Acute-on-Chronic Massive Pulmonary Embolism Treated With 3-Loop Snare

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A 70-year-old man presented with acute massive pulmonary embolism (PE). His clinical condition deteriorated despite regular heparin and thrombolytic agent treatment, and he eventually developed cardiogenic shock. A thrombus originating from the inferior vena cava was detected and acute-on-chronic embolization resulted, with an unusual clinical course. Aggressive catheter thrombectomy with pigtail rotation failed to fragment the organized thrombus, but it was successfully removed by a 3-loop snare device combined with guiding catheter under percutaneous cardiopulmonary support. (Circ J 2008; 72: 1909–1913)

Key Words: Acute pulmonary embolism; Catheter thrombectomy; Inferior vena cava; Intravascular ultrasound; Snare

Despite modern diagnostic and reperfusion strategies, overall mortality in cases of acute pulmonary embolism (PE) remains high1,2. In addition to standard treatment with anticoagulation with heparin, thrombolysis is indicated in patients with acute massive PE.3 Open surgical thrombectomy is an option if thrombolysis is unsuccessful, but mortality is still high.4,5 There is accumulating practitioner experience in the use of recently developed thrombectomy devices for catheter-thrombectomy of acute massive PE.6,7 We describe an unusual case originating from the inferior vena cava (IVC), for which we used a combination of EN Snare (the retrieval and manipulation system designed by Medical Device Technologies Inc, FL, USA) and 8Fr percutaneous transluminal coronary angioplasty (PTCA) guiding catheter (GC). We believe this is the first report of treating acute PE in this way.

Case Report

A 70-year-old man was treated at an emergency department for acute shortness of breath and collapse while playing golf. One month prior, he had suffered sudden-onset dyspnea with gradual recovery. The patient had underlying diabetes mellitus and had undergone subtotal gastrectomy for gastric cancer 1 year earlier.

On examination, initial blood pressure was 110/50 mmHg, but heart rate was 120 beats/min. The jugular vein was engorged, and auscultation revealed a systolic murmur at the left sternal border. ECG showed sinus tachycardia with a deep S-wave in lead I, Q wave with inverted T in lead III and completed right bundle branch block with T inversion in leads V1–3. Further echocardiography revealed right ventricle dilatation and dysfunction.

Initial arterial blood gas analysis demonstrated significant hypoxemia, as well as metabolic acidosis, even under oxygen supply (fraction of inspired oxygen=40%, oxygen saturation=89.7%, pH=7.346, PCO2=32.8 mmHg, PO2=55.1 mmHg, HCO3=18.8 mmol/L, base excess=–9.0 mmol/L).

Chest computed tomography revealed a large thrombus of the bilateral pulmonary arteries (PA) (Fig 1A). Additionally, calcification with peripheral hypodense material was noted in the lumen of the IVC near the level of the stomach (Fig 1B).

Further venous angiography demonstrated that neither of the patient’s legs had deep venous thrombus, but both angiography and intravascular ultrasound revealed thrombus-like material in the lumen of the IVC (Fig 2). Biopsy and pathologic examination proved it to be fibrin thrombus.

The available disseminated intravascular coagulation screen data included platelets=9.6×10^11/l (normal range (NR): 15–35×10^11/l), fibrinogen=238.2 mg/dl (NR: 200–400 mg/dl), fibrin degradation products (FDP)=21 µg/dl (NR: 0–4.9 µg/dl), international normalized ratio of prothrombin time=1.03 and activated partial thromboplastin time=26.5 s (normal control: 20–30 s). In addition, the white cell count=13.15×10^9/l (NR: 4.3–10.8×10^9/l), hemoglobin=15.7 g/dl (NR: 14–18 g/dl), alanine aminotransferase=62 IU/L (NR: 7–56 IU/L), creatinine=0.6 mg/dl (NR: 0.6–1.2 mg/dl) and C-reactive protein <0.2 mg/dl.

The patient required mechanical ventilation support because respiratory failure quickly developed. Swan-Ganz catheterization revealed a markedly high PA pressure (72/12, mean=37 mmHg).

Emergency thrombus fragmentation with a pigtail catheter and 8Fr GC thrombus suction was performed immediately. The thrombus was unusually dense and could not be macerated (Figs 1C,D). The patient then consented to aggressive thrombolysis with urokinase (240,000 units...
Fig 1. Initial computed tomographic (CT) images and pulmonary angiogram. (A) Enhanced chest CT scan shows thrombus obstructing both pulmonary arteries (PA). (B) Abdominal CT scan shows calcification with peripheral low-density material in the lumen of the inferior vena cava. (C, D) Pulmonary angiogram showing large thrombus in both PA (white arrowhead).

