

Thromboendarterectomy in Chronic Pulmonary Thromboembolism

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Abstract

Chronic pulmonary thromboembolic disease is entrapment of thrombus in pulmonary arteries from a single episode or repeated embolic episodes that subsequently organize, or thrombi that develop inside the pulmonary arteries into firm, fibrous tissue that becomes incorporated into the vessel wall.

We operated 7 patients with end stage chronic pulmonary thromboembolism over a two year period at our center. Four patients had good function class after operation on follow up at 6 to 12 months and we administered warfarin prophylaxis (with international normalized ratio, INR, of 3) for them. Three patients died in hospital postoperatively. We used extracorporeal membrane oxygenation (ECMO) for one of them but it failed on the third postoperative day (*Iranian Heart Journal 2009; 10 (4):59 -62*).

Key words: chronic pulmonary thromboembolism ■ pulmonary thromboendarterectomy

Chronic pulmonary thromboembolic disease is entrapment of thrombus in the pulmonary arteries from a single or repeated embolic episodes that subsequently organize, or thrombi that developed inside the pulmonary arteries into firm, fibrous tissue that becomes incorporated into the vessel wall.¹

These processes result in variable degrees of pulmonary artery obstruction and consequent pulmonary hypertension when obstructions become severe.¹

In 1980, Daily and colleagues reported the use of hypothermic circulatory arrest in combination with cardiopulmonary bypass (CPB) to eliminate severe back-bleeding and improve visualization of the pulmonary arteries during endarterectomy, and it is currently the preferred method of management.^{2,3}

Case Report

A 35 year-old man with a one-year history of dyspnea on exertion, two months of fever and in New York Heart Association (NYHA) functional class II was admitted to hospital. He was an IV drug abuser and had been treated adequately for tricuspid valve endocarditis with wide spectrum antibiotics. On physical examination, splitting of S2 was audible and jugular venous pulse was prominent. An electrocardiogram (ECG) showed right ventricular hypertrophy (RVH). Liver function tests, electrolytes, BUN, creatinine and hemoglobin levels were in the normal range. White blood cell count was 10400, with 47% neutrophils and 44% lymphocytes.

On three-dimensional CT scan of the heart and pulmonary vessels with IV contrast in MRP, MIP and VRI images, enlargement of

the main pulmonary artery (40mm) and left and right pulmonary arteries (20mm) was evident, low attenuated filling defect was seen in the lumen, 20x15mm in the right and 50x20mm in the left pulmonary artery, which extended to the descending branches.

The right ventricle (RV) and right atrium (RA) were larger than normal size, without evidence of remarkable filling defect. Signs of cavitory filling defect (infarct lesion) was seen in the left lung.(Fig. 2.)



Fig. 1. Chest X-ray.

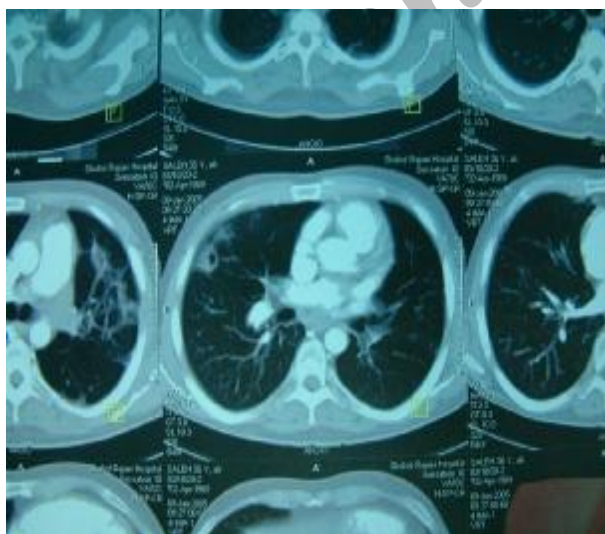


Fig. 2. CT Scan with IV contrast.

Operative technique

After inserting appropriate monitoring devices and placing ECG electrodes, a median sternotomy incision was made and cannulae inserted into the ascending aorta and both vena cavae and encircled with tapes. Cooling was initiated immediately after CPB was established. Venting was started from the right superior pulmonary vein, the aorta cross-clamped and cold cardioplegic solution infused into the aortic root, with subsequent infusions given every 15 minutes. The superior vena cava (SVC) was mobilized to the level of the innominate vein, and the right pulmonary artery was mobilized by retracting the vena cava laterally and the ascending aorta medially. General technique for establishing hypothermic total circulatory arrest (TCA) was applied. Methyl prednisolone 7mg/kg, thiopental 10.5 mg/kg, mannitol 0.3g/kg and furosemide 100mg were perfused and hematocrit maintained in the range of 18-22%.^{4,5} At the nasopharyngeal temperature of 14°C circulatory arrest was established. The main pulmonary artery was opened vertically and extended to the left pulmonary artery distally. An organized clot was seen, that extended to the beginning of the right pulmonary artery and through the left pulmonary artery to the upper and lower lobe branches. The right pulmonary artery was opened between the aorta and SVC. An endarterectomy plane was established with a sharp dissector and the intima and a portion of the media removed. The core of the thrombus was isolated circumferentially and removed from the upper lobe and remaining portion of the pulmonary artery and the arteriotomy incision was closed with a continuous 5-0 polypropylene suture. TCA time was 14 minutes. CPB was re-established and rewarming begun as closure of the artery was completed. The foramen ovale and tricuspid valve were examined via the right atrium and were normal. CBP was discontinued successfully and the procedure was completed in the standard manner. On day 10, transthoracic and transesophageal

