Management of pulmonary embolism with rheolytic thrombectomy

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Pulmonary embolism (PE) has been ascribed the cause of death in up to 10% of patients (1,2). In patients who are in shock, or who experience significant right ventricular dysfunction (RVD), therapy is aimed at reducing the thrombus load as quickly as possible. In the absence of contraindications, guidelines support the use of systemic thrombolytic therapy in these patients (3). However, even when pre-screened for absolute contraindications, the major bleeding rate can be as high as 28%, including up to 5% risk of intracranial hemorrhage (4-6). Surgical embolectomy can be very effective for large central thrombi, but requires a dedicated team and system to be in place (7). Catheter-based technology that combines clot destruction with local thrombolysis (hybrid therapy) offers a relatively simple and quick alternative that can manage both central and peripheral thrombus (8,9). In addition, it may result in improved long-term reduction in pulmonary hypertension and right heart failure (10). In the present case series, we describe our results using the AngioJet (Possis Medical, USA), a rheolytic thrombectomy device, in conjunction with a directed, recombinant tissue plasminogen activator (r-tPA) (Genentech, USA) for the management of PE.

La prise en charge de l’embolie pulmonaire par thrombectomie rhéolytique

HISTORIQUE : La thrombectomie par cathéter, qui combine la destruction des thrombus avec la thrombolysie localisée, est utilisée chez les patients ayant une embolie pulmonaire (EP) qui sont instables ou présentent une importante dysfonction du cœur droit, mais qui ont des contre-indications au traitement thrombolytique systémique.

OBJECTIFS : Évaluer les issues des patients qui ont subi une embolectomie pulmonaire au moyen d’un dispositif de thrombectomie commercial.

MÉTHODOLOGIE : Les chercheurs ont procédé à une analyse rétrospective des dossiers de patients qui ont subi une embolectomie pulmonaire entre mars 2007 et août 2009. Les patients ont été classés entre une EP massive ou submassive de forme clinique et une dysfonction ventriculaire droite modérée ou grave. Les données colligées incluaient l’indice de choc avant et après l’intervention (la fréquence cardiaque divisée par la tension artérielle systolique) et la tension artérielle pulmonaire moyenne.

RÉSULTATS : Seize patients d’un âge moyen (± ET) de 54,4±15,8 ans ont subi une embolectomie. Cinq avaient une ép massive de forme clinique (deux en choc cardiogène) et trois des 11 cas d’EP submassive avaient une dysfonction ventriculaire droite grave. Tous étaient présumés présenter des contre-indications à la lyse systémique. Les deux indices de choc (1,02±33 avant l’intervention par rapport à 0,71±0,2 après l’intervention [P=0,001]) et la tension artérielle pulmonaire moyenne (34,5±9,9 mmHg avant l’intervention par rapport à 27,1±7,1 après l’intervention [P<0,01]) se sont améliorés. Dans le groupe d’EP massive, un patient est décédé, et deux survivants ont subi une hémorragie rétropéritonéale et une insuffisance rénale transitoire. Au suivi (17,3±7,8 mois), deux patients ayant partie du groupe d’EP massive ont démontré des manifestations bénignes de cœur pulmonaire.

CONCLUSION : La thrombectomie rhéolytique est une stratégie efficace pour prendre en charge l’EP massive, notamment chez les patients qui présentent des contre-indications bien définies à une lyse systémique. Son efficacité pour traiter l’EP n’est pas aussi bien définie, mais mérite une comparaison avec la lyse systémique.
TABLE 1
Clinically submassive pulmonary embolism cases

