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BEGINNER

CASE REPORT: CLINICAL CASE

Reduction of Outflow Tract Obstruction After PCI to Proximal LAD in a Patient With HOCM

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ABSTRACT

We present a patient with hypertrophic obstructive cardiomyopathy and accompanying aortic valve stenosis who had a myocardial infarction with a significant proximal left anterior descending coronary artery stenosis. The primary percutaneous coronary intervention resulted in a notable improvement in the left ventricular outflow tract gradient and global longitudinal strain. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:384–8)

HISTORY OF PRESENTATION

A 72-year-old male patient presented with angina pectoris. Physical examination showed blood pressure of 134/85 mm Hg, heart rate of 98 beats/min, and a crescendo-decrescendo mid-peaking systolic murmur, grade 4, with punctum maximum at Erb's point. Estimated major bleeding risk was 5.8%, and pre-operative cardiac risk was 4.4%. Medical treatment included acetylsalicylic acid, candesartan, and hydrochlorothiazide.

LEARNING OBJECTIVES

- Earlier detection of coronary artery disease should be considered among old patients with HOCM, especially accompanying AVS.
- The loss of a small coronary side branch is a frequent complication of PCI. However, it is rare that this complication involves a coronary SB in a patient with HOCM with a consequent significant reduction in LVOT gradient.
- GLS may improve after regression of LVOT obstruction following septal ablation.

MEDICAL HISTORY

The patient had a known history of hypertension, hyperlipoproteinemia, coronary artery disease treated conservatively, and hypertrophic cardiomyopathy (HCM). He was in New York Heart Association functional class II, without collapse episodes or chest pain. One month earlier, the patient had been diagnosed with B-cell central nervous system lymphoma, with initiation of chemotherapy. Previous electrocardiogram showed no HCM or signs of ischemia. Transthoracic echocardiography (TTE) documented asymmetrical septal hypertrophy of 23 mm with posterior wall thickness of 18 mm, significant chordal systolic anterior motion, left ventricular outflow tract

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(LVOT) obstruction with a dagger-shaped late-peaking pressure gradient (PG) of 132 mm Hg on continuous-wave Doppler, stroke volume index of 35 ml/m^2 with normal ejection fraction (EF) of 58%, reduced average global longitudinal strain (GLS) of −11.6% without restrictive diastolic dysfunction, moderate mitral regurgitation, and moderate to severe aortic valve stenosis (AVS) with significant calcification (**Figure 1**).

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Transcatheter aortic valve replacement was not considered because the severity of AVS was not confirmed as highly relevant. On the basis of the echocardiographic evaluation, the main reason for the elevated PG was the dynamic obstruction of the LVOT due to the hypertrophic obstructive cardiomyopathy (HOCM). Moreover, planimetric quantification of the aortic valve area was unreliable because of significant valve calcification. The presentation with an acute coronary syndrome delayed the transcoronary alcohol ablation of septal hypertrophy procedure, which had been planned to be performed electively.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis was acute coronary syndrome secondary to plaque rupture, embolic phenomenon to coronary vessels, or spontaneous coronary artery dissection.

INVESTIGATIONS

On admission, electrocardiography showed sinus rhythm with ST-segment elevation in leads V_3 and V_4. Other than the left axis deviation, there were no signs HCM. Laboratory studies showed troponin T of 1,010 pg/ml, pro-brain natriuretic peptide of 4,513 pg/ml, creatinine of 1.52 mg/dl, and C-reactive protein of 10.7 mg/l. Immediate coronary angiography indicated a significant stenosis of the proximal left anterior descending coronary artery (LAD) of 90% in addition to a 40% stenosis of the distal LAD (**Figure 2**, Video 1) and a 30% stenosis of the proximal right coronary artery.

MANAGEMENT

Primary percutaneous coronary intervention (PCI) was performed. The proximal LAD lesion was crossed using a Runthrough guidewire (Terumo, Tokyo, Japan) and dilated using a Monorail balloon (Boston Scientific, Natick, Massachusetts), and then a 3.5 × 16 mm drug-eluting stent was implanted, with a very good final angiographic result (TIMI [Thrombolysis In Myocardial Infarction] flow grade 3). Coronary angiography after stent implantation showed occlusion of a septal branch (SB), which arises exactly from the treated segment of the proximal LAD (**Figure 2**, Video 2). Clopidogrel, bisoprolol, and atorvastatin therapy were added to the medical therapy.

FOLLOW-UP

Post-PCI, pro-brain natriuretic peptide was 4,520 pg/ml, and troponin T was 1,113 pg/ml. Post-PCI TTE showed no regional left ventricular (LV) hypokinesia, with preserved EF (60%). Interestingly, the classical LVOT dagger-shaped signal disappeared with a significant reduction of the provoked peak PG to 13 mm Hg on pulsed-wave Doppler. This made it possible to quantify the coexisting AVS using the continuity equation, resulting in an area of 1.2 cm^2 (**Figure 1**). Furthermore, a notable improvement was observed in GLS to −15.6% and in mitral regurgitation to a mild degree. At 4-month follow-up, TTE documented a persistent reduction in LVOT provoked peak PG (11 mm Hg), with more LV systolic function improvement (GLS −18.1%, EF 67%).

