Massive Pulmonary Embolism with Haemodynamic Collapse

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ABSTRACT
Massive pulmonary embolism with shock remains a highly fatal disease. We present twelve cases of massive embolism over the last seven years that required emergent surgery. Five patients suffered haemodynamic collapse and all died despite heroic attempts at salvage. A better outcome can only be achieved in this sub-category of patients with a rapid confirmatory diagnosis and appropriate thrombolysis and early referral to a cardiothoracic surgeon.

Keywords: Massive, pulmonary embolism, emergent surgery, haemodynamic collapse

INTRODUCTION
The severity of pulmonary embolism (PE) depends on three factors, namely, the degree of clot burden, the rapidity of clot accumulation and existing cardiopulmonary reserve. The latter is dependent on the presence of congestive cardiac failure and/or chronic pulmonary disease. Any haemodynamic consequence is reflective of the severity of “throughput” failure and backward right heart strain. In the Urokinase Pulmonary Embolism Trial, massive PE(1) was defined anatomically as equivalent obstruction of two or more lobar pulmonary arteries (about 50% of the pulmonary arterial tree). This is seen in uncompromised patients. However, the percentage of pulmonary occlusion required for haemodynamic instability and right ventricular (RV) dysfunction can vary depending on pre-existing cardiopulmonary disease. Thus, clinically massive PE is better defined by haemodynamic instability and right ventricular (RV) dysfunction rather than by a percentage of pulmonary arterial occlusion. It is a clinical syndrome of high mortality characterised by acute pulmonary arterial occlusion with resultant sudden elevation in pulmonary arterial pressures and RV failure. It results in significant hypoxemia, unstable haemodynamics and tissue hypoperfusion. The principles of treatment include reducing the clot burden by thrombolysis or mechanical removal and prevention of further clot production (deep venous thrombosis (DVT)) by anticoagulation (heparin and warfarin) and clot migration by caval filter insertion(2). We report twelve cases of massive pulmonary embolism that presented over the last seven years at the Singapore National Heart Centre that required emergent surgical embolectomy.

METHODS AND PATIENTS
A retrospective review of all surgical cases of emergent pulmonary embolectomy at The National Heart Centre from 1993 to 1999 was carried out. A summary of the findings is given in Table I.

There were 12 patients of whom five were males and seven were females. The youngest was 34 years old and the oldest 76 years old. Predisposing factors for deep venous thrombosis were present in all cases. These were: recent surgery - three cases (one subtotal colectomy, one spinal laminectomy and one liver resection for hepatocellular carcinoma); immobilisation - four cases (one fracture neck of femur, one recent cerebrovascular accident (CVA), one road traffic accident and two multiple medical problems including obesity and ischaemic heart disease); postpartum state - one case; pelvic pathology - two cases (one ulcerative colitis, one large uterine fibroid); and recurrent DVT - one case. Only five of the 12 cases had clinically evident DVT of the lower limb. Six patients had DVT on duplex examination. One patient (Case 12) had no evidence of DVT clinically or on duplex. There were three types of haemodynamic presentation: cardiovascular collapse requiring CPR (five cases), stable haemodynamics (three cases) and hypotension (four cases). Of those with collapse, two had an earlier episode of haemodynamic compromise that was successfully resuscitated. The confirmatory investigative tool was either spiral computed tomography (five cases) or pulmonary angiography (four cases). Three cases were managed based on presumptive diagnosis because of haemodynamic collapse and were rushed to the operating room. The outcome was heavily dependent on their haemodynamic state. All five (Cases 3, 5, 7, 9 and 12) that collapsed, died; four died on the operating table (Cases 3, 5, 7 and 9) and one died (Case 12) from massive cerebral infarction and eventual pulmonary
sepsis. Of the four that died on the table, two were from massive bleeding secondary to liver lacerations incurred during chest compressions. The period of CPR was prolonged in these two patients. The other two could not be weaned off cardiopulmonary bypass (CPB). All four with hypotension survived with surgery. All three with stable haemodynamics survived the embolism but one died later from mediastinitis. This was the patient with hepatocellular carcinoma and cachexia. The three stable patients were not candidates for thrombolytics because of recent surgery, recent CVA or bleeding ulcerative colitis.

