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CORONARY ARTERY DISEASE

Original Studies

Percutaneous Intervention of Left Main Coronary Artery Compression by Pulmonary Artery Aneurysm

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Background: Extrinsic compression of the left main coronary artery (LMCA) by a pulmonary artery aneurysm (PAA) has become increasingly recognized as an etiology of angina in patients with pulmonary arterial hypertension (PAH). The purpose of this study was to assess the feasibility and efficacy of LMCA stenting in the treatment LMCA stenosis because of PAA. Methods: Retrospective analysis of data on patients with PAH who presented with angina and underwent percutaneous intervention of their LMCA compression because of PAA was performed. Results: Five patients (age 51 ± 16 years, all female) with PAH presented with angina and underwent LMCA stenting between 2007 and 2009. Four had positive cardiac enzymes. LMCA compression because of a PAA was diagnosed in all patients with cardiac CT angiography after echocardiography demonstrated an enlarged pulmonary artery. LMCA stenting was successfully performed in all patients with resolution of angina and electrocardiographic abnormalities. After a mean follow-up of 16.6 ± 15.7 months (range of 5–39 months), patients remained angina free, no complications of the procedure were noted, and long term stent patency was confirmed in three of the five patients who underwent repeat cardiac CT angiography. Conclusions: LMCA stenting appears to be a feasible and durable option in patients who present with angina because of compression by PAA. This procedure was well tolerated and is of particular value given the increased surgical risk in patients with PAH. © 2010 Wiley-Liss, Inc.

Key words: stenting; pulmonary hypertension

INTRODUCTION

A common symptom in patients with pulmonary arterial hypertension (PAH) is angina, occurring in over 40% of patients [1]. The etiology of angina-like chest pain in this population has been traditionally attributed to right ventricular ischemia because of progressive hypertrophy and dilatation of this chamber [1,2]. Increasingly, left main coronary artery (LMCA) compression by pulmonary artery aneurysm (PAA) is being recognized as an etiology of angina and ischemia in this population, especially given that both sudden death and left ventricular ischemia have been demonstrated to occur in this population [3,4].

The exact incidence of PAA in patients with PAH is unknown. Many cases are asymptomatic and discovered before heart and lung transplant evaluation. However, in one series, in patients with PAH presenting with angina, 33% were found to have compression of the LMCA by a PAA [5] and in small prospective coronary angiography studies, ~40–50% of patients with PAH have been found to have >50% compression of

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Conflict of interest: Nothing to report.

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TABLE I. Clinical Characteristics

Patient No.	Age	Diagnosis	Presentation	PAA diameter (cm)	LMCA % stenosis	Stent type	Follow-up (mo)
1	67	IPAH	UA	6.5	80	BMS	7.3
2	51	IPAH	STEMI and NSVT	11.0	70	BMS	39.4
3	64	IPAH	STEMI and Cardiogenic Shock	4.2	95	BMS	26.5
4	28	ASD&PAH	NSTEMI	4.6	70	BMS	5.1
5	46	ASD&PAH	NSTEMI	5.1	60	DES	4.7

WHO, World Health Organization; IPAH, idiopathic pulmonary arterial hypertension; PAH, pulmonary arterial hypertension; ASD, atrial secundum defect; UA, unstable angina; STEMI, ST segment elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; BMS, bare metal stent; DES, drug eluting stent.

the LMCA [6,7]. The high incidence of LMCA compression is not surprising given the close anatomic relationship of the main pulmonary artery to the LMCA and proximal left anterior descending artery, previously demonstrated in normal subjects using CT angiography [8].

Revascularization with coronary artery bypass surgery has been the traditional approach toward ameliorating left ventricular ischemia. However, if the stenosis is not critical, this approach can be complicated by lack of vein graft patency and internal mammary artery occlusion or atresia because of competing blood flow. Percutaneous intervention and stenting of the LMCA is now increasingly performed as a reasonable and less invasive alternative. Currently, medium and long term follow-up of this procedure in this patient population is not available. The purpose of this study was to assess short-term success and medium to long term follow-up of LMCA stenting in patients with PAH who presented with angina because of extrinsic compression of the LMCA by PAA.

METHODS

Patient population

Data from five patients with PAH, who presented with angina and were found to have PAA causing LMCA compression between June 2006 and October 2009, was reviewed. All patients underwent percutaneous intervention and LMCA stenting for management of left ventricular ischemia and angina. We included one patient in this series that was previously reported from our institution. Review of the patient data was approved by our Institutional Review Board.

