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REVIEW

Acute pulmonary embolectomy

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Summary

Acute pulmonary embolism (PE) is a common condition frequently associated with a high mortality worldwide. It can be classified into non-massive, sub-massive and massive, based on the degree of haemodynamic compromise. Surgical pulmonary embolectomy, despite having been in existence for over 100 years, is generally regarded as an option of last resort, with expectedly high mortality rates. Recent advances in diagnosis and recognition of key qualitative predictors of mortality, such as right ventricular stress on echocardiography, have enabled the re-exploration of surgical pulmonary embolectomy for use in patients prior to the development of significant circulatory collapse, with promising results. We aim to review the literature and discuss the indications, perioperative workup and outcomes of surgical pulmonary embolectomy in the management of acute PE.

Keywords: Pulmonary embolectomy • Embolism • Surgery • Thrombolysis

INTRODUCTION

Pulmonary embolism (PE) is a common clinical condition associated with significant morbidity and mortality. Surgical pulmonary embolectomy has traditionally been reserved for the most severely compromised of patients not amenable to medical therapy. Accurate diagnosis and recognition of specific biochemical (brain natriuretic peptides and cardiac troponins) and echocardiographic surrogates of haemodynamic stress have improved risk stratification. Consequently, recent studies have demonstrated impressive outcomes of surgical embolectomy through careful patient selection.

We provide an overview of acute PE and discuss surgical pulmonary embolectomy, in terms of indications, perioperative management and its contemporary role, as part of a multidisciplinary approach, in the management of acute PE.

ACUTE PULMONARY EMBOLISM

Incidence and definition

Acute PE is a common clinical problem, with an estimated incidence of 600 000 cases annually in the USA and accounting for 50 000 to 200 000 deaths [1]. According to data from the UK General Practice Research Database (GPRD), the incidence of PE between 1994 and 2000 was 34.2 per 100 000 patient-years [2].

In recent times, acute PE has become a matter of public concern due to the wide dissemination, by the media, of the so-called economy class syndrome due to a number of dramatic cases with lethal outcomes as observed across numerous international airports. The association between pulmonary thromboembolism (PTE) and prolonged air travel, however, has been well reported in the medical literature for many years [3, 4]. Although such a risk is still poorly defined, the incidence of severe PTE during air travel appears to be low. A study of international flights arriving at the Madrid-Barajas International Airport, over a period of 6 years, found an incidence of 0.39 episodes of PTE per million passengers, with all cases involving flights of more than 6 h duration [5]. Similar observations were seen at airports in Paris and London [6, 7]. Risk factors for PE also include obesity, increasing age (mean age with acute PE is 62 years) [8], cigarette smoking, hypertension, oral contraceptive use [9] and pregnancy. Conditions limiting mobility such as recent surgery [10], cardiac failure and respiratory failure, in addition to malignancies (particularly brain, ovarian and pancreatic) [11], have also been associated with PE. Of the latter, compression of the inferior vena cava by malignancies have also been associated with PE. Approximately 20% of patients in the International Cooperative PE Registry (ICOPER) had idiopathic PE [8].

The use of low-dose subcutaneous unfractionated heparin or low-molecular-weight heparin (LMWH) in the hospital setting has

Table 1: Treatment options for PE [44]

	Indication	Contraindication
Anticoagulation	Normotensive and normal RV function INR target 2.5 (range 2.0–3.0) **Clinical scenario** — **Duration of anticoagulation** PE secondary to a reversible risk factor — 3 months, vitamin K antagonist Unprovoked PE — ≥3 months, vitamin K antagonist Recurrent unprovoked PE — Indefinite, vitamin K antagonist PE and cancer — LMWH for the first 3–6 months, followed by indefinite vitamin K antagonist or LMWH therapy	
Thrombolysis	Normotensive and RV dysfunction PE causing haemodynamic compromise	See Table 2 Severely compromised patients requiring emergency embolectomy
Catheter embolectomy	Contraindications to thrombolytic therapy Failure of thrombolytic therapy Surgical embolectomy is unavailable	Expertise or facility unavailable
Surgical embolectomy	Failed thrombolytic therapy Failed catheter embolectomy Insufficient time for effective thrombolytic therapy in critical patients	Chronic thromboembolism Active gastrointestinal/surgical site bleeding

INR: international normalized ratio.

led to a decrease in the incidence of symptomatic venous thromboembolic disease [12]. A large meta-analysis demonstrated that the perioperative use of subcutaneous unfractionated heparin in prophylactic doses (5000 units started 2 h before surgery and continued every 8 or 12 h) reduced rates of symptomatic PE (from 2.0 to 1.3%) and fatal PE (from 0.8 to 0.3%) [13].

