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Catheter-Based Therapies for Massive Pulmonary Embolism

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Abstract Massive pulmonary embolism carries a high mortality rate as a result of right ventricular failure. In addition to anticoagulation, systemic thrombolysis is the standard first line of therapy for patients with life-threatening massive pulmonary embolism. Surgical embolectomy is often considered in patients with contraindications to receiving systemic thrombolysis or when thrombolysis has failed. Surgical embolectomy is not without inherent risk and limitations. Although there is a paucity of large clinical trials, available data suggests catheter-based treatment of massive pulmonary embolism restores hemodynamic stability and thus is an alternative to surgical therapy. (Prog Cardiovasc Dis 2010;52:429-437)

Keywords: Massive pulmonary embolism; Percutaneous therapy; Thrombectomy

The clinical spectrum of acute pulmonary embolism (PE) can be broadly divided into 3 general groups. In the first group, the PE is relatively well tolerated without right ventricular (RV) strain or hemodynamic instability. These patients have a favorable prognosis when treated with systemic anticoagulation.[1] In the middle of the spectrum are patients in whom thrombotic obstruction of the pulmonary arterial bed acutely elevates mean pulmonary arterial pressure and RV afterload causing ventricular strain without systemic hypotension. Patients with such submassive PE will thus have echocardiographic signs of RV dilatation and hypokinesis, increased RV to left ventricular diameter ratio on computed tomography imaging, or elevated levels of cardiac biomarkers. Right ventricular strain is present in as many as 30% to 50% of patients presenting with PE. It is generally accepted that this group has a higher mortality rate, although some controversy exists due to heterogenous methods used to assess RV dysfunction.[2,3] In the third group, the magnitude of pulmonary artery (PA) obstruction leads to acute failure of the right ventricle and hemodynamic instability. Massive PE is thus defined by the presence of systemic hypotension (systolic blood pressure below 90 mm Hg), a decrease in systolic blood pressure by more than 40 mm Hg for at least 15 minutes, syncope, or cardiac arrest. This group accounts for 4.5% of all patients with PE and has a 90-day mortality rates as high as 50%.[4] Although anticoagulation alone is sufficient in the first group, therapeutic interventions in patients with massive PE must aim at immediate reversal of RV dysfunction. Despite paucity of well-designed trials, systemic thrombolysis remains an accepted standard of care for patients with life-threatening, massive PE. Unfortunately, as many as half of patients presenting with massive PE may have contraindication to systemic thrombolysis.[5] The delayed onset of lytic effect and its limited efficacy in the presence of organized venous thrombus can be costly. Mechanical relief of PA obstruction by surgical embolectomy or percutaneous thrombectomy can result in immediate improvement in hemodynamic status. Catheter-based therapies have been used with variable success for more than 4 decades.[6] Recent technological advances sparked renewed interest in minimally invasive treatment of these critically ill patients. There is growing evidence that such strategies do indeed impact clinical outcomes in patients with massive PE.[7]

Patients with submassive PE present a therapeutic challenge. Most of these patients can expect mortality rates below 5% when treated with systemic anticoagulation and

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Abbreviations and Acronyms
FDA = Food and Drug Administration
PA = pulmonary artery
PE = pulmonary embolism
RV= right ventricular

supportive care.[3,8] Systemic thrombolysis has not been shown to reduce mortality in this patient group and is associated with 20% rate of major bleeding complications.[9] Furthermore, the right ventricle recovers in most patients as the thrombotic burden diminishes with anticoagulation and intrinsic fibrinolytic lytic activity. Approximately 10% of patients with submassive PE can progress to hemodynamically significant RV failure.[8] There is a growing interest in treating this group of patients with catheter-based therapies to reduce RV strain. In the absence of randomized trials, it is not clear whether all patients in this intermediate risk group would benefit from invasive treatment strategies.

Mechanical therapies for massive PE

Surgical embolectomy

Surgical embolectomy offers the advantage of complete removal of proximal thrombus and immediate relief of RV strain (Fig 1). Surgical embolectomy was first proposed by Trendelenburg[10] in 1908 and successfully performed by Kirschner[11] in 1924. After decades of clinical failures, it was reintroduced in the 1960s[12,13] but continued to be associated with mortality rates exceeding 30%.[14] Recently, better patient selection and advances in surgical and anesthesiology techniques led to improved clinical outcomes and sparked renewed interest in this therapy.[15] Nevertheless, a systemic review suggests that mortality rates in the last 2 decades still approached 20%.[14] In critically ill patients, surgical embolectomy can be lifesaving but is limited to centers with surgical expertise and a system for rapid mobilization of the required resources. Moreover, the clinical factors predisposing patients to massive PE and hemodynamic compromise also increase the risk associated with surgical therapy.