| Pt | Age, years/sex | RVD | Saddle emboli | PESI score | cTNI, ng/ml | SBP, mmHg | Heart rate, beats/min | Shock index | Miller score | Mean PAP, mmHg | Surgery (days before) | Complications |
|----|----------------|-----|---------------|------------|-------------|-----------|-----------------------|------------|-------------|----------------|----------------------|---------------|
| 1  | 65/M           | Moderate - | + 115 (4)    | 0.09       | 152        | 150       | 116                   | 63         | 0.77        | 0.42           | 15 12 30 NR          | NA            |
| 2  | 38/F           | Moderate - | 78 (2)       | 0.16       | 120        | 125       | 120                   | 97         | 1.0         | 0.78           | 17 4 20 20 C section (10) | NA            |
| 3  | 68/F           | Moderate - | - 108 (4)    | 0.06       | 144        | 169       | 118                   | 133        | 0.80        | 0.78           | 16 9 55 42         | NA            |
| 4  | 47/M           | Moderate - | + 87 (3)     | 0.15       | 132        | 188       | 90                    | 100        | 0.68        | 0.53           | 24 11 30 21 ORIF femur (21) | NA            |
| 5  | 72/F           | Moderate - | - 102 (3)    | 0.18       | 163        | 160       | 121                   | 80         | 0.74        | 0.47           | 17 6 29 19         | NA            |
| 6  | 56/F           | Moderate - | - 96 (3)     | NR         | 194        | 127       | 127                   | 80         | 0.65        | 0.62           | 15 5 60 46         | NA            |
| 7  | 58/F           | Moderate - | 108 (5)      | 0.05       | 181        | 162       | 120                   | 90         | 0.70        | 0.55           | 17 6 38 32         | NA            |
| 8  | 59/F           | Severe -   | 89 (3)       | 0.15       | 139        | 101       | 105                   | 88         | 0.75        | 0.87           | 15 8 65 61         | NA            |
| 9  | 25/F           | Severe +   | 45 (4)       | NR         | 127        | 131       | 86                    | 98         | 0.68        | 0.73           | 16 8 27 26 C section (25) | NA            |
| 10 | 59/M           | Severe +   | 119 (4)      | 0.11       | 96         | 149       | 96                    | 68         | 1.0         | 0.46           | 16 8 36 34         | NA            |
| 11 | 71/M           | Severe -   | 131 (5)      | 0.01       | 148        | 114       | 110                   | 62         | 0.74        | 0.53           | 18 10 35 30 Femoral embolectomy (2) | NA            |

* Present; – Absent; cTNI Cardiac troponin; F Female; M Male; NA Not applicable; NR Not reported; ORIF Open reduction internal fixation; PAP Pulmonary artery pressure; PESI Pulmonary embolism severity index; Post Postintervention; Pre Preintervention; Pt Patient; RVD Right ventricular dysfunction; SBP Systolic blood pressure

TABLE 2
Clinically massive pulmonary embolism cases

| Pt | Age, years/sex | Saddle emboli | PESI score | cTNI, ng/ml | SBP, mmHg | Heart rate, beats/min | Shock index | Miller score | Mean PAP, mmHg | Surgery (days before) | Complications |
|----|----------------|---------------|------------|-------------|-----------|-----------------------|------------|-------------|----------------|----------------------|---------------|
| 1  | 58/M           | + 168 (5)     | 0.16       | 81          | 144       | 130                   | 144        | 1.6         | 1.0           | 23 11 40 30         | Acetabular repair (14) Cardiac arrest-transient, retroperitoneal bleed, renal failure resolved |
| 2  | 41/F           | + 111 (4)     | 1.2        | 90          | 100       | 85                    | 60         | 0.94        | 0.6           | 20 14 33 25         | ORIF ankle (8) Transient hemoptysis |
| 3  | 50/M           | - 160 (5)     | 0.15       | 88          | 136       | 120                   | 90         | 1.36        | 0.66          | 16 6 50 26         | Retroperitoneal bleed, renal failure resolved |
| 4  | 77/F           | - 167 (5)     | 0.13       | 70          | 96        | 106                   | 115        | 1.5         | 1.2           | 25 15 18 18 NR 21   | CPR on arrival, death 24 h post |
| 5  | 25/M           | + 125 (4)     | 0.05       | 76          | 105       | 198                   | 110        | 2.6         | 1.0           | 23 12 19 19        | NA            |