Despite advances in diagnosis and therapy, acute PE is still associated with high mortality. In the International Cooperative Pulmonary Embolism Registry of 2454 consecutive patients, the overall 3-month mortality rate was 17.4% [8]. Patients with PE may be divided into two groups based on clinical presentation. Massive PE, characterized by arterial hypotension (<90 mmHg or a drop of >40 mmHg for >15 min), and cardiogenic shock, have an in-hospital mortality of 25 to >50% [8, 14, 15]. Patients with non-massive PE are normotensive and have a much lower risk of death (3–15%) [16]. Recent focus on a subset of these normotensive patients with subclinical haemodynamic impairment ('sub-massive PE' cohort), characterized by right ventricle (RV) dysfunction, has shown increased mortality compared with normotensive patients without RV compromise [1, 8, 16–18].

Therapeutic options

Current therapeutic options include medical management (anticoagulation and fibrinolytic therapy), catheter embolectomy and surgical embolectomy (Table 1).

Anticoagulation with initial unfractionated heparin or LMWH (e.g. enoxaparin) followed by a vitamin K antagonist (e.g. warfarin) remains the cornerstone of acute PE therapy. Rapid risk stratification is critical and, in patients with haemodynamic compromise, fibrinolysis is recommended as first-line treatment unless major contraindications exist [19] (Table 2). Although fibrinolysis has been shown to reduce the risk of death or recurrent PE by 55% when compared with heparin [20], it carries a significant risk of intracranial haemorrhage of 3% [8], with overall major bleeding rates approaching 20% [21]. Patients must be carefully screened, as absolute contraindications to fibrinolysis exist in one-third of patients with massive PE [14].

Table 2: Contraindications for fibrinolysis and surgical pulmonary embolectomy

Absolute contraindications	Relative contraindications
Fibrinolytic therapy [19, 59]	
Haemorrhagic stroke or stroke of unknown origin at time	Transient ischaemic stroke in last 6 months
Ischaemic stroke in last 6 months	Oral anticoagulant therapy
Central nervous system neoplasms	Pregnancy
Recent major trauma/surgery/head injury within last 3 weeks	Advanced hepatic disease
Gastrointestinal bleeding within last 1 month	Infective endocarditis
Known active bleeding	Active peptic ulcer Traumatic resuscitation Refractory hypertension (SBP >180 mmHg) Traumatic resuscitation
Surgical pulmonary embolectomy [41]	
Acute on chronic PE	Failed thrombolysis
Active bleeding (GI tract or other surgical site)	Stroke (ischaemic or haemorrhagic)
Lack of qualified cardiothoracic personnel, CPB equipment and intensive care facility	Critically ill patients

SBP: systolic blood pressure; GI: gastrointestinal; CPB: cardiopulmonary bypass.

Recent evidence has shown outpatient management of acute PE in low-risk patients to be a safe alternative, with comparable rates of hospital readmission, to their inpatient counterparts [22].

Mechanical extraction of pulmonary thrombi via a catheter device (catheter embolectomy) is a novel therapeutic approach that has an observed success rate of 86% and major complications of 2.4% [23]. Distal showering of emboli from proximal clot

fragmentation may occur, and recurrence of embolus or incomplete embolectomy is more commonly seen than with surgical embolectomy, placing patients at higher risk of pulmonary hypertension. Catheter embolectomy is recommended by the American College of Chest Physicians (ACCP) for use in carefully selected patients with haemodynamic compromise who fail or are unable to receive fibrinolytic therapy, and where surgical embolectomy is unavailable [24].

SURGICAL PULMONARY EMBOLECTOMY

Background

Surgical pulmonary embolectomy was pioneered by Trendelenburg in 1908. Earlier attempts saw significant hypoxic brain injury in the few patients who survived the procedure. With the advent of cardiopulmonary bypass (CPB) and the application of heparin as an anticoagulant, Sharp [25] became the first surgeon to successfully perform pulmonary embolectomy using extracorporeal circulation in 1962. Improved diagnostic adjuncts, surgical techniques and careful patient selection have seen a decline in mortality from 35 to 19% over a 30-year period [26]. Recent reports have suggested in-hospital mortality from surgical embolectomy to be as low as 5–6% (Table 3).

Preoperative considerations

Most deaths from massive PE occur within the first few hours of the initial event [27]. Expedient diagnosis and risk stratification are paramount in patients with acute PE. As the clinical diagnosis of PE is highly unreliable due to the wide spectrum of non-specific symptoms and signs, a definitive diagnosis must be made prior to surgical embolectomy (Table 4).