echocardiography were done because of mild dyspnea and elevated jugular venous pressure and revealed left ventricular end-diastolic dimension (LVEDD) 4.3cm, LV end-systolic dimension (LVESD) 2.99cm, LV ejection fraction (LVEF) 55%, right ventricular ejection fraction (RVEF) moderately reduced; RV systolic pressure (RVSP) 55mmHg, and RV 3.9cm. Early diastolic RV outflow tract (RVOT) collapse with a large pericardial effusion about 4.1cm was seen around the heart. The patient was transmitted to the operating room and sub-xyphoid drainage was done locally and he was discharged from the hospital four days later.

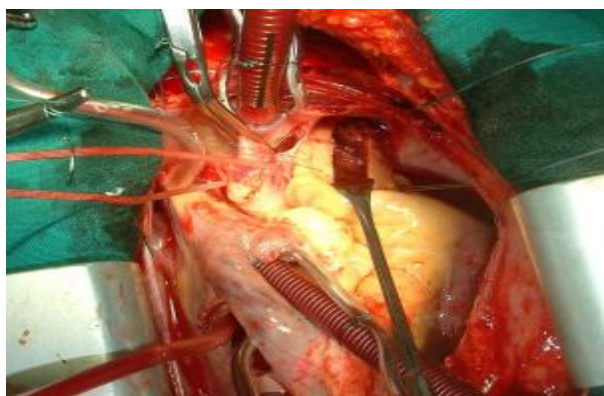


Fig 3. Organized clot in main pulmonary artery

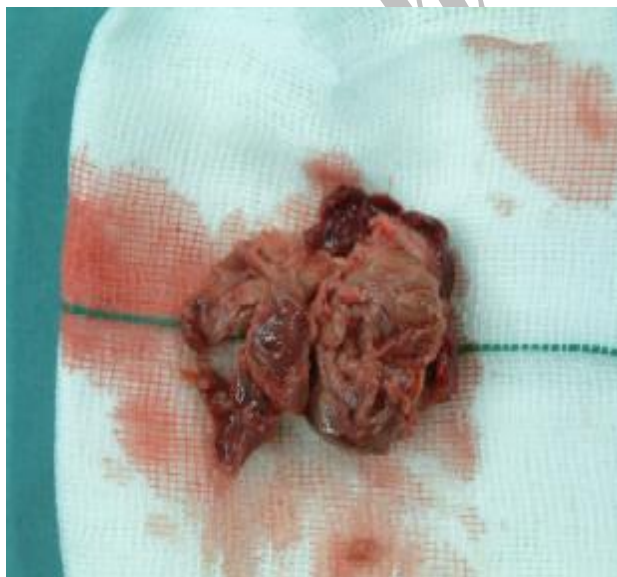


Fig 4: Organized thrombus

The second patient was a 32 year-old postpartum female who was admitted three months after labor for progressive dyspnea and periodic cyanosis. On lung CT scan with IV contrast, there were multiple thromboses in the main pulmonary artery and tributaries to segmental arteries. There was severe RV dysfunction with dilatation and hypertrophy. We operated the patient per the above mentioned technique, but after discontinuation of CPB, blood pressure dropped and extracorporeal membrane oxygenation (ECMO) was used for her. On the third postoperative day however, ECMO failed and the patient died.

We operated five further patients in this manner, three of whom went home with good functional class and we gave them prophylactic warfarin, and two patients died in the intensive care unit (ICU) on the second and third postoperative days. We opened the foramen ovale for a 35 year-old man with severe dyspnea and RV failure after completion of the operation because pulmonary pressure had not dropped, and he died on the third postoperative day because of low oxygen saturation and recurrent bronchial bleeding. The final fatality was a 53 year-old woman who had chronic pulmonary hypertension and severe dyspnea. She died on the second postoperative day in the ICU because of severe RV failure.

Conclusion

Pulmonary thromboendarterectomy is a practical method and can be life-saving in chronic pulmonary hypertension secondary to chronic obstruction of the pulmonary arteries and their branches. It can be curative and successful if used in selected patients. If the thromboses extend to the minor branches and if the pulmonary artery pressure does not drop after endarterectomy, the prognosis of the patient is poor.⁶

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