Fig 2. Venography and intravascular ultrasound (IVUS) of the inferior vena cava (IVC). (A) Filling defect on the right side of the IVC (black arrow) with surgical clips nearby (white arrowhead). (B) IVUS image of the narrowed IVC site: homogenous echo mass surrounds the calcified IVC wall.

Fig 3. Catheter thrombectomy via the right subclavian vein. (A) Design of the snare. (B) The snare is rotated to macerate the organized thrombus in the left pulmonary artery.
Loop Snare for Acute Massive PE

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[U]) in the coronary care unit. His blood pressure fell to 70/40 mmHg, and aggravated oxygen desaturation was observed, even after repeating the thrombolysis twice with 400,000 U tissue plasmin activator (tPA) on the night of admission and again the next morning (total dose: 800,000 U). Percutaneous cardiopulmonary support (PCPS) was required urgently to maintain oxygenation saturation and hemodynamic stability on the day after admission.

Catheter thrombectomy was again attempted using a combination of an EN Snare and the 8Fr PTCA GC. The EN Snare was rotated and retrieved in both PA under PTCA GC support (Fig 3). The large, dense thrombus was successfully fragmented by the Snare and using syringe suction the GC captured small pieces of mixed fresh and organized thrombus, which were then withdrawn through the introducer sheath while maintaining syringe vacuum. The procedure was repeated to extract residual clot. The pathologic result was the same as for the IVC thrombus.

Improved PA flow was immediately noted, and the PA pressure dramatically decreased from 67/22 (mean=37) mmHg to 28/15 (mean=19) mmHg after the intervention (Fig 4).

PCPS was successfully weaned 2 days later and the heparin was continued for residual PE: 20,000–30,000 U/day to maintain the activated clotting time (ACT) between 180 and 240 s. Unfortunately, a large intracranial hemorrhage (ICH) occurred 8 days later and despite emergency brain decompression, the patient died of brain hernia and hospital-acquired pneumonia. The follow-up platelet count was 13.6x10^4/µl and FDP was 5µg/dl before the ICH.

Discussion

Rare Origin of Thrombus

The origin of the thrombus causing PE in this case was unusual. Over 90% of cases arise in the proximal deep veins of the legs. Deep venous thromboembolism (DVT) usually begins in the calf vein and extends into the popliteal and femoral veins. Venous thrombi are less common in the arms, although patients with central venous catheters, particularly those with malignancies, may develop emboli from axillary–subclavian venous thrombosis.

Although cases of acute thrombosis of the IVC have been reported, most patients had tumor invasion or had undergone early implantation of prophylactic IVC filters with anticoagulant therapy to prevent severe embolic events. Such patients usually exhibit leg swelling or edema, whereas the present patient had a rare presentation. Because the IVC lumen was not totally occluded by the organized thrombus, he did not present with the typical signs and neither physical examination nor venography revealed evidence of DVT. The calcification of the IVC wall, which probably resulted from previously inserted surgical clips, induced chronic thrombus formation, so this case represents a new differential diagnosis for the predisposing factors in PE.

Fig 4. Successful thrombus fragmentation. (A,B) Pulmonary angiogram shows subtotal occlusion of the right pulmonary artery and near total occlusion of the left pulmonary artery. Pulmonary artery pressure was 67/22 (mean pressure=37) mmHg. (C,D) Pulmonary angiogram shows improved branch perfusion after the intervention. Pulmonary artery pressure was 28/15 (mean pressure=19) mmHg after treatment. The thrombus is indicated by the arrowheads.
Successful Thrombus Fragmentation by EN Snare

Standard treatment for acute massive PE is thrombolysis, but some patients with severely compromised hemodynamic conditions may be contraindicated. There are other contraindications for aggressive thrombolysis and for these high-risk patients, catheter or surgical thrombectomy is necessary.

Previous studies have reported several devices for catheter thrombectomy, such as the rotatable pigtail catheter, Greenfield transcutaneous embolectomy catheter, Amplatz thrombectomy device, PTCA GC, percutaneous transluminal angioplasty balloon and the hybrid treatment. The hybrid treatment strategy of localized fibrinolysis, clot aspiration and catheter thrombectomy is reportedly more effective than catheter thrombectomy alone.