Table 3: Survey of surgical pulmonary embolectomy results

Year	Author	No. of patients	In-hospital mortality	
			Overall	Patients with preoperative arrest
1968	Cooley and Beall [60]	11	55% (6)	100% (3/3)
1972	Clarke and Abrams [61]	26	50% (13)	100% (5/5)
1991	Meyer *et al.* [62]	96	38% (36)	58% (14/24)
1994	Stulz *et al.* [27]	50	46% (23)	61% (19/31)
1994	Gulba *et al.* [55]	13	23% (3)	Not reported
2002	Aklog *et al.* [58]	29	10% (3)	100% (1/1)
2004	Yalamanchili [63]	13	8% (1)	50% (1/2)
2005	Sukhija *et al.* [18]	18	11% (2)	Not reported
2005	Leacche *et al.* [38]	47	6% (3)	33% (2/6)
2006	Spagnolo *et al.* [42][a]	21	0% (0)	0% (0/2)
2008	Kadner *et al.* [39]	25	8% (2)	25% (2/8)
2010	Carvalho *et al.* [56]	16	43% (7)	85% (6/7)
2010	Vohra *et al.* [43]	21	19% (4)	Not reported
2011	Fukuda *et al.* [64]	19	5% (1)	0% (0/4)
2011	Zarrabi *et al.* [37][a]	30	7% (2)	33% (1/3)

[a]Retrograde pulmonary perfusion used in addition to the standard antegrade technique.

Multidetector row spiral computed tomography (CT) pulmonary angiography (CTPA) has now replaced invasive pulmonary angiography as the primary imaging test for the diagnosis of PE. Recent evidence supports its use as a stand-alone test in the exclusion of PE, with high predictive values in concordance with clinical assessment [19, 28]. Identification of central surgically accessible embolism, preferably limited to the proximal main pulmonary arteries, is required before proceeding to surgery (Fig. 1). A CT scan also provides useful prognostic information from imaging of the right ventricle (RV) and left ventricle (LV). In patients with PE, CT findings of RV enlargement indicate a 5-fold increase in 30-day mortality [29].

Apart from confirming PE, CT scan has also been useful in the detection of concomitant intracardiac pathology, such as paradoxical embolus trapped within a patent foramen ovale (PFO) or cardiac tumours (Fig. 2) [19, 30–32]. Further, extracardiac causes of thromboembolism, such as abdominal or pelvic masses compressing the deep venous system, may be discovered on CT. Occasionally, this may reveal significant concurrent thrombi within the iliac, femoral or popliteal deep venous systems [30]. Pulmonary sarcoma can rarely mimic PE on CT (Fig. 3) [31].

In the absence of PE, alternative diagnoses such as pneumonia, emphysema, pulmonary mass or aortic disease may be identified on the CT scan. Pulmonary angiography, the traditional gold standard for the diagnosis of PE, is useful when the results of CTPA are equivocal.

Echocardiography, although not a reliable diagnostic tool for acute PE, is now recognized as a powerful predictor of increased mortality in patients with PE through identification of those with RV dysfunction suggestive of early haemodynamic compromise [8, 16, 17]. This group of patients, with so-called sub-massive PE, show a 6-fold increase in the relative risk of in-hospital mortality [33]. However, such indirect signs of PE related to RV overload or

Table 4: Preoperative investigations

Computed tomography
CTPA is now considered a stand-alone diagnostic tool for PE
Detection of centrally located embolism amenable to surgery
Detects RV enlargement as a poor prognostic marker
May diagnose other intracardiac and extracardiac pathology
Serial imaging to monitor resolution of PE and effectiveness of treatment
Pulmonary angiography (conventional invasive test)
Reserved for equivocal CTPA outcome
Transthoracic echo cardiography (TTE)
Not a reliable diagnostic tool for PE
Useful in the detection of RV dysfunction; correlates with increased mortality in PE
Identifies other concomitant intracardiac pathology
Monitors resolution of RV dysfunction; an indirect marker of PE resolution and response to treatment
Transoesophageal echocardiography (TOE)
Diagnostic tool by direct visualization of central PE
Emergent on-table diagnosis of suspected PE in critically ill patients taken straight to room
Intraoperative TOE to detect concomitant intracardiac pathology; with implications for operative strategies
Biochemical markers (troponin and BNP)
Concurrent elevation of troponin and BNP signifies increased mortality in normotensive PE patients

BNP: brain natriuretic peptide; CTPA: computed tomography pulmonary angiography.