**SURGICAL TECHNIQUE**

The principles include rapid institution of normothermic CPB to overcome “throughput” failure, stabilising the haemodynamic state. This is accomplished via rapid median sternotomy and crash bicaval CPB. Alternatively, when the patient is undergoing chest compressions, rapid femoral-femoral bypass is essential to avoid disrupting the resuscitative process. With the chest opened, CPB instituted and without aortic cross clamping, a longitudinal pulmonary arteriotomy is performed distal to the pulmonary valve. Gentle and thorough evacuation of clots is performed using stone forceps. Distal clot extraction may require opening both pleurae and massaging the lungs with the aid of Fogarty balloon catheters done carefully to avoid perforating the pulmonary vasculature. After de-airing, the pulmonary artery is closed in two layers using 4/0 prolene sutures. After a short period of further reperfusion, the patient is weaned off CPB usually with the aid of inotropes for right ventricular dysfunction.

**DISCUSSION**

It is known that lower limb DVT though responsible for over 90% of pulmonary emboli, is clinically obvious in only 10% of cases. PE occurs in a setting predisposed to deep venous thrombosis. Virchow’s triad of stasis, hypercoagulability and endothelial damage is well known. The patients presented here all had risk factors for DVT and PE. These include cancer (Cases 8 and 10), congestive cardiac failure (Case 10), chronic obstructive pulmonary disease (COPD), recent abdominal, pelvic or hip surgery (Cases 1, 6, 7 and 10), immobility (Cases 2, 3, 9 and 12) and comorbid conditions like diabetes mellitus and obesity (Case 3). An interesting presentation was the patient who presented initially with a pulseless left upper limb seven days after spinal laminectomy. A brachial embolus was removed but no further investigations were done till an episode of hypotension and dyspnea. A pulmonary angiogram revealed paradoxical embolism through a patent foramen ovale. Once a diagnosis of PE is suspected in a patient with hypotension (subnormal blood pressure), a rapid diagnosis by CT or pulmonary angiogram is essential, especially those with poor cardiopulmonary reserves i.e. older patients, congestive cardiac failure and COPD. Moreover, there will be a category of patients who present with either a severe hypotensive episode or haemodynamic collapse and are successfully resuscitated.

Table 1. Characteristics and outcome of patients undergoing surgical embolectomy.

