The embolus, removed 4 days from symptom onset, was evaluated by using SEM, as previously described.³ Briefly, after washing, the thrombus was fixed by means of permeation with 2.5% glutaraldehyde and then treated with alcohol, dehydrated, gold coated, and photographed digitally with a Hitachi S-4700 SEM (Hitachi, Tokyo, Japan).

The SEM images revealed that the inner parts of the removed thrombus (Figure 1, B and C) are formed with tightly packed, thick fibrin fibers resembling a solid mass; however, in the very center there was cellular debris derived predominantly from erythrocytes. At the periphery of the clot (Figure 1, D and E), a large amount of red blood cells embedded in a relatively thin fibrin network with low amounts of platelet aggregates was visualized. No layered structure of the embolus was observed.

DISCUSSION

Contrary to the available scarce morphologic data,¹ we did not observe a layered thrombus structure. Compared with thrombi removed from a coronary artery of patients with acute myocardial infarction, which were fibrin rich with erythrocytes and almost no platelets,⁴ the pulmonary thrombus was much more organized and contained relatively more

fibrin and fewer red blood cells, which were present mainly in peripheral segments of the clot. The more compact the fibrin fiber structure, the less susceptible to lysis,² and not surprisingly, such thrombi (Figure 1) are resistant to endogenous fibrinolysis.

Thrombi from patients not receiving thromboprophylaxis are likely even more dense, as seen at higher thrombin concentrations.⁵ Our findings might also help explain why fibrinolytic therapy might fail to rescue patients with large pulmonary thrombi and comprised cardiopulmonary function, in whom embolectomy could be more effective.

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Pulmonary thromboendarterectomy in Klippel-Trénaunay syndrome

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Chronic thromboembolic pulmonary hypertension (CTEPH) occurs in 4% of patients with pulmonary embolism (PE).¹ Untreated, the likelihood of survival is poor. Klippel-Trénaunay syndrome (KTS) is a rare complex vascular malformation that typically includes the triad of capillary vascular malformation (port-wine stain), venous malformation(s), and soft tissue or bony hypertrophy.²

Patients with KTS are at increased risk for peripheral venous thrombosis and pulmonary emboli.³ When medical management of PE and CTEPH fails, pulmonary thromboendarterectomy (PTE) is an important, potentially lifesaving therapy.¹

CLINICAL SUMMARY

Three patients with KTS (2 women aged 19 and 46 years and 1 man aged 26 years, Table 1) underwent PTE. The patients had symptoms for an average of 9.6 months (range, 3–19 months) before surgery. Patient 2 had a diagnosis of deep vein thrombosis before the CTEPH diagnosis. Patient 1 previously had a right arm amputation for complications of vascular malformation. No patient had a clotting disorder. Each patient had all 3 manifestations of KTS, including capillary malformations, venous malformations, and soft tissue hypertrophy.

A single surgeon performed all operations. The surgical technique used was described by Jamieson and colleagues.¹

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TABLE 1. Demographic and procedural information for the three patients with Klippel-Trénaunay syndrome who had pulmonary thromboendarterectomy performed for chronic thromboembolic pulmonary hypertension at the Mayo Clinic College of Medicine

Patient no.	Age (y)	Gender	Preoperative DVT	Length of stay (d)	Complications	RVSP before (mm Hg)*	RVSP after (mm Hg)*	NYHA before	NYHA after	Available follow-up (y)
1	19	F	N	6	None	49	28	3	1	0.5
2	26	M	Y	18	Stroke	69	68	3	2	6
3	46	F	N	8	None	55	46	3	2	1.3

Postoperative NYHA class is listed as available from 6-month follow-up (short-term follow-up). DVT, Deep vein thrombosis; RVSP, right ventricular systolic pressure; NYHA, New York Heart Association. *As measured by echocardiography.

All patients had preoperative inferior vena cava filter placement, pulmonary angiography, and preoperative and postoperative echocardiography. Each of the 3 patients underwent bilateral PTE (Figure 1).

All patients were monitored intraoperatively and postoperatively with pulmonary artery catheters. Main pulmonary artery pressures under similar hemodynamic conditions decreased from 46 ± 11.5 mm Hg pre-procedure to 24.3 ± 6.7 mm Hg post-procedure ($P = .047$). The echocardiography-measured right ventricular systolic pressure decreased

from 49 to 28 mm Hg in patient 1 and from 55 to 46 mm Hg in patient 3, but did not change in patient 2 (69 to 68 mm Hg, Table 1). Echocardiography-assessed right ventricular function improved in all 3 patients.