Percutaneous therapies

Catheter-based techniques offer an immediate and minimally invasive way of relieving some of the hemodynamic burden of PE without exposure to thrombolysis or cardiopulmonary bypass. Interventional techniques frequently combine mechanical and pharmacologic means of thrombus reduction. Balloon angioplasty, mechanical fragmentation, and various suction thrombectomy techniques have been described and used in combination, rendering the frequently used term “pulmonary thrombectomy” somewhat inaccurate. Even stenting of the PA has been attempted.[16,17] Adjunctive catheter-directed thrombolysis delivers a high concentration of thrombolytics directly to the site of thrombotic obstruction

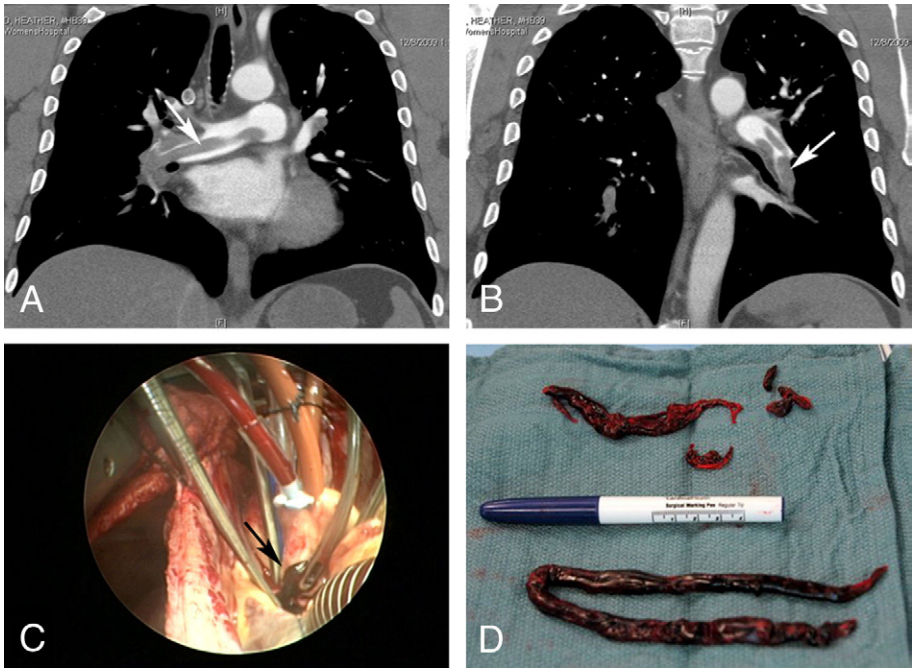

Fig 1. A massive PE (white arrows) involving proximal right and left PAs (A and B). Removal of proximal thrombus (black arrow) from the PA under direct visualization (C). A massive thrombotic cast of femoral vein removed intact from the PA (D). Such large and organized thrombus poses a challenge to percutaneous device.

allowing a reduction in dose and increased efficacy with lower risk of hemorrhagic complications.

The only percutaneous device designed and Food and Drug Administration (FDA) approved specifically for treatment of PE was the Greenfield catheter designed in the 1960s.[6] Current catheter-based strategies use devices designed for thrombus removal from hemodialysis fistulas, coronary, and peripheral arteries or clever adaptations of catheters designed for other uses. There are, understandably, no well-conducted randomized trials testing efficacy of catheter-based therapies in patients with life-threatening massive PE. A recent analysis of multiple small, single-center series suggests that percutaneous strategies can be performed safely and are potentially lifesaving in many patients.[7]

The difficulty in assessing the efficacy of catheter-based therapies for massive PE stems from the paucity of well-designed studies. A recent review of 35 case series published during the last 28 years included an aggregate of only 594 patients.[7] A trial comparing catheter-based approaches to systemic thrombolysis or surgical embolectomy is not likely to be ever conducted. The available clinical experience describes an often heterogenous patient population with variable definitions of "massive PE," arterial hypotension, PA occlusion more than 50%, mean PA pressure greater than 25 mm Hg, or shock index more than 1 (ratio of heart rate to systolic blood pressure). Angiographic definition of massive PE, initially described by Miller et al[18] (Fig 2), has been used in many series. The definition of clinical success has been equally eclectic and variably defined as reduction in thrombus burden, freedom from complications, improvement in Miller score, or acute restoration of hemodynamic stability.