* Present; – Absent; cTNI Cardiac troponin; CPR Cardiopulmonary resuscitation; F Female; M Male; NA Not applicable; NR Not reported; ORIF Open reduction internal fixation; PAP Pulmonary artery pressure; PESI Pulmonary embolism severity index; Post Postintervention; Pre Preintervention; Pt Patient; RVD Right ventricular dysfunction; SBP Systolic blood pressure

Approval for the present study was obtained from the institutional review board. Results are expressed as mean ± SD. Univariate analysis for continuous variables was performed using the independent t test for comparison between groups, while the paired t test was used for comparison within groups of pre- and postintervention parameters. Statistical significance was set at P<0.05. Statistical analysis was performed using SPSS version 14.0 (IBM Corporation, USA).

RESULTS

During the study period, 174 patients (22 of whom were inpatients at the time of diagnosis) were diagnosed with acute PE. Sixteen (4%) patients underwent pulmonary embolectomy using the Angiojet device in combination with r-tPA infused using the power-pulse-spray mode and formed the study group. The average age of the patients was 54±15.8 years, and nine were women. Two were inpatient cases (one medical, one nontrauma surgical) who were on prophylactic anti-coagulation. The remainder presented to the emergency department acutely. Two patients were transferred as possible acute myocardial infarction cases. Risk factors for venous thromboembolic disease included the following: obesity (body mass index of greater than 30 kg/m2) (n=7); chronic obstructive pulmonary disease (COPD) (n=5); relative immobility (n=4); previous deep venous thrombosis (n=3); coronary artery disease (n=2); previous cancer (n=1); and renal insufficiency (n=1). All patients were treated with heparin once the diagnosis was confirmed, and all underwent computed tomography (CT) angiography to confirm the diagnosis before the interventional team was consulted. All patients underwent transthoracic echocardiography, which in seven cases was performed before CT angiography.

Five patients with clinically massive PE (two in cardiogenic shock) and three of 11 with clinically submassive PE had severe RVD (Tables 1 and 2). Of the eight patients with submassive PE without...
TABLE 3  
Comparison between submassive and massive pulmonary embolism (PE) groups and impact of intervention

|                         | Submassive | Massive |
|-------------------------|------------|---------|
| Saddle emboli, n        | 4          | 3       |
| Age, years              | 54.6±16.4  | 54.2±13.5|
| Pulmonary embolism severity index | 105.7±32  | 125.3±33.0|
| Cardiac troponin I, ng/mL| 0.08±0.06  | 0.58±0.5* |
| Time to catheterization, h | 21±26     | 8±10*   |
| Heart rate, beats/min, pre | 109±13    | 109±21* |
| Heart rate, beats/min, post | 91±27     | 102±38  |
| Systolic blood pressure, mmHg, pre | 134±18    | 838±8*  |
| Systolic blood pressure, mmHg, post | 147±23    | 120±24† |
| Shock index, pre        | 0.82±0.1  | 1.3±0.3* |
| Shock index, post       | 0.63±0.2† | 0.80±0.3†|
| Miller score, pre       | 17.3±2.5  | 18.3±4.4|
| Miller score, post      | 8.8±3†    | 9.5±4.4†|
| Mean pulmonary artery pressure, mmHg, pre | 31±10.1   | 40±6    |
| Mean pulmonary artery pressure, mmHg, post | 268±6†    | 286±6†  |
| Intensive care unit length of stay, days | 1.5±0.8   | 12.8±14*|
| Hospital length of stay, days | 3±1.6     | 17±17†* |
| New York Heart Association class at follow-up | 1.6±0.8   | 2.0±1.0 |

Data presented as mean ± SD unless otherwise indicated. *P<0.05 between groups; †P<0.05 preintervention (pre) versus postintervention (post) within groups

The majority of complications occurred in the massive PE group (Tables 1 and 2). One patient in the massive PE group experienced a significant bradycardic episode responding to intubation and atropine, and required ventilatory support for six days. One patient in the submassive PE group required intubation postprocedure, but was extubated within 24 h.