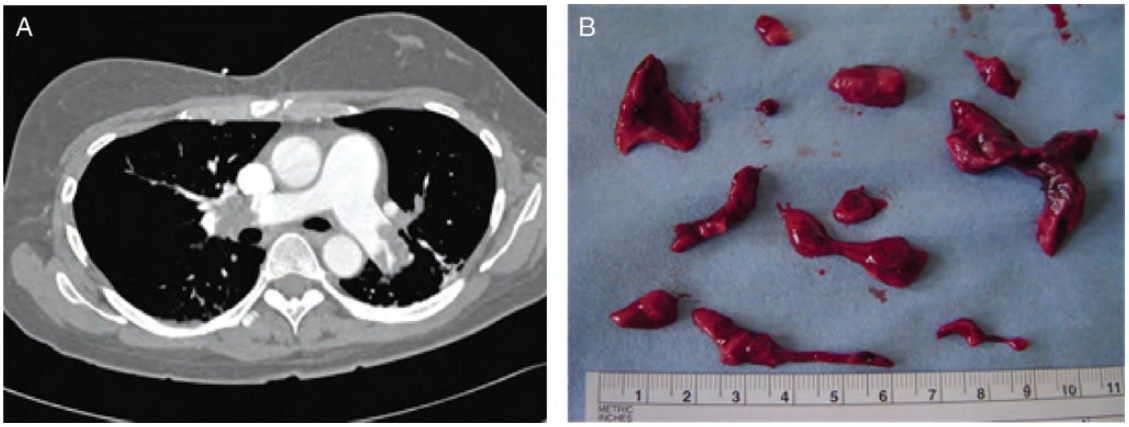

Figure 1: (**A**) CTPA showing multiple filling defects secondary to multiple pulmonary emboli. (**B**) Multiple fresh extracted pulmonary emboli (Reprinted from Choong *et al*. [30], with permission from Elsevier).

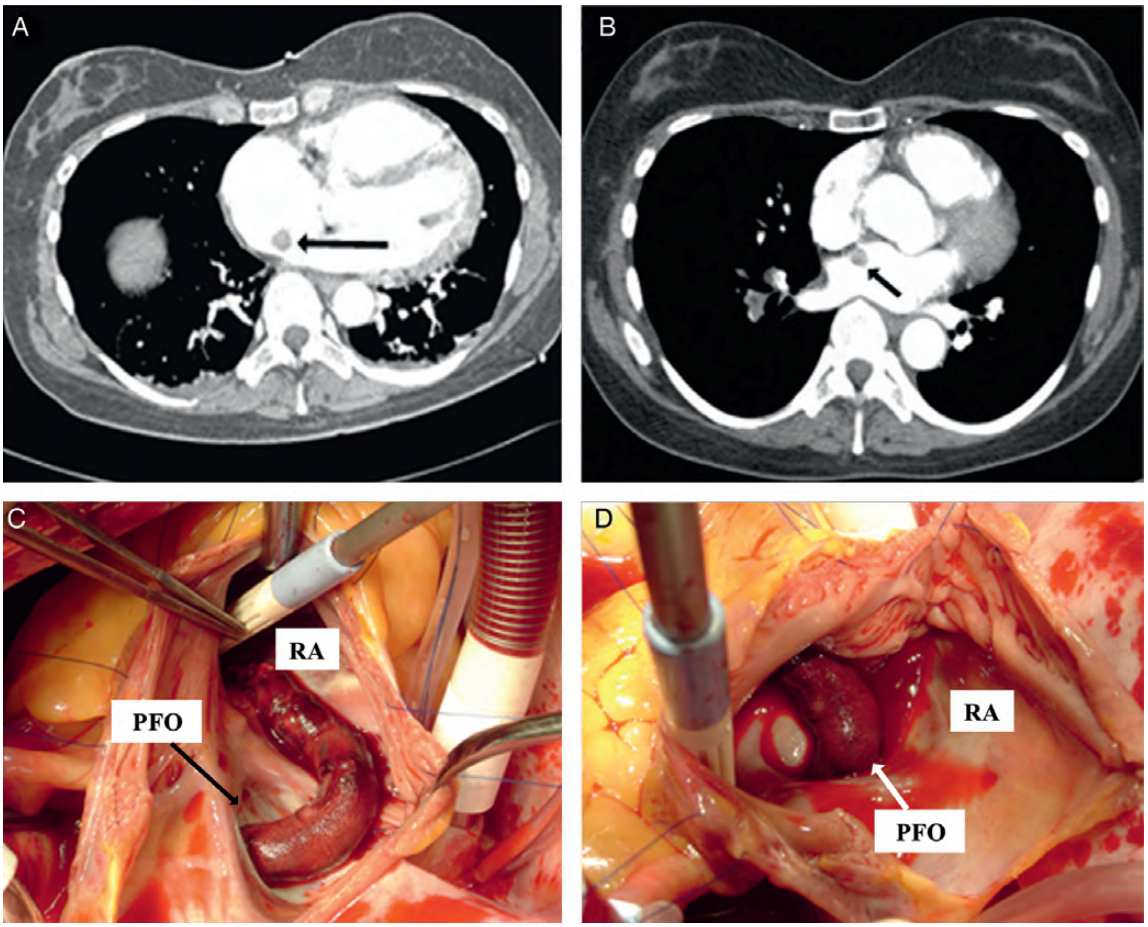

Figure 2: (**A**) CT showing a filling defect (arrow) in the right atrium secondary to an embolus trapped within the PFO. (**B**) CT showing filling defect in left atrium caused by same embolus in (**A**), traversing through PFO. (**C** and **D**) Paradoxical embolus traversing through PFO (Reprinted from [30], with permission from Elsevier).

dysfunction may be seen in concomitant cardiorespiratory disease, in the absence of PE. Echocardiography may identify acute myocardial abnormalities, pericardial tamponade or aortic dissection, which can also mimic PE [34]. In a compromised patient with suspected PE, the absence of echocardiographic signs of RV overload or dysfunction practically excludes the diagnosis as a cause of haemodynamic compromise [19].