Data collection

A detailed retrospective review of medical records was performed for each patient. The etiology of pulmonary hypertension, presentation of angina, immediate and medium to long term follow-up, and procedural complications were analyzed. Significant LMCA compression was defined as >50% stenosis of the LMCA. When available, pre- and postprocedure imaging studies including echocardiography and CT angiography were reviewed.

Procedural Details: LMCA stenting

Informed consent was obtained in all patients. Coronary angiography demonstrated a >50% ostial smooth compression of the LMCA in all five patients. Intraaortic balloon pump was placed in all patients before intervention. Intravascular ultrasound was performed and guided stent selection and postdeployment optimization in all except for one patient, whose procedure was performed at an outside hospital and required emergent intervention given the presence of cardiogenic shock. Except for this patient, all patients also had right heart catheterization performed simultaneously. One patient required 30 ppm of nitric oxide intraprocedurally.

Follow-up

Hospitalizations, outpatient visits, and pre- and postprocedure cardiac imaging studies were used to determine patients' outcomes and complications following their discharge from the hospital.

RESULTS

Clinical characteristics

Patient characteristics and initial presentation are shown in Table I. Five patients [all female, age 51 ± 16 (mean ± std), range 28–67 years] with PAH, who presented with substernal chest pain and shortness of breath, were found to have compression of the LMCA by PAA. Three patients had idiopathic PAH; whereas two had PAH associated with an atrial septal defect. Two patients presented with ST elevation myocardial infarction (STEMI), both with ST elevation in the anterior and anterolateral leads. One of these patients developed cardiogenic shock soon after presenting to the emergency department and was found to be in atrial fibrillation with rapid ventricular response. The other patient with STEMI had multiple episodes of ventricular tachycardia. Two patients presented with ST segment depression in the inferior, anterior, and lateral leads. One patient presented with sinus tachycardia as her sole EKG finding. Four patients had positive cardiac enzymes. All except for one patient presented

TABLE II. Characteristics of Pulmonary Hypertension

Patient No.	Height (cm)	Weight (Kg)	BSA (m^2)	WHO functional class	Duration of PAH	Vasodilator therapy
1	167	53	1.59	2	14 years	Epoprostenol × 10 years, Sildenafil × 4 years
2	154	50	1.48	2	20 years	Trepostinil × 1 year, Sildenafil × 6 years
3	154	52	1.49	3	8 years	Trepostinil × 8 years, Tadalafil × 2 years
4	160	61	1.60	3	15 years	Trepostinil × 2 years, Bosentan × 2 years, sildenafil × 2 years
5	165	85	1.90	3	>1 year	none

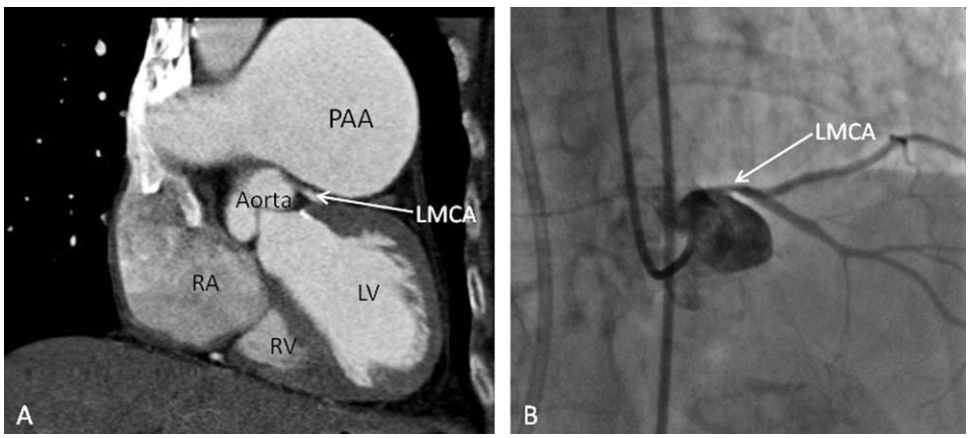

Fig. 1. Significant 80% LMCA compression was shown with cardiac CT angiography (Panel A) and confirmed by coronary angiography (Panel B) in this patient who presented with unstable angina.

with acute onset angina, ranging from 20 min to 2 hr before arrival to the emergency department. The fifth patient had worsening substernal chest pain for 2 weeks and presented with unstable angina. All patients had World Health Organization functional class II or III before presentation. The median height and BSA of patients with LMCA compression in this study was 160 cm (range 154–167 cm) and 1.59 m^2 (range 1.48–1.9 m^2). Four patients were on vasodilator therapy before presentation. The median duration of pulmonary hypertension before presentation was 14 years, Table II. One patient (patient #5) had sought medical care only intermittently and was not diagnosed with pulmonary hypertension till the year before presentation at an outside hospital. Therefore, this patient was not on any vasodilator therapy.