The challenges of catheter-based PE interventions are numerous. The large caliber of the proximal pulmonary arteries and large thrombus burden limit the effectiveness of devices designed for smaller caliber vessels. The embolus is often at least partly organized after developing in the peripheral veins and is resistant to fragmentation or dispersal with devices designed to treat acute thrombus. Most devices are designed for vessels less than 10 mm in diameter, whereas the proximal pulmonary arteries can be 3 times that size. These devices tend to move through the artery along a path of least resistance, pushed aside by organized thrombus. Catheter-based therapy, however, can be lifesaving without achieving complete thrombus removal. Even partial improvement in flow can restore sufficient cardiac output and reverse circulatory collapse. The goal of this therapy may be a clinically relevant

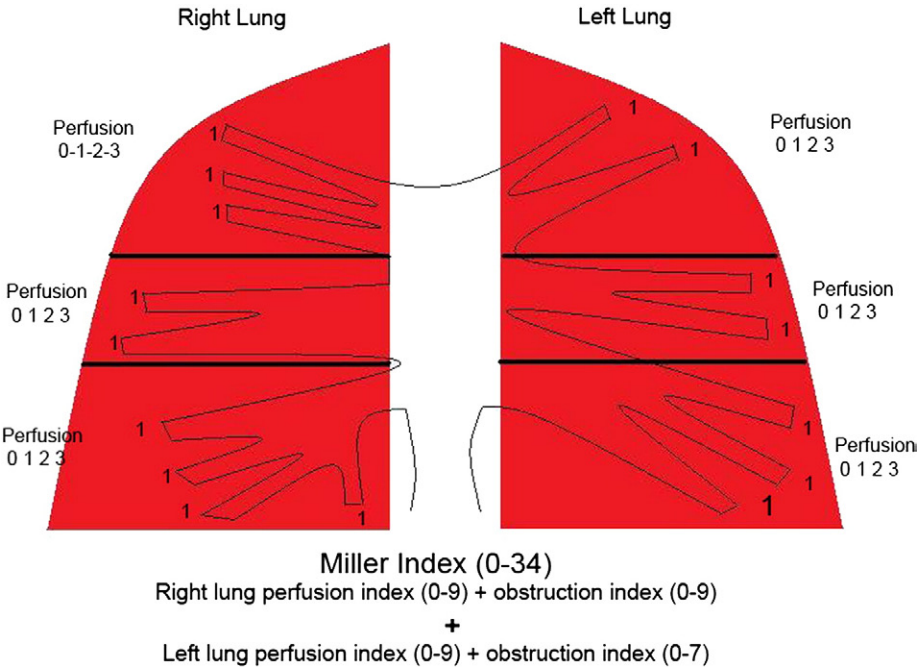

Fig 2. Angiographic quantification of PE severity. Miller score is calculated as the sum of obstruction and perfusion indexes, ranging from 0 (best) to 34 (worst) and can be compared before and after the procedure as a measure of angiographic success.[12] A Miller score of 17 or more indicates a greater than 50% obstruction of pulmonary vascular bed and forms an angiographic definition of a massive PE. Calculating the Miller obstruction index ranging from 0 to 16: 9 major segmental branches in the right PA (3 in the upper lobe, 2 in the middle lobe, 4 in the lower lobe) and 7 major branches in the left PA (2 in the upper lobe, 2 in the lingual, 3 in the lower lobe). The presence of filling defect in any of these branches is scored 1 point. The perfusion index is scored by dividing each lung into 3 zones (upper, middle, and lower), and the flow into each zone is characterized as absent (3 points), severely reduced (2 points), mildly reduced (1 point), or normal (0 points.)

reduction in thrombus burden rather than complete thrombus removal.

Percutaneous devices for pulmonary thrombectomy

Greenfield embolectomy catheter

The original device was developed in 1960s and consisted of a metal suction cup attached to a straight catheter.[6,19] It remains today as the only device ever approved by the FDA for percutaneous pulmonary embolectomy. The catheter was introduced through a surgical cut-down in the femoral or jugular vein and a large 24F introducer sheath. The catheter was advanced into the PA and suction applied to the cup through a syringe. The experience of Greenfield et al[20] in 46 patients with massive PE showed that percutaneous embolectomy resulted in a 30-day survival rate of 70%, an average reduction in mean PA pressure of 8 mm Hg and a significant increase in cardiac output from 2.59 to 4.47 L/min ($P = .003$). Access site complications and pulmonary infarct were most common complications, seen in 15% and 11% of patients, respectively. Some small series mirrored these results.[21] Subsequent modifications introduced a more steerable, 10F catheter with a 5- to 7-mm plastic suction cup, but the continued need for surgical venotomy and a large introducer sheath as well as complex delivery and operation kept it from gaining widespread clinical use.

