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Case Report

Pulmonary Embolectomy for Acute Pulmonary Embolism: A Word of Caution

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Pulmonary embolectomy is an effective treatment of acute pulmonary embolism (APE) but not for chronic pulmonary thromboembolism. We described surgical experience of two patients with APE in preexistent unidentified chronic pulmonary thromboembolism. One patient who presented with severe hypoxia but stable hemodynamics underwent successful pulmonary endarterectomy for proximal organized thrombus instead of pulmonary embolectomy. The other patient who required extracorporeal membrane oxygenation for severe hypoxia developed right heart failure because of residual distal organized thrombus after pulmonary embolectomy. Clinical and radiographical presentation of APE in chronic pulmonary thromboembolism mimics APE, and thus, candidates of pulmonary embolectomy should be carefully selected.

Keywords: pulmonary embolectomy, acute pulmonary embolism, chronic thromboembolic pulmonary hypertension

Introduction

Massive acute pulmonary embolism (APE) is a fatal condition with hemodynamic instability and requires aggressive treatment to resolve or remove thrombi in addition to anticoagulation therapy. Thrombolysis is generally recommended as a first-line therapy, and pulmonary embolectomy could be indicated for patients with massive APE who have contraindications to thrombolysis.[1)] However, the outcomes of patients who were treated with aggressive pulmonary embolectomy with or without extracorporeal membrane oxygenation (ECMO) support were reportedly promising,[2)] and even patients with sub-massive APE could be candidates of surgical treatment.[3)] We have treated surgically massive or sub-massive APE with failed medical therapy or thrombi in transit and described, herein, a critical pitfall for surgical treatment.

Case 1

A 68 year-old man with sudden onset and progressive worsening of dyspnea was referred to our hospital. The patient complained that he had not suffered dyspnea on exertion until the event and did not have risk factors of APE. Transthoracic echocardiography showed right ventricular dilatation without right ventricular hypertrophy, a D-shaped left ventricle, and tricuspid pressure gradient of 50 mmHg. Enhanced computed tomography (CT) scan showed floating cylindrical thrombi in the bilateral pulmonary arteries, enlarged pulmonary trunk, extremely dilated bronchial arteries (**Figs. 1A** and **1B**), and deep vein thrombus in both femoral and popliteal veins. He presented with hypoxia with PaO_2 of 62 mmHg with 5 L nasal cannula but was hemodynamically stable. Heparin therapy was started on a diagnosis of sub-massive pulmonary embolism (PE), but right ventricular strain and hypoxia were not improved on the next day, despite stable hemodynamics with blood pressure of 116/59 mmHg and pulse rate of 72 bpm. We performed pulmonary embolectomy under cardiopulmonary bypass instead of failed heparin therapy. An incision was made in the right main pulmonary artery and then mural chronic organized thrombus was noticed inside. We subsequently performed

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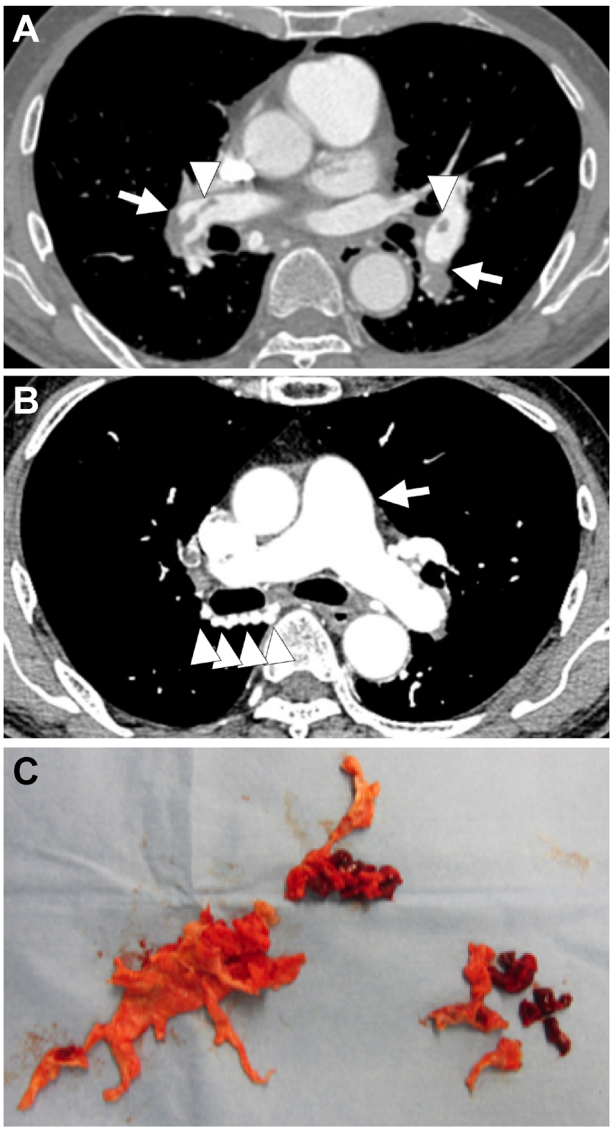