Imaging studies

All patients had echocardiography and four patients had cardiac CT angiography immediately before the procedure. The main pulmonary artery was greater than 4 cm in all patients (mean 6.3 ± 2.8 cm, median 5.1 cm, range 4.2–11 cm). Right ventricular systolic pressure, as documented by echo, was 81 ± 36 mmHg (mean ± std), median of 70 mmHg. Two patients had mild pulmonic valve regurgitation, two had moderate, and one patient had severe pulmonic valve regurgitation.

LMCA stenting

On angiography, the LMCA demonstrated extrinsic ostial compression with inferior displacement of the artery in all patients. Four patients received bare metal stents, whereas one patient received a drug eluting stent. The degree of compression by the PAA ranged from 60 to >90% in the patient who presented with cardiogenic shock. All patients had smooth tapered appearance of their LMCA (Fig. 1) with proximal compression from a superior direction with resultant inferior displacement. All patients underwent successful stenting of their LMCA with prompt resolution of symptoms and electrocardiographic findings (Fig. 2). All patients received a single stent. One patient's procedure was complicated by a right iliac artery dissection, which did not require further intervention. Patients with bare metal stents received aspirin 325 mg and clopidogrel 75 mg daily for at least 1 month. The patient who received a drug eluting stent is continuing on dual antiplatelet therapy (5 months postprocedure, goal is to continue for 1 year).

Follow-up

Immediately postprocedure, resolution of angina occurred in all patients. The patient who presented with cardiogenic shock had successful recovery shortly after LMCA stenting and was discharged home. At a

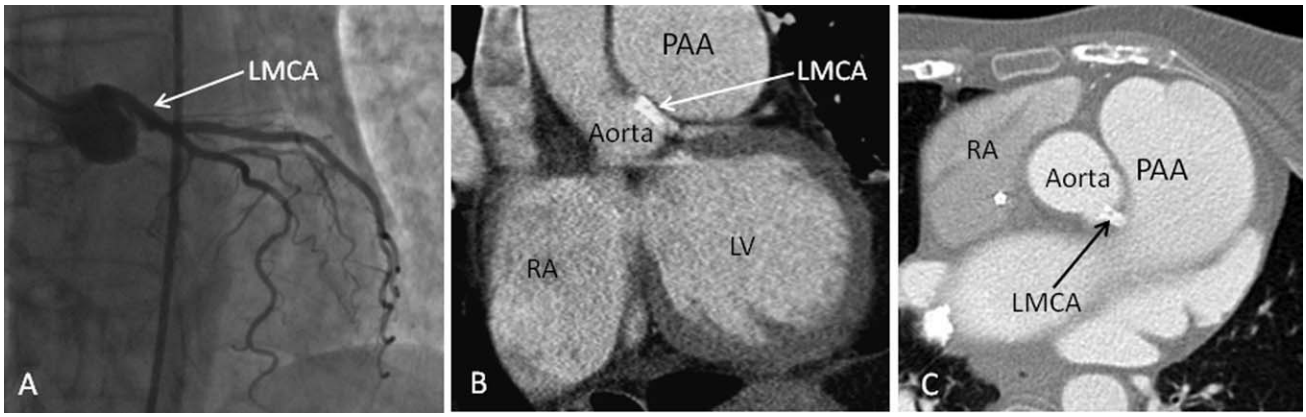

Fig. 2. Immediately postangioplasty and stenting of the LMCA, resolution of the significant 80% lesion shown in Figure 1 is demonstrated. Follow-up cardiac CT angiography in this patient at 3 months post procedure demonstrated a well-positioned patent stent in the LMCA. During this time period, the PAA in this patient had grown from 5.6 to 6.6 cm.

mean follow-up of 16.6 ± 15.7 months (range of 5–39 months), angina did not recur in any patient. Three patients underwent follow-up cardiac CT angiography at 3 months and one patient had follow-up cardiac CT angiography at 3 years poststent placement, all of which demonstrated patent LMCA stent. This was despite a growing PAA from 5.6 cm at initial presentation of angina and intervention to 6.6 cm in one patient at follow-up CT angiography. None of the patients have required any further intervention of their LMCA.