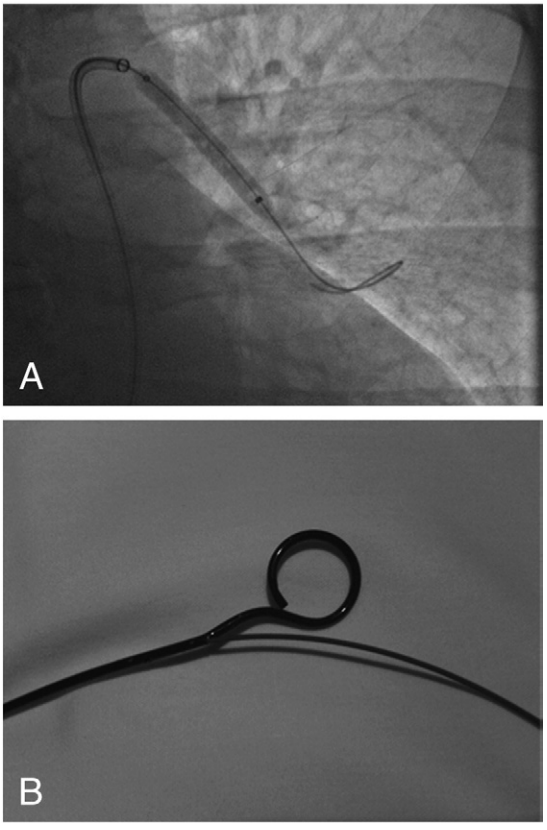

Fig 3. Mechanical fragmentation of proximal PE with a large angioplasty balloon (A) maybe helpful in a fresh thrombus but often fails with a more organized, rubbery clot. A stiff pigtail catheter can be rotated over a guide wire to fragment PA thrombus (B).

Catheter-mediated fragmentation

The notion that fragmenting a large, proximal occlusive thrombus and redistributing the obstruction into multiple smaller downstream branches comes from the concept that the volume of the peripheral branches is much larger than that of the proximal PA.[22] Thus, dispersing the clot into multiple branches will increase pulmonary blood flow. Fragmenting the thrombus will also increase the area exposed to intrinsic or pharmacologic thrombolysis. A theoretical counterargument raises a concern that fragmenting a 10-mm^3 sphere into 1000 spheres of 1 mm^3 would actually increase an obstruction of 1 cm^2 into one of 10 cm^2.[23] This could potentially increase the afterload imposed on the right ventricle. Nevertheless, clot fragmentation has been attempted with reasonable success using standard pigtail catheters, wires, large angioplasty balloons, and manually rotatable pigtail catheters (Fig 3).

Brady at al[24] advanced, rotated, and withdrew standard coronary catheters to fragment and disperse proximal thrombus in 3 patients in shock and showed hemodynamic and clinical improvement. Schmitz-Rode and colleagues[25] designed a modified high-torque pigtail catheter with an oval port on the outer curvature of the loop allowing the catheter to be advanced and manually rotated over a wire while retaining its shape. In a larger series of 20 patients with massive PE, this pigtail catheter was effective in reducing the degree of obstruction and achieving hemodynamic improvement.[26,27] Eight of these patients received concomitant thrombolytic therapy, and the short-term mortality rate was 20%. The catheter was less effective in main PA where the size mismatch between the vessel, thrombus, and smaller pigtail loop did not allow adequate fragmentation. A standard pigtail catheter can also rotate freely in the PA if it is loaded onto the guide wire through a proximal side hole (Fig 3B). A combined approach using mechanical fragmentation and local and systemic thrombolysis in 25 hemodynamically unstable patients was described by Tajima et al.[28] All patients survived with immediate reduction in PA pressures and improvement of hemodynamic parameters and pulmonary perfusion score. Cumulative experience from small series suggests that catheter fragmentation can improve hemodynamic parameters in up to 95% of patients when used in conjunction with local infusion of thrombolytics.[29]