Fig. 1 **A**: Preoperative computed tomography (CT) scan (Case1): Arrowheads show filling defects indicating floating cylindrical thrombus. Arrows show mural thrombus that forms obtuse angles with the vessel wall. **B**: preoperative CT scan (Case1): Arrowheads show remarkably dilated bronchial arteries. Arrow shows enlarged pulmonary trunk. **C**: Surgical specimen (Case1): Jamieson type I disease.

pulmonary endarterectomy (PEA) with deep hypothermic circulatory arrest and successfully removed organized thrombus from both pulmonary arteries (**Fig. 1C**). The patient recovered uneventfully without residual pulmonary hypertension and was discharged 10 days after the surgery.

Case 2

A 32 year-old woman who had suffered mild dyspnea on exertion and developed sudden progressive worsening of dyspnea was transferred to our hospital. She had no risk factors of APE but protein S deficiency. Transthoracic echocardiography showed right ventricular dilatation without right ventricular hypertrophy, a D-shaped left ventricle, and tricuspid pressure gradient of 50 mmHg. Enhanced CT scan showed enlarged pulmonary trunk, floating thrombi in the bilateral interlobar pulmonary arteries, infiltrates suggesting pneumonia in both upper lobes (**Figs. 2A–2C**), and deep vein thrombus in both popliteal veins. The patient presented with severe hypoxia, which was not improved after intubation with SpO_2 of 76% on FiO_2 of 1.0; tachycardia with pulse rate of 108 bpm; and hypotension with blood pressure of 111/70 mmHg with administration of noradrenaline (0.5γ/kg/min). We instituted veno-arterial ECMO with percutaneous femoral cannulation and performed an emergent pulmonary embolectomy. We did not make a diagnosis of chronic thromboembolic pulmonary hypertension (CTEPH) at the surgery. We found neither organized thrombus nor yellow plaques, which is often seen in CTEPH, in the main and lobar pulmonary arteries. We did not observe clearly inside the segmental arteries due to back bleeding and were not sure that stenosis in the segmental artery was caused by organized thrombus. Intraoperative transesophageal echocardiography showed patent foramen ovale with right to left shunt. It was presumed that infiltrates in the upper lobes and PE in the lower lobes caused ventilation perfusion (V/Q) mismatch leading to severe hypoxia, and right to left shunt through patent foramen ovale further exacerbated hypoxia. We performed pulmonary embolectomy and closure of patent foramen ovale under cardiopulmonary bypass (**Fig. 2D**). Poor oxygenation was sustained after weaning from cardiopulmonary bypass but gradually improved in the following several hours in the intensive care unit (ICU). However, the patient presented with hypotension and decrease in urine output thereafter. The Swan–Ganz catheter showed pulmonary hypertension with pulmonary artery pressure of 50/42 (37) mmHg and transthoracic echocardiography showed right ventricular dilatation and a D-shaped left ventricle. We presumed that unidentified or residual pulmonary hypertension caused right heart failure and treated with NO inhalation and epoprostenol. The patient was gradually recovered and extubated on postoperative day 7. Despite discontinuance of epoprostenol, transthoracic echocardiography showed no pulmonary hypertension. After a 3-month anticoagulation therapy, pulmonary angiography showed webs and bands in both inferior lobe arteries (**Fig. 2E**), which were the same obstruction seen in CT scan obtained shortly after the surgery, and right heart catheterization showed pulmonary artery pressure of 35/14 (24) mmHg. Based on her clinical course, it was presumed that the patient had preexistent unidentified chronic pulmonary thromboembolism at the time of the index PE. We assumed that