The length of follow-up averaged 17.3±7.8 months. Of the patients in the submassive PE group, seven were New York Heart Association (NYHA) class I, three were class 2 and one class 3. Of the four survivors in the massive PE group, two were NYHA class 1, one class 2 and one class 3. All patients who were NYHA class 2 were obese (body mass index of greater than 30 kg/m²), one of whom underwent previous coronary artery stenting, and both who were class 3 had pre-event COPD. Initial follow-up echocardiograms were performed between one and three months in all patients. All patients in the submassive PE group had normal or only mild RVD. Two patients in the massive PE group showed mild RVD on echocardiogram and had evidence of mild cor pulmonale. However, both patients had pre-event comorbidities including COPD and morbid obesity, and one patient had a nonocclusive caval thrombus trapped in the vena cava filter.

DISCUSSION

PE has been described as the most common, preventable cause of death in hospitalized patients, responsible for up to 10% of all in-hospital and 0.8% of postoperative deaths (1,2). Three-month mortality rates of as high as 18% have been reported (4). Approximately 4% of survivors develop significant chronic pulmonary hypertension within two years (17). Nationally, the incidence of fatal PE appears to have decreased, possibly due to increased awareness of the importance of prophylaxis and early diagnosis. Some institutions have documented an overall increase in the incidence of PE, which is probably due to increased awareness and diagnosis (1,18).

Acute outcomes are linked to comorbidities, the presence of shock and degree of right heart dysfunction (3,4,14,19-21). The simplest indicator of determining immediate prognosis is the presence of hemodynamic compromise. Patients who present with hemodynamic compromise (ie, clinically massive PE) have acute mortality rates ranging between 25% and 60%, and three-month mortality rates of approximately 50% among those who survive the initial episode (4,6,10,22,23). Patients who are hemodynamically stable throughout their course have much lower mortality rates, ranging from 1% to 15% if all causes are included (24,25).

Prognosis in hemodynamically stable (ie, clinically submassive PE) is linked to the degree of right heart dysfunction (24). Patients with marked RVD have mortality rates that range from 10% to 17% in the acute setting (4,10,24). Up to 40% of patients with submassive PE will have echocardiographic evidence of RVD, with increased mortality (26). In hemodynamically stable patients, evidence of right heart dysfunction based on CT or echocardiographic criteria is associated with
a 2.4 times increased risk of mortality (12). In the absence of echocardiographic data, electrocardiogram demonstrating right heart strain, CT angiogram demonstrating a right ventricular diameter of 90% or greater of left ventricular diameter, and/or elevated biomarkers (brain natriuretic peptide-BNP and/or cTnI independently have been associated with increased death and nonfatal complications) (3,12,23,25,26). Bova et al (25) found that among hemodynamically stable patients, the three-month mortality among those with a cTnI level of 0.07 ng/mL or greater was 21% versus 3% if it was lower than 0.07 ng/mL (P<0.01).

Relying purely on evidence of RVD as a measure of severity is not uniformly sensitive (27). It has been recognized that even in hemodynamically stable patients, those with anxiety, ‘marked’ dyspnea, who ‘appear ill’ and/or have low oxygen saturation are at increased risk of decompensation, death and nonfatal complications (3). A variety of clinical scores have been derived to help determine prognosis at presentation and to determine how aggressive intervention should be. These scores have considerable overlap and variation, and appear to be most useful when integrated into a standard plan of management (13,20). The PESI combines demographics (age and sex), comorbid conditions (cancer, heart failure and/or chronic lung disease) with clinical findings (heart rate, systolic blood pressure, respiratory rate, temperature, mental status and oxygen saturation) into five classes. Class I and II (low risk) have 1.1% and 3.1% mortality risk, respectively, class III (intermediate) 6.5% mortality risk, and class IV and V (high risk) 10.4% and 24.5% mortality risk, respectively (14).

