



Surgical management of “ab-extrinseco” main stem left coronary compression during pulmonary thromboendarterectomy

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Introduction

Extrinsic compression of the left main coronary artery (LMCA) is a common but often underestimated complication of severe pulmonary hypertension (PH) and resulting pulmonary artery (PA) dilatation.

Chronic thromboembolic pulmonary hypertension (CTEPH) is a rare cause of PH, with a challenging diagnosis considering the ambiguous clinical presentation, especially when the patient's only complaint is angina due to PA enlargement. Diagnosis of extrinsic LMCA compression demands a high index of suspicion, because these patients usually have no relevant past medical history or risk factors for coronary artery disease.

Although computed tomography (CT) and cardiac magnetic resonance imaging may be preferred first-line, noninvasive approaches, diagnosis is eventually made with coronary angiography and intravascular ultrasound. These typically show LMCA ostial stenosis, usually without associated atherosclerosis: LMCA presents as an eccentric stenosis and is sharply displaced towards the left coronary sinus. Clinically significant dislocation of the LMCA due to a dilated PA main trunk is typically associated with an LMCA take-off angle $<60^\circ$ and a PA diameter more than four centimeters (1).

Despite no official consensus on the best treatment for this secondary cause of angina, whenever possible, treatment of the underlying cause of PH is helpful. Generally, percutaneous coronary intervention is considered first-choice treatment, given the high surgical risk of patients with precapillary PH.

If surgical correction of the primary cause of PH is possible, as in the case of CTEPH with pulmonary thromboendarterectomy (PTE), the postoperative reduction in PH is often sufficient to reduce compression of the LMCA. Nonetheless, if the PA is extremely enlarged, plication of its main trunk helps reduce LMCA compression (2). Conversely, should coronary angiography detect a critical stenosis, coronary artery bypass grafting (CABG) is considered.

Clinical vignette

A 60-year-old male was admitted to the emergency department after an episode of syncope. He complained of dyspnea and unrecognized angina that had worsened in the previous months. CT confirmed CTEPH with a critical extrinsic compression of the LMCA by the enlarged PA (maximum diameter 45 mm). The patient was subsequently referred to our department and routine examinations were conducted including echocardiography, coronary and pulmonary angiography and right heart catheterization (RHC). These revealed severe precapillary PH [mean pulmonary artery pressure (mPAP) 48 mmHg, pulmonary capillary wedge pressure (PCWP) 8 mmHg, pulmonary vascular resistance (PVR) 4.3 WU] due to chronic, bilateral, segmental thromboembolism.

After 3 months of optimal but ineffective antihypertensive and anticoagulation therapy, the patient was considered eligible for surgery by our CTEPH multidisciplinary team.

Surgical technique

Preparation and exposition

Routine intraoperative monitoring includes body temperatures (skin, bladder and nasopharyngeal temperature), bilateral invasive blood pressure, central venous pressure, Swan-Ganz parameters (PAP, PCWP, cardiac output, PVR, mixed venous oxygen saturation) and transesophageal echocardiography.

Following our standard approach, a full median sternotomy is performed with an oscillating saw to avoid opening the pleural cavities and to reduce the risk of pleural effusion.

Aorto-bicaval cannulation for cardiopulmonary bypass (CBP) is then instituted: notably, the superior venous cannula is positioned through the right atrial appendage to obtain optimal exposure of the right PA. Once the left ventricle and pulmonary trunk are drained, systemic cooling is started to reach deep hypothermia at 20 degrees Celsius. The aorta is cross-clamped at 25–26 degrees and single-dose crystalloid histidine-tryptophan-ketoglutarate cardioplegia (HTK-Custodiol; Koehler Chemi, Alsbach-Haenlien, Germany) is delivered.