Fragmentation of the thrombus can be complicated by embolization of the debris into the adjacent nonobstructed branches, further increasing the RV afterload.[30] Adjunctive modalities such as catheter aspiration or rheolytic thrombectomy must be available to deal with such complications. Nakazawa et al[31] investigated the relationship between PA pressure and branch vessel embolization during catheter fragmentation. In 25 hemodynamically unstable patients, fragmentation with a pigtail catheter alone resulted in decrease in mean PA pressure from 34.2 to 30.8 mm Hg ($P < .05$), whereas subsequent infusion of local thrombolytic and manual aspiration with an 8F coronary guide further decreased mean PA pressure to 24 mm Hg ($P < .01$). Distal branch embolization, noted in 28% of the patients, caused an acute increase in mean PA pressure (from 34.1 to 37.9 mm Hg, $P < .05$). This acute decompensation was successfully managed by catheter aspiration with mean PA decrease to 25.7 mm Hg ($P < .05$). Interestingly, no clear clinical or angiographic characteristics could identify patients at risk of distal embolization.

Mechanical fragmentation with a pigtail catheter seems to be effective in reducing the severity of obstruction and improve hemodynamics with few periprocedural complications. The efficacy of this technique is further improved by concomitant use of local thrombolytic therapy.

Aspirex thrombectomy catheter (Straub Medical AG, Wangs, Switzerland). This 10F over-the-wire catheter is designed for thrombus removal in peripheral vessels larger than 8 mm in diameter. A spiral at the catheter's tip rotates at 40 000 rpm, fragments and aspirates particles at 180 mL/min. The catheter is advanced into the thrombus and gently withdrawn during aspiration. The strength of suction can be adjusted to avoid collapse and injury of the vessel around the catheter. A smaller Rotarex version of this device has been used in treatment of femoropopliteal arterial occlusions.[32] Intrapulmonary applications were first tested in animals by Kucher et al,[33] and subsequent clinical application was described in single case reports.[34,35] A retrospective series of 18 patients who underwent multimodality catheter-based treatment included 11 patients treated with pigtail catheter fragmentation and Aspirex thrombectomy. In this series, diagnosis of massive PE was based on angiographic criteria, and presence of RV strain with hemodynamic instability present in only 44% of patients. The Aspirex seemed safe with catheter-related blood loss of 101.6 ± 90.5 mL and no significant periprocedural complications. In the entire cohort, combined fragmentation and aspiration strategy led to statistically significant improvement in blood pressure and decrease in Miller score, mean PA pressure, and shock index.[36]

Amplatz catheter (Helix clot buster, eV3, Inc, Minneapolis, Minn). This 7F catheter has an impeller at its tip mounted on a drive shaft capable of generating 150 000 rpm. The vortex created by the spinning impeller pulls the thrombus into the impeller housing, macerates it, and expels it through side holes behind the impeller housing. The device does not have a central lumen for a guide wire and requires a larger 10F sheath for delivery to the PA. Clinical experience has been reported in a handful of patients with improvement in angiographic appearance of the pulmonary arteries but actual increase in pulmonary pressures in most patients.[37] A larger series of 9 patients reported promising angiographic improvement but only minor improvement in mean PA pressure.[38] In vitro studies of several thrombectomy catheters have shown higher distal embolization rates associated with the Amplatz catheter, possibly explaining why improvement in PA pressure lags behind angiographic scores of thrombus removal. As with other devices, adjunctive local thrombolysis resulted in significant improvement in clinical outcomes beyond those achieved with catheter therapy alone. The vigorous action of the impeller can be associated with variable degree of hemolysis. Hemoptysis has been described with the use of this device, although it is not clear whether it is due to reperfusion or vessel injury.[37]

Hydrolyser thrombectomy catheter (Cordis, Warren, NJ). Originally designed for management of dialysis access thrombosis, this 6F, 0.018-in guide wire compatible catheter uses the Venturi effect to create a vacuum when powered by a standard contrast injector filled with saline. A modified 7F PE-Hydrolyser with a fixed pigtail curve at the tip is available in Europe. The pigtail tip eliminates the guide wire compatibility but increases contact with the thrombus by keeping the device from being pushed into the path of least resistance around the organized thrombus. The catheter is advanced without a wire through a 9F sheath just proximal to the thrombus and the power injector activated to infuse saline at 4 mL/s at 750 psi to create vacuum and aspirate the thrombus. The device is advanced and withdrawn through the thrombus while being manually rotated. A maximum of 150 mL of saline can be infused with each run.