The goals of treatment are based on immediately reducing right ventricular outflow obstruction while supporting cardiorespiratory status and, hopefully, preventing long-term sequelae of persistent right heart failure and pulmonary hypertension. The decision of how best to intervene depends on an assessment of hemodynamic stability, RVD, a determination of how rapid the response must be and what the risks of intervention are, given the specific situation.

The mainstay of treatment in hemodynamically stable patients is therapeutic anticoagulation. Park et al (1) reviewed the nationwide incidence of PE between 1998 and 2005, using the Nationwide Inpatient Sample, obtained from the Healthcare Cost and Utilization Project of the Agency for Health Care Research and Quality. Compared with medical patients, surgical patients managed by anticoagulation experienced an overall greater mortality (10.7% versus 7.1%), major bleeding complications (1.7% versus 0.4%) and incidence of heparin-induced thrombocytopenia (2% versus 0.7%). The authors hypothesized that these differences could be explained by greater morbidity, risk of bleeding and hesitation to use aggressive anticoagulation among the surgical patients.

Long-term outcomes are also a concern. While the reported incidence of chronic pulmonary hypertension following PE managed by heparin or its equivalent alone is generally believed to be less than 5%, the incidence of pulmonary hypertension when measured using catheter or echocardiogram ranges from 11% to 69% (12,28). This raises the issue of defining the degree and significance of RVD at presentation and the role of being more aggressive initially in reducing and preventing distal thrombus burden. Using echocardiograms, Kline et al (28) compared the six-month outcomes of patients with PE managed with heparin alone or heparin plus rt-PA. The authors concluded that more than 90% of patients who presented with submassive PE and were managed by heparin alone experienced resolution of right ventricular dilatation and hypokinesis at six-month follow-up. Despite this, one-half had unchanged or worse right ventricular systolic pressure often associated with significant exercise intolerance or dyspnea at rest. They hypothesized that persistent elevation of right ventricular systolic pressure is maladaptive, particularly when associated with tricuspid regurgitation, leading to a complex of right ventricular damage, pulmonary vasospasm and inflammation related to erythrocyte hemolysis.

Thus, even hemodynamically stable patients with minimal right heart dysfunction are at some risk of early and late-term complications – a risk that increases as the RVD worsens (10,12,25). This has led to increasing emphasis on combining anticoagulation with lytic therapy, particularly in patients with moderate to severe RVD where whose clinical picture suggests that they may be nearing decompensation (3). The evidence that the benefit of systemic lytic therapy in submassive PE outweighs the risk is not entirely clear, partly because of the wide mix of cases in various reports. It is accepted that systemic rt-PA (or other lytic agents) results in earlier resolution of right heart dysfunction when compared with heparin or its equivalent alone. However, it is not entirely clear whether this results in improved survival (22,29). The risk of fatal or clinically major bleeding with systemic lytic therapy has ranged from 0.6% to 29%, with higher rates generally being reported for streptokinase than rt-PA, and higher rates for longer infusions (24 h) versus shorter (2 h) (3,22,29). It should be noted that the data suggesting that rt-PA is safer than streptokinase are limited (30). The perceived risk of hemorrhage was one reason that in a recent survey of Pennsylvania hospitals (31) only 2.4% of patients with PE were treated with thrombolitics. Major bleeding occurred in 5.4% of patients treated with thrombolitics, and this was associated with 54% mortality. A recent study (32) proposed that reducing the dose of rt-PA to 50 mg in low body weight individuals may be as efficacious with reduced complication rate.

