Modern surgical treatment of massive pulmonary embolism: Results in 47 consecutive patients after rapid diagnosis and aggressive surgical approach

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Objectives: This study retrospectively reviews an aggressive multidisciplinary approach to the treatment of massive pulmonary embolism, centering on rapid diagnosis with contrast-enhanced computed tomography of the chest to define the location and degree of clot burden and transthoracic echocardiography to document right ventricular strain followed by immediate surgical intervention when appropriate.

Methods: Between October 1999 through February 2004, 47 patients (30 men and 17 women; median age, 58 years; age range, 24-86 years) underwent emergency surgical embolectomy for massive central pulmonary embolism. The indications for surgical intervention were (1) contraindications to thrombolysis (21/47 [45%]), (2) failed medical treatment (5/47 [10%]), and (3) right ventricular dysfunction (15/47 [32%]). Preoperatively, 12 (26%) of 47 patients were in cardiogenic shock, and 6 (11%) of 47 were in cardiac arrest.

Results: There were 3 (6%) operative deaths, 2 with preoperative cardiac arrest; 2 of these 3 patients required a right ventricular assist device. In 38 (81%) patients a caval filter was placed intraoperatively. Median length of stay was 11 days (range, 3-75 days). Median follow-up was 27 months (range, 2-50 months); follow-up was 100% complete in surviving patients. There were 6 (12%) late deaths, 5 of which were from metastatic cancer. Actuarial survival at 1 and 3 years' follow-up was 86% and 83%, respectively.

Conclusion: An aggressive approach to large pulmonary embolus, including rapid diagnosis and prompt surgical intervention, has improved results with surgical embolectomy. We now perform surgical pulmonary embolectomy not only in patients with large central clot burden and hemodynamic compromise but also in hemodynamically stable patients with right ventricular dysfunction documented by means of echocardiography.

Despite advances in diagnosis and therapy, acute pulmonary embolism (PE) is still associated with a high mortality rate. According to data published in the International Cooperative Pulmonary Embolism Registry, 2454 patients with acute PE from 52 hospitals in 7 countries died within 90 days.¹ Most deaths

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TABLE 1. Risk factors for development of pulmonary embolism (n = 47)

Condition	N (%)
Immobility	17 (36%)
DVT	13 (27%)
Cancer	11 (23%)
Prior pulmonary embolism	1 (2%)
Hypercoagulability	1 (2%)
Vascular anomaly	1 (2%)
Smoking history	1 (2%)
None	2 (4%)

DVT, Deep vein thrombosis.

were directly attributable to recurrent PE. The foundation of therapy for PE is anticoagulation with heparin. Adjunctive treatment options include thrombolysis, catheter embolectomy, and surgical embolectomy. Thrombolysis has a relatively high rate of intracranial bleeding (3% as reported by the International Cooperative Pulmonary Embolism Registry),¹ and catheter embolectomy might not retrieve all of the clot material.²

In the past, surgical pulmonary embolectomy has usually been reserved for patients with massive PE who present in cardiogenic shock. This approach is associated with high mortality rates, ranging from 16% to 64%.^{3,4} In October 1999,⁵ we liberalized the indications of surgical embolectomy for acute PE at our institution to include patients with large anatomically extensive clot and moderate-to-severe right ventricular dysfunction (RVD) without shock to improve early and late survival in this condition.⁵ The rationale for this approach is based on numerous observations that implicate RVD as an early and late risk factor for right ventricular (RV) failure, RV ischemic infarction,⁶ and death.⁷

Figure 1. Contrast-enhanced CT (axial section) showing large saddle embolus in the PA (black arrows).

Figure 2. Transesophageal echocardiogram revealing RV strain and bulging of the interatrial septum (arrowheads). RA, Right atrium; LA, left atrium.

Our multidisciplinary approach is based on rapid diagnosis with computed tomography (CT) and echocardiography to identify patients with severe RVD and likely poor early outcome.^{5,8} In this article we present our series of 47 consecutive patients who underwent surgical pulmonary embolectomy over a 4-year period.