The experience with this device in the pulmonary arterial tree is not extensive.[39] In a small prospective series of 8 patients, the Hydrolyser catheter was used with concomitant systemic thrombolysis in 8 patients with PE, 5 of whom had hemodynamic compromise.[40] Treatment reduced thrombus burden by 50% on average (range, 30%-80%) and reduced mean PA pressure from 42.5 to 36.3 mm Hg (P = not significant) but had no effect on PA pressure in 5 patients. Interestingly, there was no correlation between the amount of angiographic and clinical improvement. Three of the 4 patients who had follow-up angiography continued to have pulmonary hypertension 3 months later. A larger series of 11 patients with massive PE defined by

angiographic criteria underwent Hydrolyser therapy with adjunctive urokinase used in 4 patients.[41] The estimated amount of thrombus removal reached 74% with a decrease in mean PA pressure from 45.4 ± 14.2 to 29.5 ± 13.6 mm Hg ($P < .0001$). Treatment was complicated in 1 case by self-limited hemoptysis.

Rheolytic thrombectomy with the Angiojet Xpeedior thrombectomy catheter (Medrad Interventional/Posis, Warrandale, Pa). This 6F device uses the Bernoulli principle to create a vacuum effect in a low-pressure zone behind a series of saline jets emanating from the tip of the catheter. The jets disrupt and aspirate thrombus into the catheter. The direction of the jets can be reversed with saline substituted for a thrombolytic agent, thus switching from aspiration to spraying the thrombus with a lytic agent. This "pulse spray" modality reduces thrombus volume and makes subsequent aspiration thrombectomy more effective (Fig 4). In patients with contraindication to even a small amount of thrombolytic, the device can be used as stand-alone aspiration thrombectomy catheter. The device has been widely used in treatment of acute thrombus in coronary and peripheral arteries as well as deep vein thrombosis. The 6F Xpeedior has been used in vessels greater than 6 mm in diameter, whereas smaller versions of this device are suitable for branch vessel thrombectomy.

Koning et al[42] first described rheolytic thrombectomy in 2 patients with submassive PE, and their report was followed by several small series describing this technique with and without adjunctive local thrombolysis.[43-49] In early experience with this device, clinical success, defined as immediate postprocedural hemodynamic improvement, was achieved in 75% of cases when used alone and in 87% of patients when combined with intrapulmonary thrombolytic infusion.[29] The familiarity with this device spurred wider clinical use, and most recent case series report on outcomes in a total of 120 patients with PE of variable severity.[43,44,49,50] The primary focus of these studies was angiographic improvement, and statistically significant decrease in Miller score was noted in more than 90% of cases. Fewer than half of these patients presented with hemodynamic compromise, and most procedures were performed without adjunctive local thrombolysis. Hemodynamic end points were not uniformly evaluated, but when recorded, PA pressures showed statistically significant improvement.[49] Overall, in-hospital mortality rates ranged from 7% to 21%, predominantly effecting patients presenting in shock. Bleeding and severe intraprocedural bradycardia requiring temporary pacing were the most common complications, the latter noted in 7% to 64% of patients.

Ultrasound-assisted catheter-directed thrombolysis

Ultrasound energy can been used to break up thrombus or, at lower intensity, dissociate fibrin strands without causing fragmentation.[51] The latter approach combined with simultaneous local infusion of a lytic

