The Internet Journal of Thoracic and Cardiovascular Surgery
Volume 10 Number 1

Lessons from Surgical Embolectomy for Saddle Pulmonary Embolism: A Case Report and Literature Review

E Williams, S Cawich, R Irvine, S Shah, J Williams-Johnson, G Blake, P Singh, N Hall, K Espinosa, S French

Citation

E Williams, S Cawich, R Irvine, S Shah, J Williams-Johnson, G Blake, P Singh, N Hall, K Espinosa, S French. *Lessons from Surgical Embolectomy for Saddle Pulmonary Embolism: A Case Report and Literature Review*. The Internet Journal of Thoracic and Cardiovascular Surgery. 2006 Volume 10 Number 1.

Abstract

Saddle emboli are one of the most severe forms of Pulmonary Embolism and are associated with high mortality rates. Patient salvage depends upon making an early diagnosis, although there is still much controversy regarding the ideal therapeutic strategy for confirmed cases. We present a case where embolectomy was performed to extract a saddle embolus and explore the available management options.

INTRODUCTION

Large saddle emboli are common findings at necropsy. They are regarded as one of the most severe forms of Pulmonary Embolism (PE). This diagnosis carries a high mortality rate because the patients are usually clinically unstable and are prone to sudden circulatory arrests.

Patient salvage hinges upon making an early diagnosis. But the ideal therapeutic strategy for confirmed cases remains controversial because there is inadequate data presently to develop evidence based management guidelines ([1,2]). Thrombolytic agents may be utilized, but are limited by a high incidence of intracranial haemorrhage ([2,3]). Surgical embolectomy is a more invasive alternative in already unstable patients, but it is considered an option in selected cases ([2,3,4]). We present a case where embolectomy was performed to extract a saddle embolus and explore the available management options.

CASE REPORT

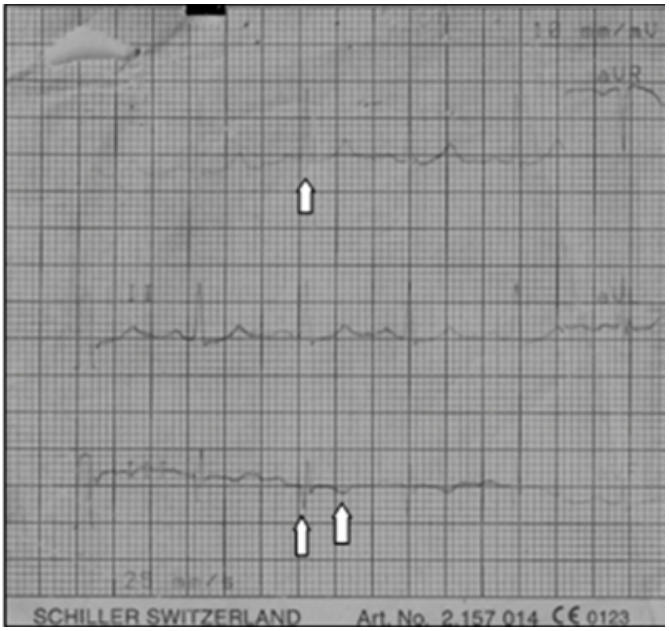
A 37 year old man presented to the emergency room with right flank pain for two days duration. No other symptoms were present. He had an unremarkable medical history, was not a smoker and did not take any regular medications.

Initial vital signs were normal with a blood pressure of 114/73mmHg and heart rate of 92/minute. Chest and nervous system examinations were normal, but there was tenderness without peritonitis at the right flank and costo-vertebral angle. Supported by a dipstick urinalysis detecting haematuria3+, a diagnosis of right ureteric colic was made.

One hour after presentation, the patient experienced dizziness and exacerbated pain. He became hypotensive with blood pressures of 86/56mmHg and heart rates of 114/minute. A 12 lead electrocardiogram (ECG) revealed sinus tachycardia and a "$S_1Q_3T_3$" pattern suggesting right heart strain (Fig. 1). A qualitative Troponin-I rapid-assay kit® (Spectral Cardiac STATus Troponin I Rapid Test, Spectral Diagnostics) was positive, suggesting myocardial injury. An arterial blood gas analysis on 10L oxygen revealed pH 7.38, PCO_2 31.5mm/Hg and PO_2 63.2mm/Hg. The measurements of serum electrolytes, glucose levels, haematologic and clotting indices were all within normal reference ranges.