Echocardiography can identify other concomitant pathologies such as free-floating right heart thrombi (FRHTI), PFO or atrial septal defect (ASD) [17]. FRHTI with acute PE is associated with high early mortality of 80–100%, and anticoagulation alone is frequently inadequate, with pulmonary embolectomy often indicated in this situation [19]. Likewise, PFO in a patient with significant PE signifies a particularly high risk of death (10-fold

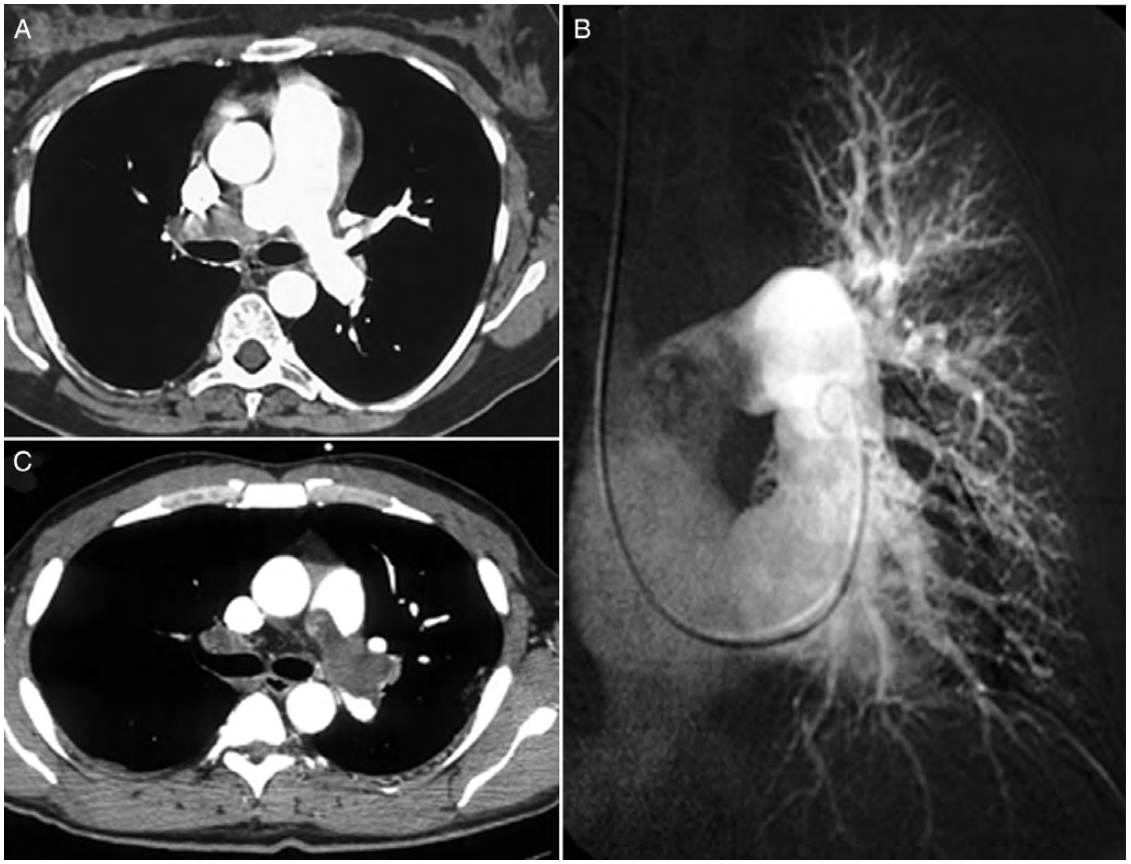

Figure 3: Pulmonary sarcoma mimicking PE on the CT scan. (**A**) CT demonstrating complete occlusion of the right pulmonary artery. (**B**) Pulmonary arteriogram in the same patient as *A* showing total occlusion of the right pulmonary artery, whereas the left pulmonary artery and its branch vessels were normal. (**C**) The CT scan showing a massive saddle 'embolus' at the bifurcation of the pulmonary artery in another patient. (Reprinted from Choong *et al*. [30], with permission from Elsevier).

increase) and arterial thromboembolic complications (5-fold increase) [17].

In patients treated with thrombolysis, a baseline echocardiogram is advised. Persistent clinical instability and residual echocardiographic RV dysfunction imply unsuccessful thrombolysis. In this scenario, rescue surgical embolectomy is observed to have a lower mortality rate than repeat thrombolysis (7 vs 38%) [35].