Several meta-analyses have been performed in an attempt to add clarity; however, they have resulted in somewhat contradictory conclusions. Most studies document a 1% to 2% incidence of intracranial hemorrhage (30). The meta-analysis by Agnelli et al (29) found an increased risk of bleeding in the heparin plus systemic lytic group (12.9% versus 8.6%), a reduction in death (4.6% versus 7.7%) and a reduction in recurrent PE (6.6% versus 10.9%). Overall, the combined treatment group had a significant reduction in the combined end points of death and/or recurrent PE compared with heparin alone (10.4% versus 17.3%; P=0.03). The study by Tardy et al (27) focused on patients who were clinically submassive and in whom the lytic therapy was rt-PA. They found no overall difference in death and/or recurrent PE (3.5% with rt-PA versus 4.6% without), nor major bleeding episodes (4.9% with rt-PA versus 4.6% without). However, they believed that patients with RVD and elevated levels of cardiac biomarkers were a subgroup that probably would benefit more from systemic lytic therapy. Wan et al (33) also found minimal differences when all patients were considered. The addition of systemic lytic therapy was associated with a trend toward reduced recurrent PE (2.7% versus 4.3%), all-cause mortality (4.3% versus 5.9%) and an increase in major bleeding (9.1% versus 6.1%). One possible confounding variable is that women may experience an increased risk of bleeding with diminished benefit (34).

The risk/benefit ratio of adding systemic lytic therapy tends to shift toward favouring more aggressive approaches the ‘sicker’ the patient is. When Wan et al (33) considered studies that included patients who were hemodynamically unstable, the addition of lytic therapy was associated with a marked reduction in death (6.2% versus 12.7%), a significant reduction in death and/or recurrent PE (9.4% versus 19%) and a significant increase in major bleeding (21.9% versus 11.9%). The MANagement strategies and Prognosis of Pulmonary Embolism-3 (MAPPET-3) study (35) – a prospective randomized trial of heparin plus rt-PA versus heparin plus placebo alone in clinical submassive PE cases but who had pulmonary hypertension and RVD – found low death rates in both groups (3.4% in treatment versus 2.2% in the control groups; P=0.73) but a marked reduction in the need to escalate therapy (defined as need for intubation, surgical or catheter embolectomy or late systemic thrombolysis and/or vasopressors) (11%) in treatment versus 25% in the control groups (P=0.005)). The same authors previously published data demonstrating a nearly 50% reduction in mortality rates in the subset of patients with impending right heart failure (36).

Thus, the risks/benefits of systemic lytic therapy are not entirely clear. The ACCP practice guidelines state the following: systemic lytic therapy (intravenous rt-PA 100 mg over 2 h) be administered in patients with hemodynamic compromise (Grade of recommendation 1B); systemic lytic therapy should be considered in selected patients who are...
hemodynamically stable but are judged to be high risk and have low risk of bleeding (Grade of recommendation 2B) (3). There is evidence that systemic lytic therapy is associated with a much more rapid resolution of right heart strain and, possibly, a reduction in late right heart dysfunction.

The ACCP practice guidelines do not recommend surgical or catheter embolectomy as initial management for PE, except in the setting of contraindications or such severe clinical compromise that there is not sufficient time for systemic therapy (Grade of recommendation 2C) (3).
Contraindications to systemic lysis include trauma or surgery within two weeks, active source or bleeding, endocarditis or intracranial disease (3,37).

Small case reports and series have reported successful surgical embolectomy under emergent circumstances. Probably the largest series was reported by Leacche et al (7). During a four-year period, 47 patients underwent embolectomy using cardiopulmonary bypass, with normothermia and without cardioplicecar arrest. The procedures were performed to treat large central thrombus events; 26% of the patients were in cardiogenic shock while 11% were in arrest. Indications were contraindications to thrombolysis (45%), failure of medical management (10%) and RVD (32%). There were only three (6%) operative deaths and the three-year survival rate was 86%; the majority of the late deaths were secondary to cancer. This report demonstrated that if there is a system in place, surgical embolectomy can be very effective for central thrombi. However, there are some patients who may not tolerate the level of systemic heparinization required for cardiopulmonary bypass. In addition, surgical thrombectomy is effective only for large central thrombi.