Methods

From October 1999 through February 2004, 47 patients underwent surgical pulmonary embolectomy for treatment of acute PE. The study included 30 (30/47 [64%]) men. Mean age was 59 ± 14 years (median, 58 years; range, 24-86 years). Preoperative risk factors for development of PE are presented in Table 1. The most common presenting symptom was dyspnea (n = 40, 85%) followed by chest pain (n = 15, 32%), hemodynamic instability (n = 12, 26%), syncope (n = 6, 13%), and cardiac arrest (n = 6, 13%). Echocardiography was performed in 42 (89%) of 47 patients, and moderate or severe RVD was present in 40 (95%) of 42

TABLE 2. Indications for surgical embolectomy (n = 47)

Indication	N (%)
Contraindications to thrombolysis	21 (45%)
Recent surgical intervention	10 (21%)
Active bleeding	3 (6%)
Stroke	4 (9%)
Other	4 (9%)
Failed medical treatment	5 (10%)
Failure of thrombolytics	4 (9%)
Failure of catheter embolectomy	1 (2%)
Large RA-RV thrombus	5 (10%)
RV hemodynamic dysfunction	15 (32%)
Large PFO	1 (2%)

RA-RV, Right atrium–right ventricle; PFO, patent foramen ovale.

ACD

Figure 3. Kaplan-Meier survival curve after surgical pulmonary embolectomy (n = 47 patients, including the 3 operative deaths).

patients (Figure 1). RVD was defined as the presence of (1) an RV/left ventricular end-diastolic diameter ratio of greater than 1 in the apical 4-chamber view, (2) an RV end-diastolic diameter of greater than 30 mm, or (3) paradoxical RV septal systolic motion.

Chest CT scans were performed in 41 (87%) of 47 patients (Figure 2), and pulmonary angiography was performed in 5 (11%) of 47 patients. In one patient no diagnostic studies were done because the patient experienced cardiac arrest on arrival to the operating room, where he was scheduled for delayed chest closure after mitral valve repair. The diagnosis of PE was made intraoperatively.

Indications for surgical pulmonary embolectomy are summarized in Table 2. At initial evaluation, hypotensive patients were defined as those with a systolic blood pressure of less than 100 mm Hg, and normotensive patients were defined as those with a systolic blood pressure of greater than 100 mm Hg. Patients in shock were so designated if they presented with symptoms of hypoperfusion (ie, oliguria, cold and clammy skin, and lactic acidosis) or were in cardiac arrest. Two groups of patients were identified on the basis of echocardiographic examination and clinical status: normotensive patients with RVD (n = 15) and hypotensive patients with RVD (n = 28). In the group of patients who were hemodynamically unstable, 21 were not eligible for thrombolysis and received medical treatment. Five patients treated medically had hemodynamic deterioration despite treatment.

Surgical Technique

Transesophageal echocardiography was performed in all patients to assess the intracardiac structures for patent foramen ovale or atrial septal defect, which, if found, would lead to changes in cannulation and myocardial protection strategies. Epicardial echocardiography was also used in some cases to determine the location of intracardiac thrombi and identify direct cannulation sites. After median sternotomy, the patients were heparinized and cannulated for cardiopulmonary bypass (CPB). The operation was

performed under normothermic conditions without cardioplegic arrest unless repair of a patent foramen ovale or atrial septal defect was required. A longitudinal arteriotomy of the main pulmonary artery (PA) trunk was performed, and if necessary, an additional arteriotomy of the right PA between the ascending aorta and the superior vena cava was made. Alternatively, a transverse arteriotomy in the distal main PA extending onto the left or right PA was carried out. Clots were removed under direct vision by using gallbladder stone forceps and suction. Fogarty catheter clot extraction was avoided to reduce the possibility of injury to the PA branches. Temporary reductions in CPB flow were occasionally needed to permit better clot visualization. After weaning from CPB, an inferior vena caval filter was inserted in most cases through the right atrial purse string.

Statistical Analysis

The data are expressed as means $\pm$ SD or percentages. Long-term survival rates were calculated by using the Kaplan-Meier method. We used the STATA 7.0 for Windows (STATA, College Station, Tex) statistical software package.

Results

We observed the following distribution of emboli with respect to the pulmonary arteries: bilateral PAs and main trunk in 39 (83%) of 47 patients, left PA only in 2 (4%) of 47 patients, and right PA only in 6 (13%) of 47 patients. Inferior vena caval filters were inserted in 38 (81%) of 47 patients. Thirty-six patients received the filter after surgical embolectomy, and 2 received the filter preoperatively.