Critically, ill patients with contraindications to thrombolysis who are taken directly to the operating room must have an emergent transoesophageal echocardiography (TOE) performed prior to sternotomy to ensure the appropriateness of surgery. Large central PEs may be directly visualized on TOE. Anecdotally, in patients who have already received thrombolysis, on-table TOE may reveal a PE that has significantly improved by the time the patient is prepared for surgery, thereby obviating the need for operative management. In view of that, some surgeons consider it reasonable for the medical team to proceed with thrombolysis, providing there are no contraindications, while waiting for the surgical team to assess the patient. The rationale is that thrombolysis may provide potential benefits and any potential bleeding risks associated with thrombolysis can be dealt with in room if necessary.

Elevated cardiac biomarkers, such as troponin and brain natriuretic peptide, can identify patients with PE who are likely to have poorer outcomes treated with anticoagulation therapy alone [16]. Simultaneous measurements of troponin and brain natriuretic peptide were found to stratify normotensive PE patients more accurately than either test alone. PE-related 40-day mortality in patients with concurrent elevation of both markers >30% [36].

A multidisciplinary approach based on rapid diagnosis and risk stratification is key in identifying PE patients with significant RV dysfunction who are likely to have worse early outcomes, thereby allowing early surgical management of amenable patients.

Indications

Most patients with PE do not require pulmonary embolectomy. Surgical embolectomy is indicated in patients with: (i) massive PE, confirmed on angiography if possible; (ii) haemodynamic instability despite anticoagulation therapy and resuscitative efforts; (iii) failure of thrombolytic therapy or a contraindication to its use and (iv) in critical patients with insufficient time for systemic thrombolysis to be effective [19, 24].

The decision to proceed to surgical embolectomy must be in the setting of a definitive diagnosis, as a clinical diagnosis of PE is often unreliable. In highly unstable patients taken directly to the operating room without a definitive diagnosis, on-table TOE and colour flow Doppler can help confirm PE.

The traditional recommendation for surgery only in the most compromised patients, often after failure of medical management, delays surgery and accounts for the high mortality

rates observed. However, recent interest has focused on pulmonary embolectomy as first-line treatment in haemodynamically stable patients showing echocardiographic RV dysfunction. Early surgery in these impending circulatory failure patients has shown promising results [37, 38].

Failure of thrombolysis. Meneveau *et al.* [35] found that in patients with massive PE who failed initial thrombolytic therapy, pulmonary embolectomy resulted in an in-hospital outcome superior to that of repeat thrombolysis. Although the risk of major bleeding was similar between the groups, all bleeding events in the repeat-thrombolysis group proved fatal. Other studies also support surgical embolectomy as a successful treatment strategy in massive PE when compared with medical treatment, with lower mortality rates, a lower number of haemorrhagic events and recurrent thrombosis [39].

Concomitant cardiac pathology. Echocardiographic detection of concomitant FRHTI, PFO or ASD is also indications for pulmonary embolectomy. FRHTI is considered a sitting time bomb, with impending PE and potential circulatory collapse, making surgery time critical. It is associated with high rates of morbidity and mortality, as discussed earlier. PFO and ASD allow for paradoxical thromboembolism in the systemic circulation. Spectacular 'paradoxical embolus caught red-handed' has also been reported in the literature, where FRHTI was found trapped within the PFO, necessitating emergency surgery (Fig. 4) [30].

Cerebrovascular accident (ischaemic and haemorrhagic). Ischaemic or haemorrhagic stroke patients who are critically ill as a result of massive PE may benefit from pulmonary embolectomy when no alternative rescue procedure is available [40]. Minimizing the duration on extracorporeal circulation, early cessation of anticoagulation following the reversal of RV dysfunction, and the insertion of inferior vena cava (IVC) filter were shown to achieve good outcomes in these patients [26].

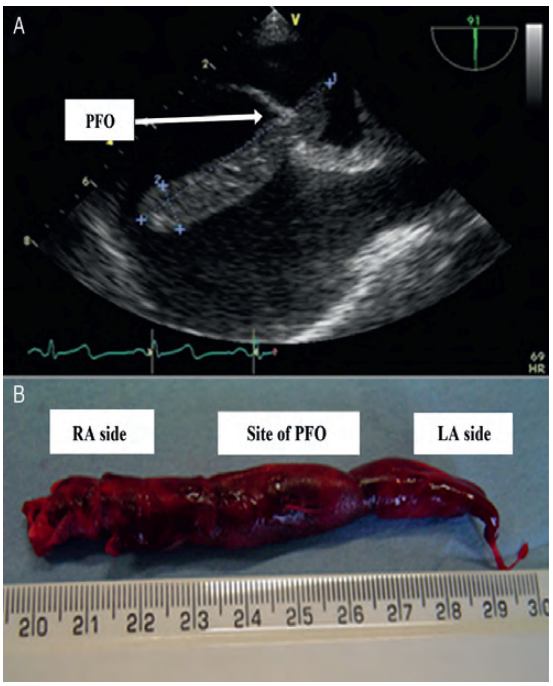

Figure 4: (**A**) Echocardiography showing a 9-cm long mass (7 cm long in the right atrium and 2 cm long in the left atrium), traversing through a PFO. (**B**) Nine-cm long embolus removed intact (Reprinted from Choong *et al.* [30], with permission from Elsevier).