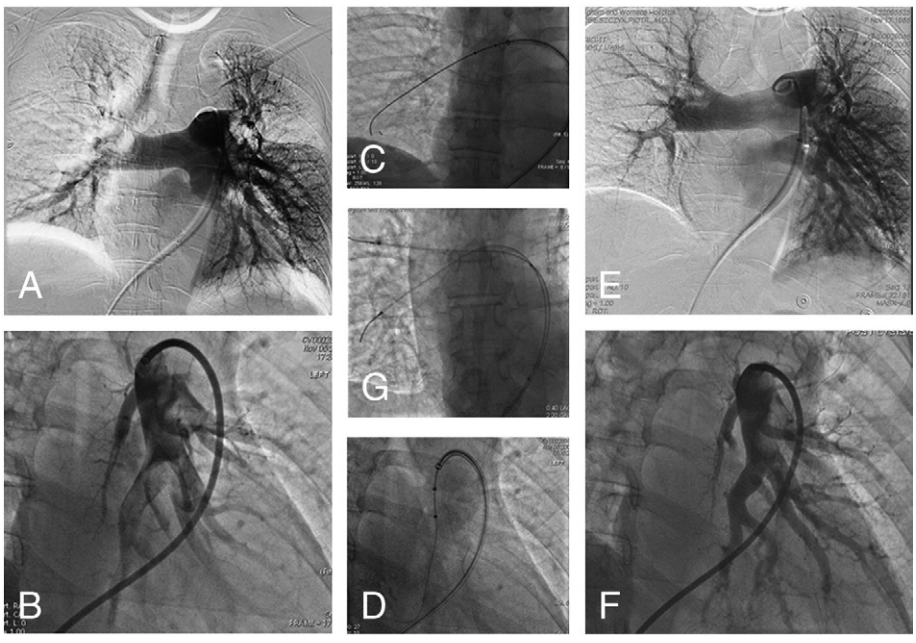

Fig 4. Rheolytic pulse-spray thrombectomy using an Angiojet catheter. Digital subtraction angiography of a massive, bilateral PE (A) and selective cine angiogram of the left PA (B). A 6F Xpeedior Angiojet catheter in the right and left main PA (C and D). Note the small caliber of the device in a large PA. Pulmonary angiography after pulse-spray thrombectomy of right and left main PA (E and F). Infusion catheters placed in the upper and middle branches of the right PA for adjunctive catheter-directed thrombolysis (G).

agent can enhance drug penetration into the thrombus and augment thrombolysis at lower doses and shorter infusion.[52,53] This can lead to increased success and lower rate hemorrhagic complications. The EkoSonic Endovascular System (EKOS, Bothell, Wash) simultaneously delivers low-intensity, high-energy ultrasound to loosen the clots composition and a thrombolytic agent to penetrate deeply into the thrombus. It has the potential to penetrate a more organized clot embolized from peripheral veins. The 5.2F device, with an ultrasound emitting segment ranging in length from 6 to 50 cm, can be delivered through a 6F sheath over on 0.035-in guide wire across the thrombosed vessel. The central wire contains a series of transducers 10 mm apart and emitting ultrasound at 2.2 MHz. A separate lumen allows infusion of saline to cool the ultrasound filament. A third port allows infusion of the thrombolytic agent through multiple side holes along the segment emitting acoustic energy. Clinical experience in PE therapy has been limited so far. Chamsuddin and colleagues[54] described a series of 10 patients with PE and RV strain but unclear hemodynamic status. Treatment with the EKOS catheter and tPA infusion (mean dose, 0.88 mg/h) for a mean of 24 hours resulted in complete thrombus resolution in 76% of patients, near complete resolution in 18%, and partial resolution in 6% of cases. One patient developed access site hematoma, and nonlethal hemoptysis was observed in another patient. In a larger series of 25 patients, 11 patients underwent treatment with the EKOS catheter and tPA (mean dose of 0.86 ± 0.16 mg/h) for a mean of 17 hours. These patients were retrospectively compared with patients undergoing traditional catheter-directed thrombolysis with tPA (33%) or urokinase (67%). In all patients presented with a PE causing RV strain, hemodynamic instability was present in 20% of these patients.[55] Treatment with the EKOS device improved the Miller score from 18.65 ± 3.25 on presentation to 5.84 ± 1.57 after the procedure. Complete thrombus resolution was noted in all 9 of the surviving patients. In the catheter-directed thrombolysis group, infusion for a mean of 26.7 hours (mean tPA dose of 0.93 ± mg/h) decreased the Miller score from 17.29 ± 3.86 to 7.38 ± 2.26, and complete thrombus removal was noted in 50% of patients. This strategy seems to offer a reduction in the cumulative dose of thrombolytic agent and likely improves thrombus resolution. The duration of treatment makes it useful as an adjunctive therapy once hemodynamic stability has been restored in massive PE. Its use and efficacy in more stable patients with submassive PE is intriguing but untested.