Three (6%) patients died within 30 days of the operation. A 50-year-old man died on postoperative day 19 after recurrent PE and severe RV failure and before we adopted the routine use of caval filters. Reoperative embolectomy with RV assist device placement was not successful. A 34-year-old man who previously underwent knee arthroscopy presented in cardiac arrest. He did recover hemodynamically after pulmonary embolectomy but never regained consciousness and was declared brain dead on the second postoperative day. The third patient was an 84-year-old woman who presented for embolectomy with worsening hypoxia after thrombolytic treatment failure. She experienced aortic dissection during cannulation and died as a result of intractable bleeding after aortic repair.

Two (4%) patients had reoperations for mediastinal bleeding, and 2 (4%) had deep sternal wound infections. An RV assist device was implanted in 2 (4%) patients, one of whom was successfully weaned.

Median follow-up was 27 months (range, 2-50 months), and follow-up was 100% complete in 44 surviving patients. There were 6 (12%) late deaths. Actuarial survival at 1 and 3 years' follow-up was 86% $\pm$ 5% (CI, 0.70-0.90) at 1 year and 83% $\pm$ 6% (CI, 0.66-0.92) at 3 years, respectively (Figure 3). Five of the 6 patients with late deaths were given diagnoses of cancer at the time of surgical embolectomy.

Discussion

The principal finding of this report is that use of expanded indications for pulmonary embolectomy was associated with a reduced operative risk. We considered embolectomy not only in patients with traditional indications, such as failed medical therapy, but also in hemodynamically stable patients with large central clot burden and documented RVD. A multidisciplinary approach, with 24-7 availability, rapid precise diagnosis, and rapid transfer to the operating room, is the key to our management strategy. Spiral CT offers rapid imaging acquisition and direct visualization of emboli within the pulmonary arteries and subsegmental vessels.⁹ The widespread availability of chest CT scanning renders this the preferred diagnostic tool for rapid and noninvasive detection of large central clot and superior to the classical contrast pulmonary angiography, which can cause hypotension. Cardiac biomarkers, such as troponin and brain natriuretic peptide increase, can identify patients with PE who are likely to do poorly on anticoagulation therapy alone.¹⁰

Thrombolytic treatment offers a more rapid rate of resolution of pulmonary emboli than the standard treatment with heparin alone and is indicated in patients with massive PE. There are, however, few studies focusing on the long-term outcome of patients who receive thrombolysis treatment and no data comparing surgical and medical treatment in patients eligible for both treatments.¹¹⁻¹³

Catheter embolectomy as a therapeutic modality can be performed during contrast pulmonary angiography. However, commercially available catheters tend to fragment the embolus rather than extracting it,² causing the embolus particles to propagate further into the pulmonary circulation, thus placing patients at risk for subsequent pulmonary hypertension.

Identification of a central surgically accessible PE (within the main trunk or left or right main PA) is required before considering surgical therapy. The best surgical candidates are those patients with a large amount of clot limited to the central main branches. Patients with most of their clot burden located peripherally do not do well with surgical intervention. Patients with central clotting that extends peripherally do well with surgical intervention, but the surgeon must be prepared to systemically cool on CPB to permit modulation of CPB flows for better visualization during clot removal.

We have extended the indications for surgical intervention beyond the traditional indications for massive PE (ie, failure of lytic treatment or hemodynamic compromise) to include hemodynamically stable patients with massive central clot burden and signs of RVD on echocardiogram. The latter represents a controversial group.^{11,12} Our approach was recently replicated by Yalamanchili and colleagues,¹⁴

with 8% mortality. Development of shock and multisystem organ failure as a consequence of RV failure is associated with at least 30% mortality, whereas if cardiac arrest occurs, mortality approaches 70%.⁷

A decrease of mortality in surgical PE series from 57% in the 1960s¹⁵ to 26% in the 1990s¹⁶ to 6% in our series has been documented. In the past, pulmonary embolectomy was the treatment of last resort for patients with PE because it was associated with high mortality. The average morbidity from different series between 1982 and 1999 is about 30%.¹⁴ This approach has changed over time. Increasingly more often, centers are reporting PE as an integral part of their treatment algorithm for patients with both massive and submassive PE, where massive PE is defined as the presence of persistent systemic hypotension or cardiogenic shock and signs of RVD, and submassive PE is defined as moderate-to-large clot, presence of RVD, and normal arterial blood pressure.¹⁷