DISCUSSION

This small case series demonstrates that unprotected LMCA stenting is a feasible option for the treatment of extrinsic LMCA compression because of a PAA. Acutely, all patients tolerated the procedure and were successfully discharged from the hospital. This was true even in the case of the patient who presented with cardiogenic shock. Furthermore, the procedure was successfully performed in all patients, regardless of the type of presentation, including STEMI, NSTEMI, and unstable angina. Although current guidelines recommend surgical intervention as first line therapy for these patients, unprotected LMCA stenting is a particularly attractive option, especially given the higher risk associated with surgical intervention in patients with PAH, as the mean right ventricular systolic pressure in this study was 81 ± 36 mmHg (range of 51–134 mmHg, median of 70). Furthermore, in previous studies of percutaneous treatment of focal ostial or shaft LMCA lesions for athersoclerosis, this approach appears safe with a low incidence of restenosis [9].

There have been 10 case reports of patients with PAH presenting with angina because of LMCA compression who were treated with a percutaneous procedure [3,4,8,10–15]. One of these patients was reported previously by our institution [14]. All patients had relief of symptoms and ischemia postprocedure. Although acutely successful, the average follow-up reported in these case reports had been 1 month, with longest angina-free follow-up being 6 months in one case report [15]. The average follow-up in this study was 16.6 months, with the patient with longest follow-up being angina free and without recurrence of left ventricular ischemia for 39 months. The stents in patients, who had follow-up cardiac CT angiography, remained completely patent, even despite a growing aneurysm in one patient.

Four of the patients in this series received bare metal stents, whereas one patient received a drug eluting stent. The choice of stent type was predominantly determined by LMCA diameter (larger diameter vessels receiving bare metal stents), and likelihood of future surgery, including heart and lung transplantation in one patient, which would preclude long term dual antiplatelet therapy with aspirin and clopidogrel. In previous case reports, a variety of stent types were also used, including five patients who received bare metal stents [3,4,15] and four patients who received drug eluting stents (the stent type in one case report was not disclosed) [10–13].

The exact incidence of PAA causing LMCA compression in patients with PAH is not known. In patients with atrial septal defects, the incidence ranges from 4.8 to 44% [7,16,17]. The incidence was 19% in one prospective study, which enrolled 36 consecutive patients with PAH [5]. All patients with LMCA had presented with angina. This is in good agreement with our study, as all the patients in this case series also had PAA diameter greater than 40 mm, with the average PAA diameter being 6.3 ± 2.8 cm. The high incidence of

LMCA compression because of PAA is not surprising given the close anatomic relationship of the pulmonary artery and right ventricular outflow tract to the left coronary system, demonstrated in previous study of normal patients with CT angiography. The right ventricular outflow tract was on average 3.8 ± 1.2 mm from the LMCA at its closest location [8].

The mechanism of LMCA compression and the characteristics of the PAH patients in which it occurs is unclear. It has been hypothesized that the degree of shunting may predispose to PAA formation. The incidence of PAA and LMCA compression is higher in patients with atrial septal defects [5,7,16,17]. Furthermore, interpatient variability in the anatomy of the origin of the LMCA may also explain why certain patients develop LMCA compression and others, with very large aneurysms, do not. It can be hypothesized that a more posterior and leftward origin might be protective. In a multivariate analysis of patients with PAH looking at age, sex, cause of PAH, presence of angina, mean pulmonary artery pressure, pulmonary trunk diameter, and pulmonary trunk to aortic diameter ratio, only pulmonary trunk diameter ($\geq$40mm) and pulmonary trunk to aortic diameter ratio ($\geq$1.21) remained significant characteristics of patients with LMCA compression [5]. A third potential mechanism may be patient stature and diameter of the mediastinum, with patients with smaller stature being predisposed to LMCA compression. The median height of patients with LMCA compression in this study was 160 cm (range 154–167 cm).

LIMITATIONS

This study has important limitations, mainly driven by the small number of patients and the fact that two of these patients did not have follow-up CT angiography to confirm stent patency. However, given the detailed medium to long term clinical follow-up available on these patients, and the fact that they all remained angina free, the lack of follow-up CT angiography is unlikely to be a significant limitation.

CONCLUSIONS

Percutaneous intervention with unprotected LMCA stenting is a feasible option in patients with PAH and LMCA compression due to a PAA, even in the most emergent setting, including cardiogenic shock. In addition to acutely relieving angina and ischemia, this procedure appears to have good medium and long term success, and is particularly attractive given the higher risk of surgery in patients with PAH.

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