Figure 1

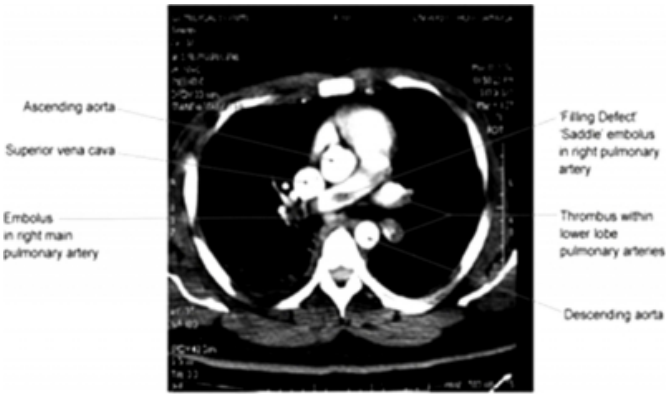
Figure 1: recorded Tracing of the 12 lead electrocardiogram demonstrating the characteristic “SQT” pattern suggestive of right ventricular strain.



There was haemodynamic improvement after a bolus infusion of 1L intravenous crystalloids, with blood pressure rising to 119/75mmHg. To narrow differential diagnoses of a myocardial infarction, pulmonary embolism or aortic dissection, a contrast-enhanced spiral computed tomographic scan (CT) of the chest with 5x8mm slices was completed. It revealed a saddle embolus sitting within the pulmonary trunk extending into both pulmonary arteries, right ventricular dilatation and a small right pleural effusion (Fig. 2).

Figure 2

Figure 2: Contrast-enhanced spiral CT of the chest demonstrating a massive saddle PE. The embolus appears as a filling defect in the pulmonary trunk and extends into the right pulmonary artery.



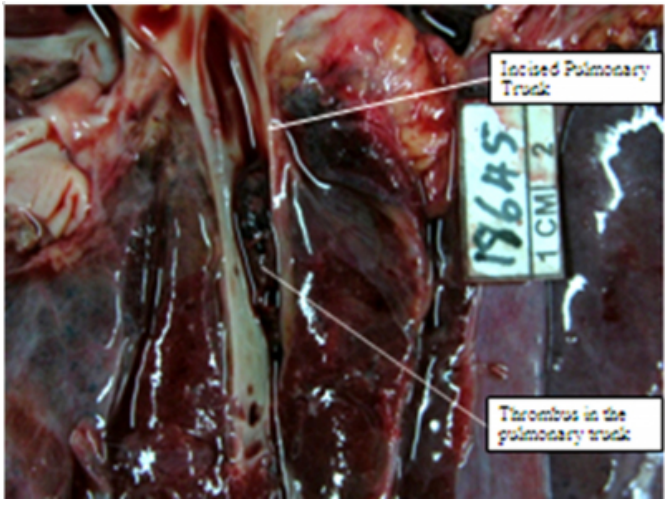
Despite aggressive resuscitation, episodic haemodynamic instability continued. This prompted our decision to proceed to operative embolectomy. Across a median sternotomy incision, cardiac bypass was established and the pulmonary trunk incised. This revealed an extensive embolus approximately 16cm in length that extended across the pulmonary trunk into both pulmonary arteries. The right atrium and ventricle were markedly distended and hypokinetic. The saddle embolus was removed and the arteriotomy closed with 5/0 polypropylene sutures. Un-fractionated intravenous heparin was commenced immediately post-operatively and the patient was admitted to the ICU.

Over the subsequent 48 hours, he became haemodynamically unstable with a persistent severe metabolic acidosis. Trans-oseophageal echocardiography (TEE) performed in the ICU revealed right ventricular hypokinesia, but no evidence of recurrent emboli. His condition deteriorated despite large doses of inotropes to maintain mean arterial pressure at 60mmHg. He soon experienced a cardiac arrest from which he could not be resuscitated, and was pronounced dead two days after operation.

A postmortem examination revealed an intact pulmonary arterial anastomosis and a recurrent ante-mortem saddle embolus was found within the pulmonary artery (Fig. 3). There were no demonstrable risk factors identified to explain this patient's propensity for saddle emboli, despite embolectomy and adequate anticoagulation.

