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Case Report

Extrinsic compression of left main coronary artery due to pulmonary artery aneurysm and pulmonary hypertension ☆,☆☆

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ABSTRACT

Idiopathic pulmonary arterial hypertension is a serious condition that carries a poor prognosis. While exertional dyspnea is the most common symptom, angina like chest pain, most often due to right ventricle ischemia, may occur at advanced stages. We present a patient with pulmonary hypertension symptomatic for dyspnea and angina in whom computed coronary tomography angiography showed compression of the left main coronary artery by a large pulmonary artery aneurysm. Percutaneous coronary intervention and stenting was performed resulting in significant clinical improvement. This case emphasizes the role of different cardiovascular imaging modalities for the diagnosis of rare conditions.

Introduction

Idiopathic pulmonary arterial hypertension (PAH) is a rare disease affecting 48-55 cases/million adults with an observed high mortality risk in advanced stages [1]. Although the cardinal symptom is progressive dyspnea on exertion, other nonspecific symptoms like fatigue, weakness and syncope may be present. Exertional chest pain, occurring in up to 15.8% of patients, is usually attributed to right ventricle ischemia and not related to atherosclerotic coronary obstructive disease [2].

We describe the case of a patient with PAH and angina secondary to left main coronary artery compression (LMCA) by a

Abbreviations: PAH, Pulmonary Arterial Hypertension; PA, Pulmonary Artery; TTE, Transthoracic Echocardiography; TAPSE, Tricuspid Annular Plane Systolic Excursion; PASP, Pulmonary artery systolic pressure; CCTA, Coronary Computed Tomography Angiography; LMCA, Left Main Coronary Artery; PCI, Percutaneous Coronary Intervention; IVUS, Intravascular Ultrasound.

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large pulmonary artery (PA) aneurysm, highlighting the relevance of different imaging modalities for its diagnosis.

Case report

We present the case of a 57-year-old man, with past medical diagnosis of idiopathic PAH, pulmonary artery aneurysm and permanent atrial fibrillation.

The patient reported previous episodes of syncope (the last episode occurring one year before current medical contact). He complained of dyspnea and angina with mild to moderate exertion, functional class II-III according to New York Heart Association classification. He was on Sildenafil 100 mg per day, Acenocoumarol and Spironolactone 25 mg per day.

On physical examination, the patient was eupneic at rest and his blood pressure was 114/73 mmHg. A first cardiac sound of varying intensity and an enhanced pulmonic second heart sound were audible. There were no other remarkable findings.

Given the patient's clinical history and the reported symptoms, the following differential diagnoses were deemed most likely: atherosclerotic coronary heart disease, extrinsic coronary compression, anomalous origin of coronary arteries and angina like chest pain due to severe pulmonary hypertension.

During the work-up, the following studies were performed:

A 12 lead electrocardiogram showed atrial fibrillation with a heart rate of 60 bpm, QRS axis: +120º, QRS duration: 120 msec. A complete right bundle branch block was present.

Laboratory tests were unremarkable.

A transthoracic Doppler echocardiography (TTE) was requested showing severe right ventricular systolic dysfunction and left ventricular anterior wall hypokinesis. Tricuspid regurgitation was considered moderate to severe and pulmonary artery systolic pressure (PASP) was estimated at 80 mmHg.

A Coronary Computed Tomography Angiography (CCTA) showed severe dilation of right chambers and PA aneurysm with maximum diameter of 80×74.4 mm. There was a significant extrinsic compression of the LMCA by the PA aneurysm (Fig. 1). Main pulmonary branches were dilated. Pulmonary and systemic venous return were normal. No intracardiac shunts were present. PA to aortic diameter ratio was 2.9:1. The angle between LMCA and left sinus of Valsalva was 26°.

Coronary angiography showed LMCA extrinsic compression with significative lumen reduction of the coronary vessel throughout its course. There were no other obstructive coronary lesions (Fig. 2).

Right heart catheterization revealed the following pressures: PA Pressure: 68/33 (mean 45) mmHg, Pulmonary Capillary Pressure: 15 mmHg and Right Atrium Pressure: 8 mmHg. Cardiac Output: 3.7 liters/min.

Based on these findings a dynamic compression of the LMCA secondary to aneurysmal dilation of the PA was confirmed.

The case was discussed among a multidisciplinary team and due to the high estimated surgical risk related to the underlying pathology, an endovascular approach was offered to the patient. Percutaneous coronary intervention (PCI) of the LMCA with a 4.0 × 28 mm drug eluting stent was performed. The procedure was completed with proximal optimization technique with a 4.5 × 12 mm noncompliant balloon (Figs 3 and 4).

The hospital stay was uneventful and the patient was discharged from the institution 24 hours after the procedure on a triple antithrombotic regimen (clopidogrel, aspirin, and acenocoumarol).

Forty days after the procedure the patient remained asymptomatic. A new CCTA showed LMCA stent patency without evidence of external compression (Fig. 5).