The average estimated blood loss was 1273 ± 629 mL (range, 900–2000 mL). All 3 patients had blood transfusions and intraoperative surgical blood cell salvage (“Cell Saver”) transfusions intraoperatively. Cardiopulmonary bypass was used for an average of 265 ± 35 minutes (range, 237–304 minutes) with circulatory arrest for an average of 44 ± 12 minutes (range, 37–58 minutes). Chest tubes were placed in all patients, with an average output of 517 mL (range, 340–705 mL), and were removed after an average of 3 days (range, 1–6 days). Patients 1 and 3 had a hospital length of stay of 6 and 8 days, respectively, and had no complications. Patient 2 was in the hospital for 18 days secondary to a postoperative stroke, consisting of left upper-extremity weakness. He also had recurrent postoperative pleural effusions, necessitating repeat chest tube placement. At the time of discharge, all effusions had resolved, and the patient’s weakness was improving with therapy.

There were no deaths. All patients reported marked symptomatic relief postoperatively. All patients were treated with long-term anticoagulation postoperatively. Inferior vena cava filters were not removed in any of the 3 patients. Average available follow-up is 2.6 years (range, 0.5–6). No patient has had a postoperative deep vein thrombosis or PE. Patient 2, while symptomatically improving, had no significant change in pulmonary pressures postoperatively.

DISCUSSION

We report 3 patients with KTS who had recurrent pulmonary emboli and CTEPH, all of whom had a PTE performed. Surgery in patients with KTS is high risk, particularly with associated risks of hemorrhage, infection, and thrombosis.³ One of our 3 patients had a complication: Patient 2 had a stroke and recurrent CTEPH despite thromboendarterectomy.

The use of PTE in patients with KTS was first reported in 1991.⁴ A 53-year-old man with KTS and CTEPH underwent a successful PTE, but he died 10 days postoperatively secondary to recurrent massive PE. In 2000, a 24-year-old

FIGURE 1. Fully dissected organized thrombus taken from the right pulmonary arterial tree of patient 1.

man was described with failure of medical treatment for recurrent pulmonary emboli.⁵ PTE was successfully performed, with no recurrence of CTEPH at the 7-month follow-up. An 11-year-old boy was reported to have undergone pulmonary thromboendarterectomy (Huiras and colleagues³), but he died after recurrent PE.

CONCLUSIONS

PTE is a difficult operation to perform. The procedure was developed and advanced by Jamieson and colleagues.¹ As experience with the technique has grown, many centers have seen mortality decrease to less than 5%. There are 3 previously reported cases of PTE performed for KTS, but 2 of the patients died after surgery.³⁻⁵ PTE should be performed only by surgeons who are experienced with the

operation. In selected patients, PTE can be an effective and potentially lifesaving curative procedure, and it is an important therapeutic option in patients with CTEPH.

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Successful surgery for atrioesophageal fistula caused by transcatheter ablation of atrial fibrillation

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Catheter ablation (CA) is an effective and safe technique to treat atrial fibrillation; however, it can cause rare but lethal complications.¹ Atrioesophageal fistula (AEF) caused by diffusion of the ablative energy through the left atrial wall to the esophagus (because of the absence of pericardium) is lethal if not treated surgically.

CLINICAL SUMMARY

A 35-year-old man had a 3-year history of atrial fibrillation that had been permanent for the last 2 years. Amiodarone had proven ineffective, and an electrical cardioversion failed. Echocardiography showed the left side of his heart (left ventricular ejection fraction, 60%) to be preserved with a left atrial diameter of 27 mm. CA performed at another institution was uneventful, and the patient was discharged the next day in sinus rhythm.

Thirty-eight days later, the patient had fever, chest pain, and vomiting and was urgently admitted to a peripheral hospital, where he presented a left hemiplegia and convulsive crisis. There was no early lesion observed on cerebral computed tomography (CT) scan. Blood cultures were positive for streptococcus salivarius, transthoracic echocardiography showed no evidence of endocarditis, and lumbar puncture results were unremarkable. A specific antibiotic therapy was initiated, and the patient was transferred to the University Hospital of Rangueil. Cerebral magnetic resonance imaging showed widespread ischemic changes consistent with multiple emboli, and CT scan showed cerebral, splenic, and renal emboli associated with the presence of air within the mediastinum and left atrium, suggesting an AEF (Figure 1).

Because of the patient's poor condition, a silicone-covered stent was initially placed in the esophagus under radiology to seal off the AEF, but the stent migrated into the stomach a few hours later.

Surgery was performed via a left thoracotomy because of a left pleural effusion. The left femoral vessels were accessible, and a cardiopulmonary bypass was on standby. After careful dissection, the AEF was identified and the esophagus was separated from the left atrial wall. A preventive suture was done on the atrial wall despite the absence of perforation. The esophageal perforation (Figure 2, B) was closed using adsorbable monofilament sutures. A pediculated autologous pericardial patch was then placed between the

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