Figure 3

Figure 3: Post-mortem photograph demonstrating the pulmonary trunk that has been incised longitudinally to demonstrate an embolus (arrows) within the artery.



DISCUSSION

The management of a massive PE requires a multi-disciplinary approach that involves radiology, medical and surgical specialties. Establishing an early diagnosis shortly after presentation is paramount to ensure patient salvage.

Pulmonary angiography is the gold standard investigation for establishing the diagnosis, but it is is invasive and complicated ([5]). Pulmonary scinitgraphy has been utilized, but lacks specificity ([5]). Furthermore, both investigations are usually unavailable under emergency conditions. Newer imaging techniques such as TEE and contrast-enhanced CT of the chest are being used increasingly with good success ([5,6]).

A contrast-enhanced spiral CT of the chest can immediately confirm the presence of a proximal PE and exclude an aortic dissection. This is important since inadvertent administration of thrombolytics or anticoagulants in patients with aortic dissection could have lethal consequences. In stable patients, CT of the chest is more sensitive than TEE because it can reliably assess distal branches of the pulmonary artery down to the level of segmental vessels ([7,8]). The diagnostic accuracy of CT is directly related to the severity of the patients' clinical presentations ([9,10]). In suspected massive PE, spiral CT is reported to have 100% sensitivity and specificity ([9]). But in patients with intermediate-probability lung scintigraphs, the diagnostic prowess falls to 63% sensitivity and 89% specificity ([10]).

On the other hand, TEE is inexpensive and rapidly performed at the bedside, but it is heavily user-dependent ([5,6]). TEE can detect emboli in the main pulmonary arteries in approximately 70% of patients with haemodynamically significant PE, but is ineffective in detecting emboli within peripheral vessels ([5,6]). Unlike CT, TEE may provide additional information regarding homogeneity and mobility of emboli, their relation to the vascular lumen in cross-sectional planes and resultant flow disturbances ([5,6]). This information may be useful to evaluate the age of the embolus and its susceptibility to thrombolysis.

The preferred imaging modality is still unsettled, but it is clear that both TEE and CT are alternatives in the diagnosis of massive PE. Contrast-enhanced spiral CT was the chosen in this case by default because TEE is not readily available at our institution. The diagnosis was strengthened by classic ABG results and the "$S_1Q_3T_3$" pattern with sinus tachycardia present on the ECG tracing. Although non-specific ST changes and tachycardia are the most common findings in PE, the "$S_1Q_3T_3$" pattern is pathognomonic. The Troponin-I levels were likely elevated due to myocardial injury because of high right ventricular pressures from volume overload.

Patients with saddle emboli are in a precarious condition because at any point there may be worsening of pulmonary vascular obstruction leading to sudden circulatory arrest. Patient salvage is heavily dependent upon emergent correction of these haemodynamic disturbances. Arterial hypotension and right ventricular dilatation are recognized to be poor prognosticators associated with increased mortality and recurrence ([1,3,4]). Their presence prompted our decision to proceed with embolectomy in this case.

The ideal therapeutic strategy remains unsettled because there is currently insufficient data available to make evidence-based management guidelines. Emergent surgical embolectomy has been touted as the better therapeutic option because it can rapidly remove the embolus ([3,11,12,13,14,15]). Several small retrospective series have examined embolectomy for massive PE and report that operative mortality ranges from 8% ([11]) to 46% ([12]). A collective review of 200 patients across six series revealed that 72% of patients survived embolectomy and were discharge from hospital alive ([3,11,12,13,14,15]).

Mature surgical judgment is needed for appropriate patient selection because these patients usually have significant co-morbidities that render them poor risk candidates for anaesthesia. It has been suggested that patients over the age of 60 with pre-surgical cardiac arrests, concomitant cardio-pulmonary disease, recurrent PE or immobility have worse outcomes ([3,12,13]). Based on the absence of any of these poor prognosticators, we believe that our patient was a good surgical candidate. His demise was likely due to the recurrent saddle PE despite adequate anticoagulation.

Thrombolysis promises embolus dissolution without the associated operative and anaesthetic risks. It also dissolves smaller emboli within the distal vasculature that the surgeon cannot access. But these benefits come at the cost of increased intra-cranial and gastro-intestinal bleeding ([16,17,18]). Catheter directed thrombolysis also requires skilled interventional radiologists and specialized equipment that may not be universally available, especially in developing countries.