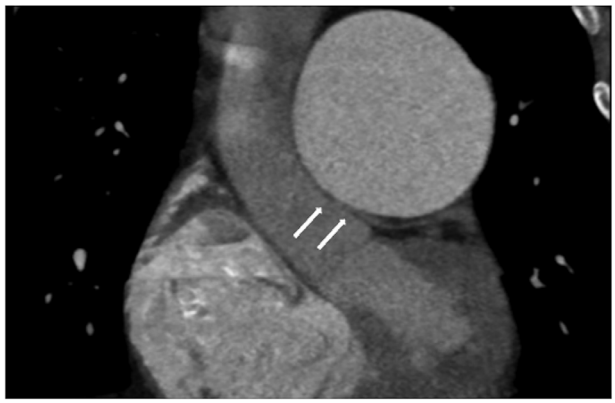

Fig. 1 – Computed tomography. Large PA Aneurysm. White arrows show LMCA external compression by PA aneurysm. LMCA: left main coronary artery; PA: pulmonary artery.

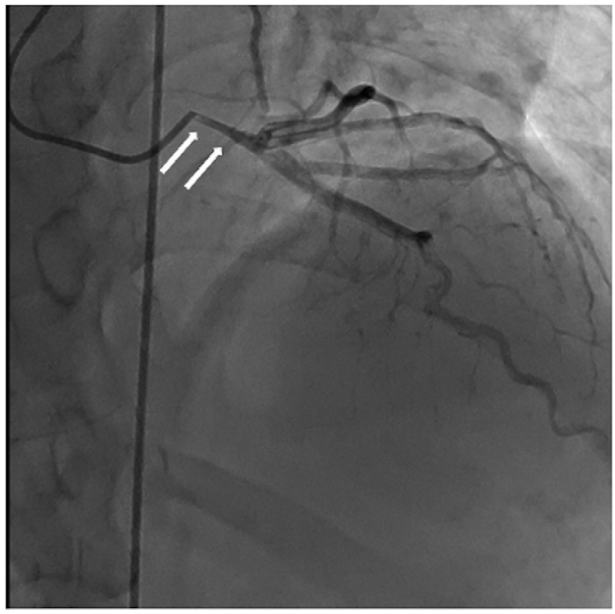

Fig. 2 – Coronary angiogram in anteroposterior cranial view. White arrows show LMCA extrinsic compression with significative lumen reduction. No other coronary obstructions are present. LMCA: left main coronary artery.

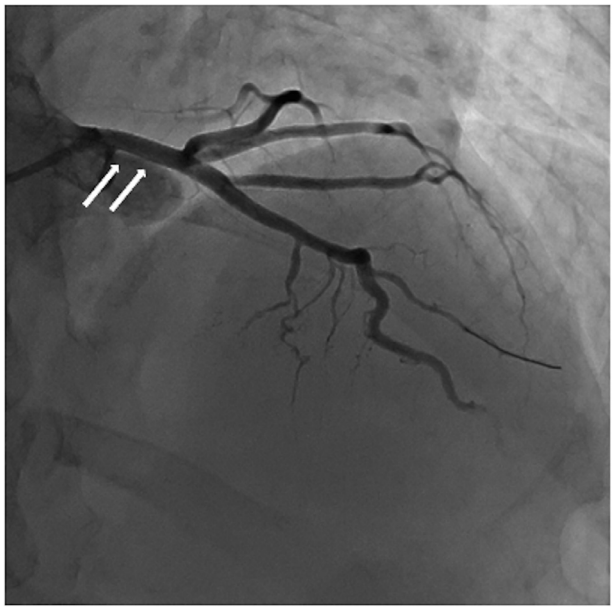

Fig. 3 – Coronary angiogram in anteroposterior cranial view after PCI. White arrows show adequate LMCA stent expansion. LMCA: left main coronary artery; PCI: percutaneous coronary intervention.

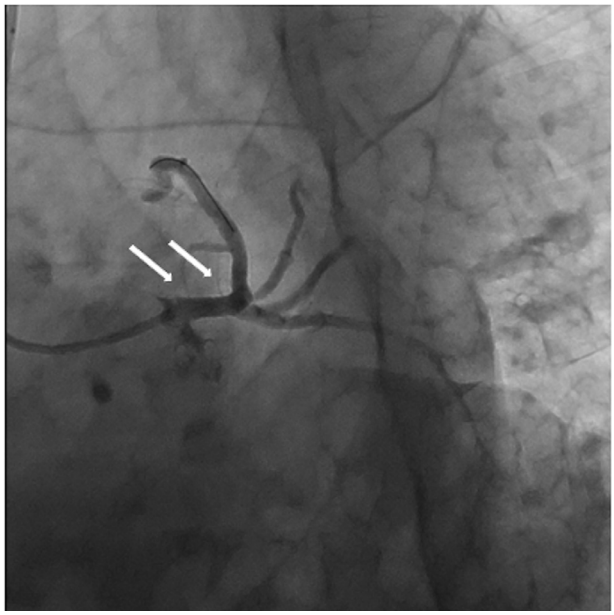

Fig. 4 – Coronary angiogram in anteroposterior caudal view after PCI. White arrows show adequate LMCA stent expansion and no side branch compromise. LMCA: left main coronary artery; PCI: percutaneous coronary angioplasty.