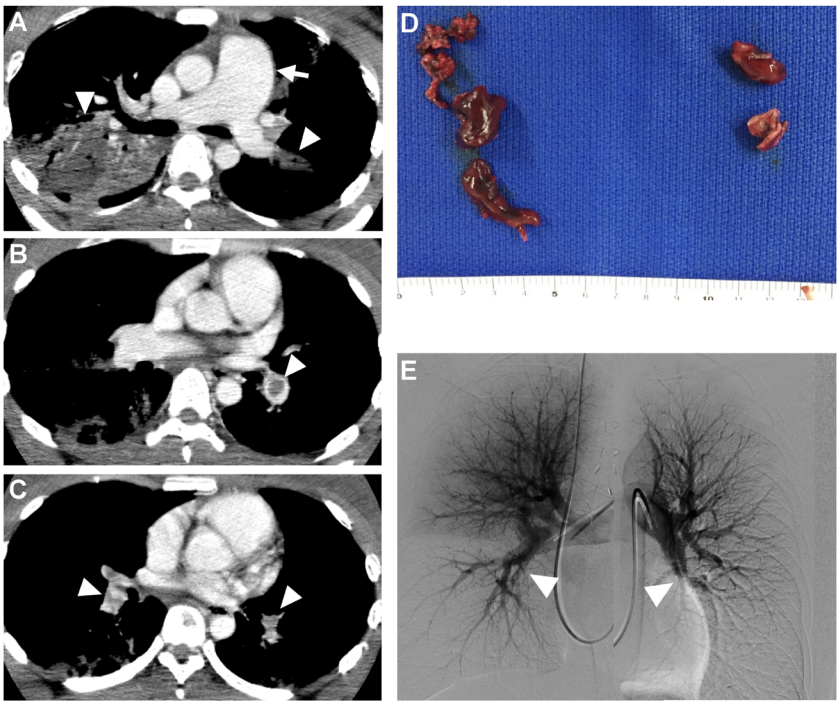

Fig. 2 **A**: Preoperative computed tomography (CT) scan (Case2): Arrowheads show infiltrates in both upper lobes. Arrow shows enlarged pulmonary trunk. **B**: Preoperative CT scan (Case2): Arrowhead shows central filling defect indicating floating cylindrical thrombus in the left pulmonary artery. **C**: Preoperative CT scan (Case2): Arrowheads show central filling defects in the lower lobe arteries. **D**: Surgical specimen (Case2): Fresh red thrombi. **E**: Postoperative pulmonary angiography (Case2): Arrowheads show severe stenosis (webs and bands) in the lower lobe arteries.

severe PH after the surgery might have been attributed to residual fresh thrombus and hypoxia-induced vascular spasm since pulmonary artery pressure returned to almost normal with anticoagulation therapy and PGI2 administration.

We obtained the patients' consent to the submission and publication of this case report, and they signed a standard publication consent form of our institution.

Discussion

Pulmonary embolectomy is a simple and effective procedure for APE but is not effective and rather harmful for CTEPH, resulting in residual pulmonary hypertension leading to fatal right heart failure. It is thus mandatory to make a definitive diagnosis of APE or CTEPH prior to surgery. Clinical presentation with stable hemodynamics despite severe hypoxia may be indicative of APE in CTEPH. A normal right ventricle cannot tolerate sudden increase of pulmonary artery pressure and cause hemodynamic instability secondary to right heart failure. Pulmonary hypertension with a mean pulmonary artery pressure of greater than 40 mmHg despite massive embolic obstruction indicates chronic pulmonary artery obstruction allowing right ventricular hypertrophy.[4]

Multidetector CT angiography is a standard modality to diagnose APE and CTEPH. Vascular signs specific to CTEPH include intimal irregularities, webs or bands, abrupt vessel narrowing, pouch defect, and crescent-shaped mural thrombus that forms obtuse angle with the vessel wall.[5]

However, these findings may not be evaluated properly.

This is because a fresh thrombus filled in the pulmonary artery can hide these signs, especially in shocked patients and patients receiving ECMO support, as it does not obtain sufficient quality CT images. Therefore, it is also important to evaluate indirect signs related to pulmonary hypertension (enlargement of pulmonary arteries and right ventricular hypertrophy), collateral vessels (dilated bronchial arteries), and parenchymal signs (mosaic perfusion pattern).[5] However, it must be noticed that the presence of organized thrombus on CT scan may not necessarily indicate CTEPH. Guérin et al. showed that at least two signs of CTEPH on CT were seen in 20% of patients who were not diagnosed with symptomatic CTEPH during a follow-up.[6]

We made a diagnosis of APE for our patients on the basis of CT findings showing central filling defects. The vascular

signs of CTEPH was concealed with fresh thrombi, which formed obtuse angles with the vessel wall though. However, indirect signs including enlarged pulmonary trunk and dilated bronchial arteries (Case 1) suggested preexistent pulmonary hypertension and chronic organized thrombus. Prominent right ventricular hypertrophy was not observed in both patients possibly because of mild pulmonary hypertension. To make a definitive diagnosis for surgical treatment, indirect signs as well as vascular signs of CTEPH must be taken into consideration.

Anticoagulation and/or thrombolysis are a better option for patients with highly presumed preexistent CTEPH, which can provide an opportunity to determine the extent of chronic organized thrombus. Venoarterial extracorporeal membrane oxygenation as an initial intervention could be effective in triage patients with PE prior to the appropriate therapy in case of massive PE.[2] If proximal organized thrombus is noticed at the time of pulmonary embolectomy, PEA can be subsequently performed at an experienced PEA center. Otherwise, ECMO support with or without chest closure and delayed PEA or balloon pulmonary angioplasty would be necessary for life saving.[7]

Conclusion

The clinical and radiological presentation of APE in preexistent CTEPH closely mimics APE. Pulmonary embolectomy for APE in CTEPH is not effective, and thus, candidates of emergent pulmonary embolectomy should be carefully selected.

Disclosure Statement

All authors have no conflicts of interest to declare.

Additional Note

The permission to publish this case report has been granted by the patients.

Author Contributions

Study conception: TI, KI
Data collection: TI, KI
Analysis: TI
Investigation: TI, KI
Writing: TI
Funding acquisition: none
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspects of the work: all authors

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