There are large series evaluating thrombolysis in patients

with massive PE. A total of 2,454 consecutive patients were evaluated in the International Cooperative PE Registry ([16]). These patients had 17.4% mortality 90 days after thrombolysis ([16]), which is lower than the mortality after embolectomy (28%) in our collective review ([3,11,12,13,14,15]). But, there was a high rate of intra-cranial bleeding (3%) when thrombolysis was employed for PE ([16]). Similar results were reported from smaller PE registries ([17,18]).

After dissolution or removal of the emboli and adequate anticoagulation, 15.1% of patients will develop a symptomatic recurrence of major PE ([19]). This occurred in our patient who succumbed to an undiagnosed recurrence. A TEE was performed at the bedside but it could not detect the recurrence, highlighting its user-dependent nature. Unfortunately the cardiovascular instability did not allow safe transport to the radiology department for CT, angiography or scintigraphy that may have made the diagnosis.

It has been suggested that the placement of a caval filter may prevent a recurrent PE. Filters are not commonly employed because they have not been shown to be better than adequate anticoagulation to prevent emboli in patients with free-floating deep venous thrombi ([20]). They also do not address the existing pulmonary embolus and therefore cannot improve the clinical scenario ([20,21,22]). Although two prospective trials have suggested that symptomatic recurrences in medically treated patients are reduced ([19,21]) the most recent Cochrane database review has suggested that any perceived benefit of filters is counterbalanced by increased recurrent deep vein thrombosis and chronic venous insufficiency, without any demonstrable survival advantage ([22]).

Although their place in the treatment of massive PE remains uncertain ([22]), some authorities have purported filter placement at the time of surgical embolectomy ([15,23,24,25]). Currently the evidence base for this recommendation is weak, stemming mostly from case reports ([24,25]) and small series with low recurrent PE rates ([15,23]). We cannot deny that a filter may have been life saving in this patient who succumbed to the recurrent PE. Based on this experience we shall seriously consider same-sitting filter placement when patients at our institution are selected for embolectomy in the future. Until better trials are designed, we hope that this case will contribute to the literature in support of caval filters.

CORRESPONDENCE TO

Dr. Shamir Cawich Department of Surgery, Radiology, Anaesthesia and Intensive Care. The University of the West Indies, Mona, Kingston 7, Jamaica, WI. E-mail: socawich@hotmail.com

References

1. Goldhaber S. Thrombolysis in pulmonary embolism. A large-scale clinical trial is overdue. Circulation 2001;104:2876-2878.
2. Gulba D, Schmid C, Hans-Georg Borst, Lichtien P, Dietz R, Luft F. Medical compared with surgical teatment for massive pulmonay embolism. The Lancet March 1994; V343:576-8.
3. Aklog L, Williams C, Byrne G, Goldhaber S. Acute pulmonary embolectomy- a contemporary approach. Circulation 2002;105:1416-1419
4. Georghiou G, Brauner, Brauner R ,Berman M, Stamler A, Glanz L, Bernardo V, Erez E. Sucessful resuscitation of a patient with acute massive pulmonary embolism using emergent embolectomy. Ann Thorac Surg 2004;77:697-97
5. Torbicki A, Pacho R, Jedrusik P, Pruszczyk P. Noninvasive diagnosis and treatment of a saddle pulmonary embolism. Chest 1996; 109:1124-6.
6. Pruszczyk P,Torbicki A, Pacho R, Chlebus M, Kuch-Wocial A, Pruszynski B, Gurba H. Noninvasive diagnosis of suspected severe pulmonary embolism. Transesophegeal echocardiography vs spiral CT. Chest 1997; 112: 722-28.
7. Remy-Jardin M, Remy J, Wattinne L, Giraud F. Central pulmonary thromboembolism: diagnosis with spiral volumetric CT with the single-breathe-hold technique and a comparison with pulmonary angiography. Radiology 1992; 185(2): 381-87.
8. Teigen CL, Maus TP, Sheedy PF, Stanson AW, Johnson CM, Breen JF, et al. Pulmonary embolism: diagnosis with contrast enhanced electron beam CT and comparison with pulmonary angiography. Radiology 1995; 194(2): 313-19.
9. Blum AG, Delfau F, Grignon B, Beurrier D, Chabot F, Claudon M, et al. Spiral-computed tomography versus pulmonary angiography in the diagnosis of acute massive pulmonary embolism. Am J Cardio 1994; 74(1): 96-98.
10. Goodman LR, Curtin JJ, Mewissen MW. Detection of pulmonary embolism in patients with unresolved clinical and scinigraphic diagnosis: helical CT versus angiography. Am J Roentgenol 1995; 164:1369-74.
11. Sa YJ, Choi SY, Lee JH, Kwon JB, Moon SW, Jo KH, Wang YP, et al. Off-pump open pulmonary embolectomy for patients with major pulmonary embolism. Heart Surg Forum. 2007;10(4):E304-8.
12. Stulz P, Schlapfer R, Feer R, Habicht J, Gradel E. Decision making in the surgical treatment of massive pulmonary embolism. Eur J Cardiothorac Surg. 1994;8(4):188-93.
13. Ullmann M, Hemmer W, Hannekum A. The urgent pulmonary embolectomy: mechanical resuscitation in the operating theatre determines the outcome. Thorac Cardiovasc Surg. 1999;47(1):5-8.
14. Jakob H, Vahl C, Lange R, Micek M, Tanzeem A, Hagl S. Modified surgical concept for fulminant pulmonary embolism. Eur J Cardiothorac Surg. 1995;9(10):557-561.
15. Bauer EP, Laske A, Von Segesser LK, Carrel T, Turina MI. Early and late results after surgery for massive pulmonary embolism. Thorac Cardiovasc Surg. 1991;39(6):353-6.