In the current case, the EN Snare device was initially used for thrombus fragmentation. Because the patient had suffered an episode of dyspnea 1 month prior to admission, he was believed to have acute-on-chronic thrombus formation in his PA and so the density of the thrombus was higher than typically observed with acute PE. The organized thrombus was refractory to both systemic thrombolysis and initial catheter thrombectomy.

The special characteristic of this case was the fibrin thrombus in the PA, which was the likely cause of the failed pigtail fragmentation. Failed catheter thrombectomy is reportedly associated mainly with chronic PE resulting from multiple embolic episodes.

The EN Snare was originally designed to catch and retrieve broken wires or balloon catheters and its advantages for fragmenting organized thrombus are several. First, its 3-nitinol loop structure easily penetrates the thrombus and increases the contact area. Second, it is easily rotated by hand and easily retrieved back into the GC. Third, its design and elasticity and flexibility minimize the risk of PA perforation. Fourth, the EN Snare is available in most catheterization laboratories, although a notable disadvantage is its high purchase cost. An important supplementary device in this case was the 8Fr PTCA GC, which helped aspirate small thrombus fragments.

The EN Snare device was clearly more effective than the pigtail catheter for breaking down the organized thrombus into small fragments. Clinical success was immediate, and the fragmented clot could be exposed to heparin and tPA.

Notes and Risks in Using EN Snare

We initially tried using the loop snare to fragment the hard thrombus and there are important aspects to note here. First, the snare profile should be based on the size of the PA because the bilateral PA enlarge in pulmonary hypertension. We chose a snare with an expandable loop, ranging from 27 to 45 mm. Because of its elasticity and flexibility, the loop can adequately cover and effectively fragment a large thrombus. Second, the En Snare is 120 cm in length and is manufactured in a 100-cm GC. It has a small, freely movable section while the GC is added to a Y-shape connector for pressure monitoring. To overcome the limitation of this design for thrombectomy, we shortened the GC by cutting the proximal 15 cm of an 8Fr GC and the distal 5 cm from a regular 7Fr sheath and then joined them.

There are also some potential risks. When the 8Fr GC is placed in the main PA, the procedure could stimulate the right ventricle and possibly induce a cardiac arrhythmia, such as frequent pre-excited ventricular capture or even short-run ventricular tachycardia. A 0.035-inch wire and 5Fr small-curve catheter can be put inside the 8Fr GC for gentle advancement into the PA and we found this combination gave adequate support for careful and easy manipulation.

Because the surface of the En Snare loop is not as smooth as that of a pigtail catheter; endothelial damage may occur during rotation of the snare in the PA, which could become a nidus for subsequent thrombus formation.

Risk Factors for ICH

Although catheter embolectomy with the EN Snare was clinically successful, the patient eventually died from ICH and brain hernia. The lack of history of head trauma and the 8-day withdrawal of thrombolytic agent suggest the ICH was related to heparin use. The risk of intracranial bleeding associated with IV unfractionated heparin is reportedly <2% and older patients (≥70 years) have a clinically increased risk of major bleeding.

During admission, the present patient received continuous IV heparin adjusted to maintain the ACT at 180–240 seconds (normal=107±13 s). Although initial thrombocytopenia (platelet count=9.6×10⁹/l) was noted on the 1st admission day, the platelet count gradually recovered to 13.6×10⁹/l without platelet transfusion. FDP was also rechecked and it decreased from the initial 21 μg/dl to 5 μg/dl before the ICH. It is unlikely that either DIC or heparin-related thrombocytopenia occurred before the ICH. Initial thrombocytopenia of acute PE has been reported and could be reasonably explained in the present patient by platelet consumption.

If aggressive intervention can be performed early, at the time of initial hemodynamic change, the clinical usage of heparin might be shortened. Nevertheless, the role of aggressive catheter thrombectomy and the clinical efficacy of the EN Snare for improving prognosis in cases of acute PE require further investigation in prospective studies.

Conclusion

We described a case of acute PE with the rare origin of thrombus from the IVC, which was probably stimulated by previous suture clips used during subtotal gastrectomy.

The 3-loop EN Snare successfully fragmented the organized thrombus in the PA, with supplementary PTCA GC suction. As an adjunct to thrombolytic therapy, this hybrid treatment can achieve immediate hemodynamic improvement after acute massive PE. It is minimally invasive and offers an attractive alternative for patients with contraindications to thrombolysis.

References

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