<table>
<thead>
<tr>
<th>No</th>
<th>Age &amp; Sex</th>
<th>Risk Factor</th>
<th>Hemodynamics</th>
<th>Outcome</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73 F</td>
<td>Fracture neck femur, DVT on duplex</td>
<td>Hypotension 100/60 mmHg</td>
<td>Alive</td>
<td>IVC filter</td>
</tr>
<tr>
<td>2</td>
<td>71 M</td>
<td>Recurrent CVA, calf DVT evident</td>
<td>Stable</td>
<td>Alive</td>
<td>IVC filter, TL contraindicated</td>
</tr>
<tr>
<td>3</td>
<td>50 F</td>
<td>Obesity, IHD, D/M, immobile, DVT on duplex</td>
<td>1st collapse resuscitated 2nd collapse during TL</td>
<td>Dead</td>
<td>Failure to wean, DIVC</td>
</tr>
<tr>
<td>4</td>
<td>48 M</td>
<td>Recurrent calf DVT, not on warfarin</td>
<td>Stable</td>
<td>Alive</td>
<td>IVC filter</td>
</tr>
<tr>
<td>5</td>
<td>26 F</td>
<td>One month postpartum, DVT on duplex</td>
<td>Collapse in another hospital Rushed to OR</td>
<td>Dead</td>
<td>1.5 hour CPR, P-CPB, liver tear</td>
</tr>
<tr>
<td>6</td>
<td>64 M</td>
<td>Day seven spinal laminectomy, brachial embolus removed earlier</td>
<td>Hypotension</td>
<td>Alive</td>
<td>IVC filter, TL contraindicated</td>
</tr>
<tr>
<td>7</td>
<td>34 F</td>
<td>Large uterine fibroids, calf DVT evident</td>
<td>Collapse in another hospital</td>
<td>Dead</td>
<td>P-CPB, liver tear</td>
</tr>
<tr>
<td>8</td>
<td>76 M</td>
<td>Hepatocellular cancer, liver surgery, DVT on duplex</td>
<td>Stable</td>
<td>Dead</td>
<td>Mediastinitis, DIVC</td>
</tr>
<tr>
<td>9</td>
<td>35 F</td>
<td>Right leg DVT after RTA</td>
<td>Collapse</td>
<td>Dead</td>
<td>Failure to wean from CPB, DIVC</td>
</tr>
<tr>
<td>10</td>
<td>64 F</td>
<td>Day four subtotal colectomy, calf DVT evident</td>
<td>Hypotension, two episodes; 2nd episode while awaiting TL</td>
<td>Alive</td>
<td>IVC filter</td>
</tr>
<tr>
<td>11</td>
<td>38 F</td>
<td>Bleeding ulcerative colitis, DVT on duplex</td>
<td>Hypotension 90/60 mmHg</td>
<td>Alive</td>
<td>IVC filter, TL contraindicated</td>
</tr>
<tr>
<td>12</td>
<td>66 M</td>
<td>Hypertension, IHD, D/M, immobility; duplex negative</td>
<td>Earlier hypotension then collapse whilst awaiting surgery</td>
<td>Dead</td>
<td>Massive cerebral infarction</td>
</tr>
</tbody>
</table>

M = male, F = female, DVT = deep venous thrombosis, CVA = cerebrovascular infarction, IHD = ischaemic heart disease, D/M = diabetes mellitus, TL = thrombolytics, DIVC = disseminated intravascular coagulopathy, P-CPB = percutaneous cardiopulmonary bypass, OR = operating room, CPR = cardiopulmonary resuscitation, IVC = inferior vena cava, RTA = road traffic accident.
This group should undergo expedient confirmatory investigations and then surgery or thrombolysis as they may not survive a second collapse due to an already compromised cardiopulmonary reserve. The next embolus is usually fatal as seen in Cases 3 and 12. Thrombolysis has also been shown to be life-saving in the group of patients with overt haemodynamic instability and even shock\(^4\). But once haemodynamic collapse occurs, open surgery is usually the only method, rapid enough to have any possible measure of success by regaining throughput and avoiding massive cerebral infarction. Even then with backward failure and resultant liver congestion, shock liver and CPR inflicted liver lacerations may result, contributing to rapid exsanguination and the demise of the patient (Cases 5 and 7). This study is too small to analyse which features are bad prognostic indicators. However, it is well

known from larger studies that with haemodynamic collapse before surgery, the mortality is high (more than 50\%)\(^5-7\). In our experience all five cases that suffered haemodynamic collapse died. In the current era of thrombolysis, appropriate indications for surgical embolectomy would include cases where thrombolysis is inappropriate or ineffective\(^8\). Saveliev\(^9\) attempted to determine factors that would select embolectomy over thrombolysis in massive embolism and determined that a high Miller’s index\(^10\) of 27 or more and significant embolism, as salvage may be possible with rapid surgical evacuation. Even then, cerebral infarction and liver laceration are notable events that follow. Early referral to a cardiothoracic surgeon should thus be considered in cases of significant embolism, as salvage may be possible should haemodynamic deterioration occur.

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REFERENCES