16. Goldhaber SZ, Visani L, De Rossa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet. 1999;353:1386-1389.
17. Meyer G, gisselbrecht M, Diehl JL, et al. I ncicence and predictors of major haemmorrage complication from thrombolytic therapy in patients with massive pulmonary embolism. Am J Med. 1998; 105: 472-477.
18. Hamel E, Pacouret G, Vincentelli D, et al. Thrombolysis or heparin therapy in massive pulmonary embolism with right ventricular dilation: results from a 128- patient monocentre registry. Chest. 2001;120:120-125.
19. PREPIC Study Group. Eight-year follow-up of patients with permanent vena cava filters in the prevention of pulmonary embolism: the PREPIC randomized study. Circulation. 2005; 112(3):416-22.
20. Pacouret G, Alison D, Pottier JM, Bertrand P, Carbonnier B. Free-floating thrombus and embolic risk in patients with angiographically confirmed proximal deep venous thrombosis. A prospective study. Arch Intern Med. 1997;157(3):305-8.
21. Decousus H, Leeizorovicz A, Parent F, Page Y, Tardy B, Girad P, et al. A clinical trial of vena caval filters in the prevention of pulmonary embolism in patients with proximal deep-vein thrombosis. N Eng J Med. 1998;338(7):409-15
22. Young T, Aukes J, Huges R, Tang H. Vena caval filters for the prevention of pulmonary embolism. Cochrane Datase Syst Rev. 2007 Jul 18;(3):CD006212
23. Vos LD, Tielbeek AV, Bom EP, Gooszen HC, Vroegindeweij D. The Günther temporary inferior vena cava filter for short-term protection against pulmonary embolism. Çardiovasc intervent Radiol. 1997;20(2):91-7.
24. Rosenberger P, Shernan SK, Rawn JD, Scott J, Eltzschig HK. Critical role of inferior vena caval filter placement after pulmonary embolectomy. J Card Surg. 2005;20(3):289-90.
25. Moon SW, Jo KH, Wang YP, Kim YK, Kwon OK. Off-pump open pulmonary embolectomy for major bilateral pulmonary emboli: report of a case. Surg Today. 2006;36(3):274-6.

Author Information

Eric W. Williams
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Shamir O. Cawich
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Roger Irvine
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Sundeep Shah
Department Radiology, University of the West Indies

Jean Williams-Johnson
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Garfield Blake
Department Pathology, University of the West Indies

Praim Singh
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Nadia Hall
Department Pathology, University of the West Indies

Kurdell Espinosa
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies

Simone French
Department of Surgery, Radiology, Anaesthesia and Intensive Care, University of the West Indies