Very few studies have compared medical versus surgical treatment for PE. In a nonrandomized comparison of surgical and medical treatment in hemodynamically compromised patients with massive PE, the medical group had an increased mortality rate, increased number of hemorrhagic events, and a higher rate of recurrent PE.¹⁸

Significantly higher mortality rates are observed in patients who undergo cardiopulmonary resuscitation (CPR).⁷ Furthermore, patients brought to the operating room undergoing continuous CPR have a higher mortality than those undergoing intermittent CPR with stable hemodynamics on arrival to the operating room (80% vs 40%, respectively).¹⁹ We experienced similar findings with our cohort. Six patients had cardiac arrest and required CPR, of whom 2 died. One patient had recurrent PE in the hospital and experienced a cardiac arrest. The other patient was transferred from an outside hospital under continuous CPR, which continued until the operation, and spontaneous rhythm was never established preoperatively. These results raise questions about the appropriateness of aggressive surgical therapy with out-of-hospital cardiac arrest and prolonged unsuccessful CPR. RVD without shock is a controversial, but we believe reasonable, indication for surgical embolectomy, because as PA-RV pressures increase, the right ventricle might ultimately fail. RV ischemia caused by interatrial septum displacement might reduce coronary artery perfusion to the right ventricle, which can cause RV ischemic infarction and death.^{6,7}

Another controversial issue centers around the perioperative placement of a vena caval filter. The recurrence rate of PE after surgical pulmonary embolectomy is as high as 5%.³ In a randomized trial assessing use of inferior vena caval filter placement versus no filter in patients with proximal deep-vein thrombosis, there were no differences in early or

late mortality.²⁰ However, the same study revealed the initial efficacy of filters for the prevention of PE in the first year. Because one of our first patients experienced a fatal recurrent PE, we now always place a vena caval filter at the time of surgical pulmonary embolectomy.

Limitations

The retrospective nature of our study is the main limitation. A prospective randomized trial of medical versus surgical treatment would be optimal.

Conclusion

In this report we document that surgical pulmonary embolectomy can be performed with low perioperative mortality and good midterm survival. Factors important to achieving a good result include a multidisciplinary approach with rapid noninvasive diagnostics, proper risk stratification, and availability of immediate surgical treatment. Thus we now offer surgical pulmonary embolectomy not only to patients with large central clot burden and severe hemodynamic compromise but also to hemodynamically stable patients with RVD documented by means of echocardiography.

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Discussion

Dr Kwok Yun (Los Angeles, Calif). The current study is a continuation of an initial series of 29 patients that was initially published in *Circulation* in March of 2002. From this study, the authors conclude that in addition to the traditional indications of hemodynamic instability or failure of medical management, surgical intervention should be extended to stable patients with signs of impending RVD and failure on the basis of echocardiography. What stands out in this report is not so much indisputable evidence that pulmonary embolectomy should be performed in this latter group of patients but rather the impressive success with those who were unstable and presented in cardiac arrest. This leads to my first question. According to the initial report, one of the lessons learned from the first 29 patients was that cardiac arrest was a relative contraindication to emergency surgical intervention. Yet 5 of the next 18 patients in the series had cardiac arrest. Has the philosophy regarding these high-risk patients changed in your institution, and if so, why?

Dr Byrne. It is all individualized. In an elderly person with comorbidities, we probably would not want to do the operation. All we have are the ones whom we operated on, so the ones who did have cardiac arrest, I think 6 of the 47, were typically young, otherwise healthy people who were previously healthy and had CPR. Now we appreciate that getting someone back is a tough problem because they do not oxygenate their blood very well, and they could end up brain dead. In fact, 2 of the 6 patients did die here, and therefore there was a 33% operative mortality instead of 6% overall. I agree it is a controversial area, but it is all individualized. For a young, otherwise healthy person, I think you should give that person a shot.

Dr Yun. Although only 3 patients died within 30 days of operations, according to your article, another patient could not be weaned from RV assist device, giving a hospital mortality of 8.5%. How many of the 5 late deaths were actually in-hospital deaths beyond the 30-day period?