Catheter embolectomy is attractive because it can limit the systemic impact of thrombolitics, can extract central and peripheral emboli, and can be performed in a rapid and relatively easy manner. Furthermore, it can be performed on patients undergoing cardiopulmonary resuscitation (Figure 1) (38). A variety of techniques have been described, including simple fragmentation. The concern of simply fragmenting thrombus is that while reducing central flow restriction, creating multiple small peripheral emboli may prevent reduction of pulmonary hypertension and, possibly, lead to either a failure of acute and/or late reduction in pulmonary hypertension and right heart failure (39). Catheters that permit thrombus disruption and aspiration appear to be associated with markedly improved results, even if used without local delivery of thrombolytics (6). However, using the catheter to core through thrombus permits locally delivered thrombolytics to work on deeper thrombus layers and potentially have an effect on more distal small-vessel occlusions, resulting in improved acute success, with some evidence that late-term complications are reduced (6,8,10,39). Furthermore, the greater surface area of exposure combined with direct injection allows for much smaller doses to be administered, with greater local and markedly reduced systemic impact (8). Kucher (11) reviewed more than 300 reported cases. Clinical success rates were the lowest with fragmentation techniques; however, all techniques had improved success when combined with local thrombolytic injection. Kuo et al (6) presented a review of 594 patients from 35 studies (six of which were prospective) of catheter-directed thrombectomy. They found that the pooled frequency of success was significantly higher in studies in which at least 80% of subjects received local thrombolytic therapy during the procedure. The overall clinical success rate was 86.5%, with a major complication rate of 2.4% and a minor complication rate of 7.9%. While most cases demonstrate almost immediate resolution of right heart strain and pulmonary hypertension, some may take 24 h to see the clinical benefit; occasionally leaving catheters in place for ongoing thrombolysis has been done.

There are several catheters available that enable combinations of mechanical lysis, aspiration and local thrombolytic injection. Despite the success rate, particularly in unstable patients, it should be noted that use of these devices for pulmonary embolectomy are considered to be off label (6). Specific local complications can include hemoptysis, pulmonary artery perforation, bradycardia and hemoglobinuria. These complications, particularly of bradycardia and hemoglobinuria, appear to be more frequent with the AngioJet than other devices (6). While often attributed to thrombus release, it has been the concern that concurrent with the increased rate of bradycardia may not be immediately apparent, particularly in patients with clinical submassive PE, while the most discernable improvement – albeit accompanied by increased complication rate – was seen in the clinically massive PE group. By keeping run times short, the incidence of bradycardia was low. At follow-up, patients did not appear to experience significant RVD.

Whether at least some of these patients may have benefited from systemic rather than local therapy is not clear. It is also not clear whether patients not referred for embolectomy could have benefited from catheter-based or systemic lysis. It has been suggested that the management of PE be individually determined by each institution, following trauma principles in which selected centers develop a protocol (whether it be systemic medical therapy or embolectomy by surgical or percutaneous means) and act as central referral centers for high-risk patients (6). Our institution has an apparent bias against systemic thrombolytic therapy, which needs to be systematically re-evaluated; this bias clearly affects how we can interpret the results as a system. Thus, we cannot prove that our approach is unequivocally superior to systemic thrombolysis, but it appears to be effective, particularly in patients with massive PE, and short outcomes indicate a durable result. While in selected cases, influenced by our selection bias, patients with clinical submassive PE appeared to experience some benefit; however, it is not entirely clear whether systemic lytic therapy could not have achieved the same result. As a result, we are in the process of redefining our indications for choosing between systemic anticoagulation and percutaneous thrombectomy, and revisiting whether our perceived contraindications to systemic thrombolysis are justified.

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