Massive Pulmonary Embolism with Haemodynamic Collapse

M G Caleb

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

Singapore Med J 2002 Vol 43(1):025-027

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 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

National Heart Centre
17, Third Hospital Avenue
Singapore 168752

M G Caleb, MBBS, FRCS Edin, MMed (Surg)
Consultant

Correspondence to:
M G Caleb
Tel: (65) 436 7598
Fax: (65) 224 3632
Email: mcgeorge88@hotmail.com

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

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

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.

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 normo-thermic 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[3]. 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.

This group should undergo expedient confirmatory investigations and then surgery or thromobolytics 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]. Savelyev[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 haemodynamic disturbances including refractory systemic hypotension, severe pulmonary hypertension or mean right ventricular pressure greater than 25 mmHg were important. For our post-operation survivors, we inserted a vena caval filter (Simon nitinol; Bard Peripheral Technologies, Covington, Georgia, USA) transvenously on Day 2 after surgery, although the clinical validity of caval filter insertion in this post-operation setting is controversial. There is no definite evidence of efficacy in this setting. Caval filters are known to increase the risk of late recurrent deep vein thrombosis when used alone to prevent pulmonary embolism in proximal deep vein thrombosis as seen in the only randomised trial of caval filters[11] and our post-operative patients may be exposed to similar risks. Nevertheless, caval filters have been recommended in patients who have contraindications to anticoagulation or complications from anticoagulation. It is also recommended in patients who have PE that are treated medically[12]. All survivors in our group receive warfarin routinely for at least six months unless there are contraindications to anticoagulation (Case 11). Another issue of concern that can be seen from this small study is how soon should a referral be made to a cardiothoracic surgeon. Cases of pulmonary embolism requiring only heparin and warfarin can be treated in hospitals that do not have cardiothoracic surgeons[13] (Class I & II). Ninety percent of such cases are successfully treated by anticoagulation alone. Once a diagnosis of submassive or massive embolism (Class III & IV) is made, they should be transferred to a centre that has facilities for both thrombolysis and open embolectomy. This should also include cases that are not suitable for or who fail to respond to anticoagulation and hence require thrombolysis, caval filter insertion or surgery. Some of these patients may become unstable during investigations or treatment as seen in Cases 3, 10 and 12.

CONCLUSIONS

Massive pulmonary embolism remains a highly fatal disease. An episode of severe hypotension in a patient with deep vein thrombosis and suspected pulmonary embolism requires a rapid diagnosis to enable expedient thrombolysis or surgery. However, when haemodynamic collapse occurs, CPR is often futile and salvage may only 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.

ACKNOWLEDGEMENTS

The author acknowledges the contributions of the surgeons of the National Heart Centre, whose cases have been used in this study.

REFERENCES

1. The urokinase pulmonary embolism trial. JAMA 1970; 214:2163-72.
2. Goldhaber SZ. Pulmonary embolism. Review N Engl J Med 1998; 339:93-104.
3. Stein PD, Willis PW III, DeMets DL. History and physical examination in acute pulmonary embolism in patients without pre-existing cardiac or pulmonary disease. Am J Cardiol 1981; 47:218-23.
4. Jerjes-Sanchez C, Ramirez Rivera, Garcia ML, et al. Streptokinase and heparin versus heparin alone in massive pulmonary embolism: a randomised controlled trial. J Thromb Thrombolysis 1995; 2:227-9.
5. Hyers TM, Hull RD, Weg JG. Anti-thrombotic therapy for venous thromboembolic disease. Chest 1995; 108; 335S-351S.
6. Clarke DB, Abrams LD. Pulmonary embolectomy: a 25-year experience. J Thorac Cardiovasc Surg 1986; 92:442-5.
7. Gray HH, Morgan JM, Paneth M, Miller GA. Pulmonary embolectomy for acute massive pulmonary embolism; an analysis of 71 cases. Br Heart J 1988; 60:196-200.
8. Doerge HC, Schoendube FA, Loeser H, et al. Pulmonary embolectomy; a review of a 15 year experience and role in the age thrombolytic therapy. Eur J Cardiothorac Surg 1996; 10:952-7.
9. Savelyev VS. Massive pulmonary embolism: embolectomy or thrombolysis? Int Angiol 1985; 4:137-40.
10. Miller GA, Sutton GC, Kerr IH, Gibson RV, Honey M. Comparison of streptokinase and heparin in treatment of isolated acute massive pulmonary embolism. BMJ 1971; 2:681-7.
11. Decousus H, Leizorovicz A, Parent F, Page Y, et al. A clinical trial of caval filters in the prevention of pulmonary embolism in patients with proximal deep vein thrombosis. N Engl J Med 1998; 338:409-15.
12. Sixth ACCP consensus conference on anti-thrombotic therapy. Chest 2001; 119/1(suppl).
13. Glenn's Thoracic and cardiovascular surgery. 6th edition, 1996. Vol 2. Chapter 110.