Contraindications

Pulmonary embolectomy is contraindicated (Table 2) in acute-on-chronic PE, where there is a persistent high risk of pulmonary hypertension, right heart failure and intractable pulmonary haemorrhage. Surgical endarterectomy is indicated in this situation [41].

Active bleeding, from the gastrointestinal tract or other surgical site, is another contraindication owing to the significant risk of potentiating further haemorrhage in the setting of heparinization while on CPB. However, in stroke patients with fatal PE, rescue surgical embolectomy may be feasible if no alternative therapies are available [40].

Management of acute massive PE following failed thrombolysis is not well defined. Despite the perceivable concerns of surgical embolectomy in this cohort, Meneveau *et al.* [35] showed that previous thrombolysis is not an absolute contraindication. Outcomes in surgical embolectomy following failed thrombolysis were found to be superior to repeat thrombolysis in terms of lower mortality and recurrence of PE [35].

While haemodynamic instability constitutes the most frequent indication for surgical embolectomy [26], it is recognized that out-of-hospital cardiac arrests without restoration of spontaneous heart beat preoperatively have a particularly dismal prognosis.

Operative technique and complications

We recommend intraoperative TOE for all patients to assess for the presence of intracardiac pathology, such as PFO or ASD, which may affect cannulation and myocardial protective and operative strategies.

A median sternotomy is used. The pericardium is entered and CPB is established using bicaval cannulation and ascending aortic perfusion after institution of heparin. Other alternative sites for venous drainage and arterial return may also be contemplated in accordance with the patient's condition and surgeons' preference. In the absence of concomitant cardiac procedures, cardioplegic or fibrillatory arrest and aortic cross-clamping can be avoided to minimize potential cardiac ischaemic injury. The pulmonary trunk is exposed and a longitudinal arteriotomy starting 1.5 cm distal to the pulmonary valve and extending to the proximal left pulmonary artery is performed. Forceps and suction catheters are used to gently extract clots, under direct vision, where possible. Fogarty catheter extraction of peripheral clots must be done with great care to avoid injuring the thin-walled pulmonary artery branches. Alternatively, the pleural spaces are entered and gentle compression of each lung with concurrent arterial tree saline irrigation can help dislodge small peripheral clots into larger vessels, and they may then be suctioned out. This manoeuvre should not be used in patients on thrombolytic therapy as intractable endobronchial haemorrhage may occur. An additional right pulmonary arteriotomy is made for further clot extraction, if necessary. Following embolectomy, the arteriotomy is closed and the patient is weaned off CPB. Inotrope assistance is frequently required in patients with preoperative right ventricular dysfunction.

Infrequently, retrograde pulmonary perfusion has been used as an adjunct to the conventional antegrade technique described above. It may help dislodge clots in the peripheral pulmonary vasculature not directly visualized. Its proponents further refer to its ability to prevent air embolism, thereby minimizing subsequent lung injury [37, 42].

Recurrent pulmonary embolism. Recurrence of PE is a serious complication. To date, the efficacy of IVC filter insertion after pulmonary embolectomy remains controversial. In ICOPER patients with massive PE, IVC filters appeared to reduce recurrent PE and mortality at 90 days. This is supported by Meneveau *et al*. [35] in their study comparing repeat thrombolysis and rescue embolectomy. Recurrent PE was responsible for one-third of deaths in the former, while no recurrence was demonstrated in the surgical group. All surgical embolectomy patients had IVC insertion prior to sternal closure. In contrast, Vohra *et al*. [43] observed no recurrent PE following surgical embolectomy despite the lack of filter insertion.

The significant mortality arising from re-embolization has resulted in some surgeons preferring to insert caval filters where possible, either at the end of embolectomy or postoperatively by radiologists.

Current ACCP guidelines recommend vena cava filter use in the setting of acute PE if anticoagulation is contraindicated or in the presence of recurrent venous thrombosis despite adequate anticoagulation [24, 44]. The initial beneficial effect of IVC filters may be counterbalanced by an increased risk of deep-vein thrombosis [45].

