmonary blood flow and lung perfusion. The pharmacological options are, specific recombinant tissue type plasminogen activator (rtPA), Streptokinase. and urokinase.[1] Clinician should be vigilant to exclude contraindications for fibrinolysis. Primary reperfusion was not done in our patientas it was contraindicated with recent history of traumatic brain injury. She received therapeutic anticoagulation.

Therapeutic anticoagulation for at least three months should be given for all patients with PE. [1,3] If patients present with recurrent VTE, oral anticoagulation treatment should be continued for indefinite duration. [1] Carefully selected patients with low-risk PE, can be considered for early discharge and outpatient care. [1]

CONCLUSION

Pulmonary embolism is the third most common cardiovascular disease in the world. The diagnostic and therapeutic methods are developing over the time. Clinical suspicion, quick identification and management according to guidelines are important steps in management. The timely identification and meticulous management of the condition made a successful recovery in our patient.

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