Dr Byrne. The RV assist device was weaned in that patient. It was able to be weaned, but your question is how many out beyond 30 days? Well, we have the late, 2-year follow-up. We have 100% follow-up out to a median of 2 years, and there were 6 late deaths; 5 of 6 had cancers.

Dr Yun. Regarding the subset of 15 patients with preserved arterial pressure but significant RVD, did the decision to operate

rely solely on echocardiographic findings or was it based on a risk stratification scheme, such as the Geneva Prognostic Index, or additionally guided by the use of biomarkers, such as troponin and B-type natriuretic peptides?

Dr Byrne. That is a good question. We did not use any of the biomarkers. In the patients on whom we operated, all were symptomatic. About a third were hemodynamically stable, but their sole indication other than symptoms was impending RV failure, as documented by those echocardiograms that showed massively dilated dysfunctional RV. We do not have data on how many patients in whom we said, okay, their right ventricle is not that bad, let's hold off, but that is something that we need to do.

Dr Yun. To be the devil's advocate, in the Management Strategy and Prognosis of Pulmonary Embolism 3 trial comparing thrombolysis plus heparin with heparin alone in stable patients, the overall mortality was 2.7%, without significant differences between the 2 treatment arms. Unfortunately, the report does not specify the mortality rate of patients with impending hemodynamic instability because of RVD, which constituted about 30% of the cases, the same as in your series, which was about 32%. However, even if one assumes that all deaths occur in this subset of patients, the worst-case mortality would still be at a respectable 8.5%. Compared with the 6.5% in this series, what is the rationale for operation in the stable patient with RVD without some sort of a randomized trial unless there is a contraindication to thrombolysis?

Dr Byrne. That is a really good question. I think it comes down to a multidisciplinary approach that many cardiologists have bought into because they believe the operation is very effective and safe. I think it is just going to take some education, debate, and eventually a randomized trial to figure this one out. To be able to stratify RVD, we all need that denominator that we talked about before—the people with RVD but who did not undergo an operation—and see what their outcome was.

Dr Scott Mitchell (*Stanford, Calif*). We have a very aggressive group of interventionalists at Stanford, and therefore if we see these patients in the condition in which they are still clinically stable but have a contraindication to heparin or systemic thrombolysis, then we undergo regional thrombolysis with some of these mechanical devices. This seems like the perfect milieu when the patients are still stable. My question is, do you have experience

with those devices, and do you have the same aggressive interventionalists?

Dr Byrne. I think the answer is because we do not have the same aggressive interventionalists, we do not have that treatment arm at our hospital.

Dr John Chen (*Honolulu, Hawaii*). This is a very sick group of patients, and that is a quite an achievement to save their lives. We do not have a CAT scanner at the door, and therefore my question is as follows: Did you, in the course of your study, find any particular factors that would predict who these patients are?

Dr Byrne. There is a whole body of literature looking at people at risk for PE. If someone showed up with shortness of breath with or without chest pain who did not have other obvious causes, they got run through the CAT scan pretty quickly. There is a low threshold to consider it. It all has to do with thinking of the diagnosis and considering it and running them through the CAT scan.

Dr Chen. It sounds like at the Brigham they do not do a history and physical examination anymore. The CAT scan is the study of choice?

Dr Byrne. Well, you know, that emergency department. I try to go down there as little as possible because they might run me through the CAT scan. (laughter)

Dr Thor Sundt (*Rochester, Minn*). This is probably as much a comment as a question. Could you detail for us your technical approach to the right PA? I have found it particularly helpful to open the right PA behind the aorta, as one does for pulmonary thromboendarterectomy, and have taught the residents to do just that. I just wanted to highlight that technical point, which we believe is important to enable complete extraction of thrombus. I prefer this rather than trying to reach around a right-angle corner with a stone forceps from an incision in the main PA. Is this your practice?

Dr Byrne. Any time there is clot on the right side, we do that maneuver. It takes just a few minutes. I find it helpful, by the way, to stand on the left side of the table for these cases. I put on a head light, and I can see down that right PA beautifully. The left PA is of course going straight down, and you can see there, but the right PA would be very hard to—you would be putting your head in the field to try to look down the right PA, but from the left side of the table you can see it really well.