Massive endobronchial haemorrhage. There are several reports of massive endobronchial haemorrhage following pulmonary embolectomy [46–49]. Two aetiological factors have been suggested: (i) direct mechanical injury to the pulmonary arterial wall during the act of clot removal and (ii) reperfusion injury after the re-establishment of pulmonary blood flow, either during or after CPB. The control of haemorrhage under heparinized conditions is challenging. Prompt identification of the side or site of arterial injury is vital. Successful management of this complication has been achieved by: (i) the use of a double-lumen endotracheal tube for selective collapse of the lung and segregating haemorrhage into a solitary main-stem bronchus [50]; (ii) tamponading the airway with an inflated Fogarty catheter [51] and (iii) thoracotomy with staple wedge resection of the infarcted portion of lung [46]. The immediate institution of positive end-expiratory pressure (20 cm H_2O) may also aid haemostasis [49].

Postoperative management

Routine postoperative care, as for any cardiac surgery patient, is instituted. Although most patients can be weaned off CPB, those requiring significant resuscitation leading to embolectomy may need postoperative mechanical assistance for persistent RV failure. Renal failure and ischaemic brain injury from inadequate preoperative circulatory state may become apparent in these patients.

In the absence of excessive bleeding, heparin anticoagulation is commenced 6 h postoperatively and continued until an oral vitamin K antagonist is therapeutic (INR range 2.0–3.0). The duration of long-term anticoagulation should be based on three factors: (i) the risk of recurrence after cessation of treatment; (ii) the risk of bleeding during treatment and (iii) the patient's preference. The recommended duration of anticoagulation therapy is listed in Table 1.

Surgical outcomes

Without prompt accurate diagnosis and aggressive treatment, acute PE is frequently fatal. Massive and sub-massive PE remain a major therapeutic challenge, with mortality rates of up to 60% in the first 6 h of the initial event [8, 52–54].

Surgical pulmonary embolectomy has traditionally been indicated in severely compromised patients who have failed conventional medical management. In a systematic review by Stein *et al*. [26], patients who experienced cardiac arrest before pulmonary embolectomy had a 3-fold increase in surgical mortality compared with those who did not (59 vs 20%). In the same study, it was shown that, over a 50-year period, the proportion of pulmonary embolectomy patients who presented with preoperative haemodynamic instability had remained unchanged: 74% from 1961 to 1984, and 74% from 1985 to 2006. Despite the critical preoperative status of most patients selected for embolectomy, surgical mortality rates have decreased substantially over time (Table 3). A multidisciplinary approach to diagnosis, treatment and postoperative care has even seen favourable results in patients who present with cardiac arrest. A recent study by Kadner *et al*. [39] revealed an 8% 30-day mortality rate in this population.

To date, very few studies have compared medical and surgical treatment options for patients with massive PE. The two retrospective studies that offer such insights both favoured surgical embolectomy over thrombolysis in terms of lower mortality and PE recurrence. Among a small cohort of patients with shock and massive PE, Gulba *et al*. showed a mortality of 23% in the 13 surgically treated patients compared 33% in the 24 patients who could not be promptly operated on (thus treated with thrombolysis). Recurrent PE rates of 15 and 25% were also observed, respectively. In the other study by Meneveau *et al*., among 40 patients who failed initial thrombolysis, 14 proceeded to rescue surgical embolectomy, while 26 were treated by repeat thrombolysis. Again, the surgical cohort achieved lower rates of mortality (7 vs 38%) and PE recurrence (0 vs 35%) compared with the medical cohort [35, 55]. Despite the positive surgical outcomes, it is difficult to generalize these observations given the small, retrospective cohort. A prospective randomized trial would be ideal, albeit difficult to implement.

Multiple surgical authorities have emphasized the critical importance of early surgical intervention. Recently, several major centres have liberalized the use of surgical embolectomy to include haemodynamically stable patients with echocardiographic evidence of moderate-to-severe RV dysfunction [38, 53, 56, 57]. Such findings are now recognized as strong independent predictors of RV failure and mortality in patients with PE. Of note, a retrospective study by Leacche *et al*. [38] examined 47 such patients who underwent early pulmonary embolectomy and observed an impressive operative mortality of 6%. An earlier study from the same institution analysing 29 overlapping patients showed a mortality of 10% [58]. Similar results have been demonstrated by Digonnet *et al*. [57, 58]. The use of retrograde pulmonary perfusion as an adjunct to antegrade pulmonary embolectomy has also seen favourable results, with 6 and 0% in-hospital mortality rates reported by Zarrabi *et al*. [37] and Spagnolo *et al*. [42], respectively. However, comparative

prospective studies are lacking to establish its role as a routine surgical technique.

CONCLUSIONS

Recent advances in surgical techniques, coupled with a multidisciplinary approach to diagnosis, risk stratification and perioperative care, have significantly decreased the mortality associated with surgical pulmonary embolectomy. Sufficient evidence exists to support widening its role, from a strictly rescue therapy for the most severely compromised patients, to those who show evidence of RV dysfunction in the absence of haemodynamic collapse.

Conflict of interest: none declared.

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