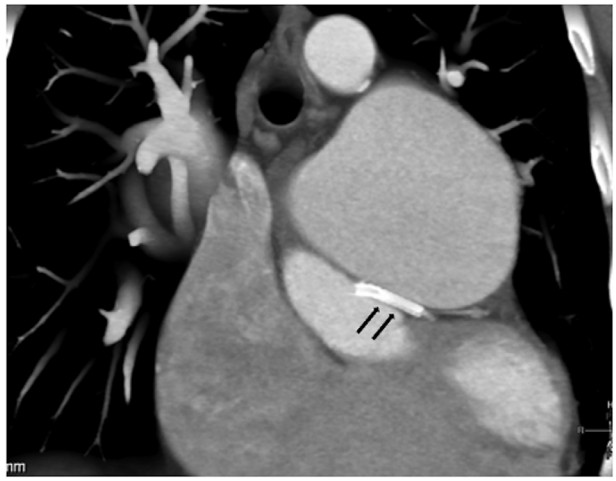

Fig. 5 – Follow-up Computed Tomography. Black arrows show adequate LMCA stent expansion without external compression. LMCA: left main coronary artery.

Discussion

This is a clinical case report of a middle-aged patient with idiopathic PAH and limiting angina secondary to extrinsic obstruction of the LMCA due to aneurysmal dilation of the PA, a rare cause of angina, dyspnea, and left ventricular dysfunction [3–4].

Galiè et al, reported that LMCA compression due to aneurysmal dilation of the PA occurs in up to 40% of patients with PAH and angina-like symptoms, with a higher prevalence in younger patients and females [2].

A thorough clinical evaluation and a high index of diagnostic suspicion are key factors for the achievement of this diagnosis, since patients with PAH generally do not have atherosclerotic coronary heart disease and are unlikely to undergo invasive diagnostic studies [2].

Coronary computed tomography angiography allows an adequate appreciation of the coronary arteries and their anatomical relation with adjacent structures. In addition, various measurements can be made such as PA diameter, PA to aortic diameter ratio and the angle between LMCA and the left sinus of Valsalva. The highest proportion of LMCA compression occurs in patients with a PA diameter >40 mm, PA to aortic ratio >1,5:1 and an angle of the LMCA with the left sinus of Valsalva <30°, the latter being a predictor of significant myocardial ischemia [2,3,5].

The definitive diagnosis of extrinsic LMCA compression is made with invasive coronary angiography. Mainly, an eccentric narrowing from the ostium in a distal direction might be observed. Intravascular ultrasound (IVUS) can be helpful showing a slit-shaped thinning of the LMCA at its ostium without evidence of intravascular damage [6,7].

There are multiple treatment options for this complex pathology including: coronary artery bypass surgery, PCI, congenital defects or valve heart disease surgical correction and lung or cardio-pulmonary transplantation.

Surgical revascularization should be offered to patients with complex coronary anatomy for PCI or in the presence of atherosclerotic/calcified lesions involving more than one epicardial vessel. It should also be considered where there is compression of the LMCA associated with significant valvular or congenital heart disease requiring surgical correction.

Lung or cardiopulmonary transplantation takes on greater relevance in patients with severe lung parenchymal involvement or in those who do not respond to medical treatment of PAH, whether significant biventricular systolic function impairment in present or not.

Percutaneous revascularization is an appropriate strategy to resolve ischemia caused by LMCA compression and should be considered as the first option in patients with anatomic features that might predict a favorable outcome and clinical conditions that pose a high surgical risk. It is a safe option due to the absence of atherosclerotic disease and its ostial location of the LMCA lesion [2].

Intravascular ultrasound is another useful tool for diagnosis and PCI guidance. As these lesions are secondary to compression, IVUS use may be considered optional when treating these patients. Its usefulness lies on the ability to confirm stent apposition and expansion. Similar results are obtained with the use of optical coherence tomography [7].

Drug-eluting stents are highly recommended where atherosclerosis is associated with obstruction in addition to compression. They have lower rates of in-stent restenosis, while short dual antiplatelet therapy might be safe in high bleeding risk patients with newer stent platforms [8].

Conclusions

Compression of the left main coronary artery due to aneurysm of the pulmonary artery and pulmonary hypertension is a rare entity that carries a high morbidity and mortality risk. Its management requires high clinical suspicion and a multidisciplinary approach. Percutaneous coronary angioplasty has shown to be safe and effective for the treatment of this patient.

Patient consent

Written, informed consent was obtained from the patient for publication of this case.

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