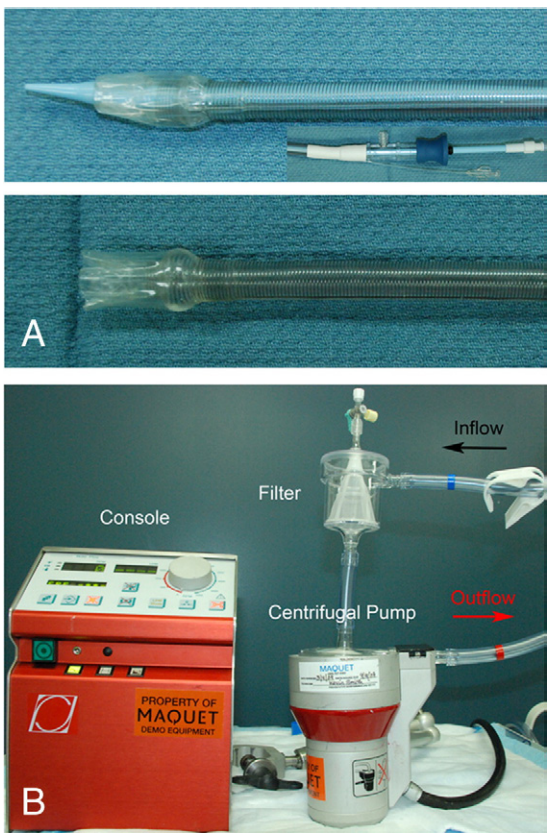

Fig 5. The AngioVac Aspiration System consists of a 25 Fr. cannula with a balloon-actuated, funnel-shaped tip (A), a centrifugal pump, a filter, and standard bypass circuit (B). Aspirated blood (inflow) is filtered and and returned to the circulation (outflow.)

AngioVac embolectomy catheter (Vortex Medical, Norwell, Mass) is a novel embolectomy system approved by the FDA for removal of undesirable intravascular material. The device resembles a cardiopulmonary bypass circuit and consists of an expandable, funnel-shaped cannula, a centrifugal pump, a filter, and standard bypass tubing (Fig 5).

The cannula is connected to a circuit with a pump that applies adjustable amount of suction at the catheter tip, filters blood and aspirated debris through a filter, and returns the blood via another sheath into the peripheral venous system. The current version of the cannula requires surgical venotomy for entry into the venous system but can then be delivered to the PA over a guide wire and obturator. The current clinical experience is limited, but the device potentially offers a percutaneous method of en-block removal of large thrombus without the need for cardiopulmonary bypass.

Complications of catheter-based therapies

Many complications of percutaneous interventions are shared across the devices. Injury to the PA and its branches can result in catastrophic complications, such as rupture of the PA,[48] pericardial tamponade, and life-

threatening hemoptysis. Such complications are fortunately rare and can be avoided by avoiding smaller subsegmental branches. Major bleeding complications can be as low as 2.4% in patients who do not receive concomitant systemic thrombolytics.[7] Worsening of hemodynamic parameters due to distal embolization can occur in some patients and is difficult to predict.[31,56] Some complications are device specific. Significant bradycardia can occur with Angiojet device and often requires intraprocedural temporary pacing. Interestingly, the Angiojet device has been associated with a higher rate of periprocedural complications compared with other devices. This may be related to a much wider use of this device but has led some authors to caution against its use in treatment of PE.[7]

Conclusions

Catheter-based therapy for massive PE can be a lifesaving therapy. There are no large-scale studies examining this treatment modality, but available data suggest that hemodynamic stability can be restored in 86.5% of patients. In the absence of systemic thrombolysis, the rate of major and minor periprocedural complications can be as low as 10%.[7] Our current experience comes mostly from single-center, retrospective series and selected patients, but high mortality and morbidity associated with massive PE make this therapy an attractive alternative. Unlike surgical embolectomy, percutaneous interventions can be instituted rapidly and widely because most hospitals are equipped with angiography suites. It may very well be that the future of PE care will mirror our current therapy for ST-segment elevation myocardial infarction: rapid triage and immediate catheter-based reperfusion therapy.

There is no single device or strategy that has been proven superior to others. Catheter fragmentation techniques seem to be most commonly used. Adjunctive local thrombolysis is used in about 60% of all PE interventions.[7] The experience of the last 3 decades suggests that reducing clot burden sufficiently to restore hemodynamic stability may be superior to prolonged procedures and attempts to achieve complete thrombus removal. Pulmonary artery pressures depend, in large part, on the patency of the distal peripheral vessels. Reduction in proximal obstruction without excessive distal embolization, followed by catheter-directed, local thrombolysis, may be the superior strategy.

Although patients with PE and hemodynamic compromise are logical candidates for aggressive intervention, the best treatment strategy for patients with evidence of RV strain without hypotension or severe hypoxia remains to be determined. Catheter-based therapy can reduce the angiographically concerning thrombus burden, reduce RV strain, and possibly impact downstream mortality and morbidity. Although such approach can be performed relatively safely in many patients, there are no data to suggest that this therapy would be safe or beneficial in all patients with submassive PE. The Pulmonary Embolism Response to Fragmentation, Embolectomy and Catheter Thrombolysis registry will attempt to define the role of catheter therapies in this patient group.[57]

Statement of Conflict of Interest

All authors declare that there are no conflicts of interest.

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