Operation

A longitudinal arteriotomy of the right PA is performed. Finding the cleavage plane is crucial to avoid damaging the arterial wall. The fibrotic, chronic thrombus is dissected from the wall and progressively removed. Care must be taken not to shatter the thrombus, because levering on it is crucial to perform the endarterectomy of the most distal branches including the subsegmental ones. During the procedure, 20 minutes of deep hypothermic circulatory arrest (DHCA) at 20 degrees Celsius is feasible and can be interrupted by 10 minutes of isothermal reperfusion if necessary (3). The purpose of adopting DHCA is to abolish the retrograde flow, which may impede a complete distal dissection of thrombi (4).

When a satisfactory PTE is achieved, the artery is reconstructed, and the same steps are carried out for the left PA. In consideration of this patient's PA dilatation, while rewarming, the trunk of the PA was reduced by linear plication and reinforced with two strips of Teflon felt, using two running 5/0 polypropylene sutures.

Finally, weaning from CPB is a critical moment, often requiring inotropic support or mechanical circulatory

support because of residual PH and temporary right ventricular failure (5).

Post-operative outcome

Post-operative observation is focused on cardiac output, peripheral and pulmonary pressures and resistances. Provided no major bleeding occurs, anticoagulation is started 6 hours after intensive care unit arrival with intravenous heparin and target aPTT ratio of two. On postoperative day five, oral anticoagulation is started in association with low molecular weight heparin (LMWH) until the INR is >2.5. Ceftazidime, a third-generation cephalosporin, is infused as empiric pulmonary antibiotic prophylaxis during the first seven postoperative days.

The patient's postoperative course was uneventful and he was discharged 19 days after surgery and was asymptomatic six months later. A significant hemodynamic improvement was measured with RHC (mPAP 35 mmHg, PVR 3.1 WU). A CT scan showed a reduced PA trunk diameter (maximum diameter 36 mm) without compression of the LMCA.

Comments

CTEPH is the only cause of PH with a curative surgical treatment other than lung transplantation. However, not every patient is suitable for surgery so a multidisciplinary evaluation is mandatory. Moreover, extrinsic compression of the LMCA and related angina may complicate and delay the diagnostic and therapeutic decision process.

Generally, available treatment options for symptomatic extrinsic compression of LMCA are either percutaneous or surgical. Considering the high-risk profile of these patients, mainly related to their pre-capillary PH, percutaneous revascularization has an advantageous risk-to-benefit ratio and is considered the first-line treatment (1).

If surgical treatment of CTEPH is feasible, PTE and the resulting drop of pulmonary pressure resolve the angina. However, when long-term PH causes extreme PA dilatation and persistent angina, linear plication of its main trunk should be considered during PTE. Finally, CABG is only considered when a critical atherosclerotic stenosis is discovered with coronary angiography.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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References

1. Galiè N, Saia F, Palazzini M, et al. Left Main Coronary Artery Compression in Patients With Pulmonary Arterial Hypertension and Angina. *J Am Coll Cardiol* 2017;69:2808-17.
2. Lee MS, Oyama J, Bhatia R, et al. Left main coronary artery compression from pulmonary artery enlargement due to pulmonary hypertension: a contemporary review and argument for percutaneous revascularization. *Catheter Cardiovasc Interv* 2010;76:543-50.
3. Jamieson SW, Kapelanski DP, Sakakibara N, et al. Pulmonary endarterectomy: experience and lessons learned in 1,500 cases. *Ann Thorac Surg* 2003;76:1457-62; discussion 1462-4.
4. Martin-Suarez S, Gliozzi G, Loforte A, et al. Pulmonary thrombo-endarterectomy for chronic thromboembolic pulmonary artery hypertension. *Curr Chall Thorac Surg* 2021;3:13.
5. Martin-Suarez S, Gliozzi G, Fiorentino M, et al. Role and management of extracorporeal life support after surgery of chronic thromboembolic pulmonary hypertension. *Ann Cardiothorac Surg* 2019;8:84-92.

1. Galiè N, Saia F, Palazzini M, et al. Left Main Coronary Artery Compression in Patients With Pulmonary

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