DISCUSSION

Generally, adult patients with HCM can develop atherosclerotic coronary artery disease (1). On the other hand, SB occlusion occurs frequently after main vessel stenting, causing a limited myocardial infarction (2). Our patient had an obstructive atherosclerotic lesion in the proximal LAD involving ≥1 SB supplying the first septal unit at the mitral-septal contact level. The acute progressive stenosis along with the drug-eluting stent implantation caused occlusion of one of those SBs. As in transcoronary alcohol ablation of septal hypertrophy (3), this led to a small infarction of the first septal unit, thereby decreasing local contractility and LVOT obstruction.

Average LV longitudinal myocardial systolic function is depressed in HCM, especially in the presence of LV obstruction, and in significant AVS (4,5), despite preserved LV EF. Significant improvement in GLS of the left ventricle has been observed after transcatheter aortic valve replacement and surgical aortic valve replacement (6,7), but not after transcoronary alcohol ablation of septal hypertrophy among

ABBREVIATIONS AND ACRONYMS

AVS = aortic valve stenosis

EF = ejection fraction

GLS = global longitudinal strain

HCM = hypertrophic cardiomyopathy

HOCM = hypertrophic obstructive cardiomyopathy

LAD = left anterior descending coronary artery

LV = left ventricular

LVOT = left ventricular outflow tract

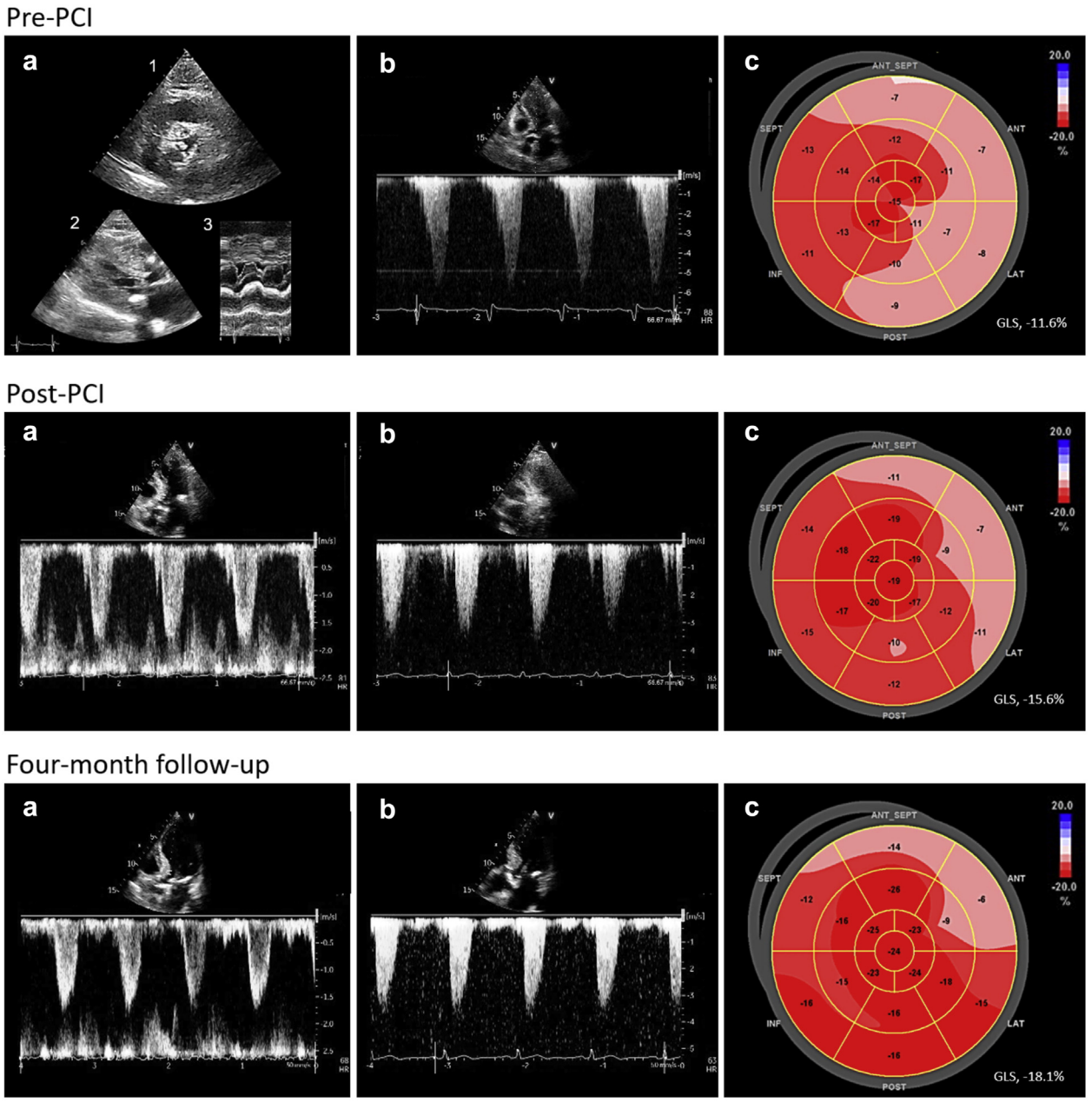
PCI = percutaneous coronary intervention

PG = pressure gradient

SB = septal branch

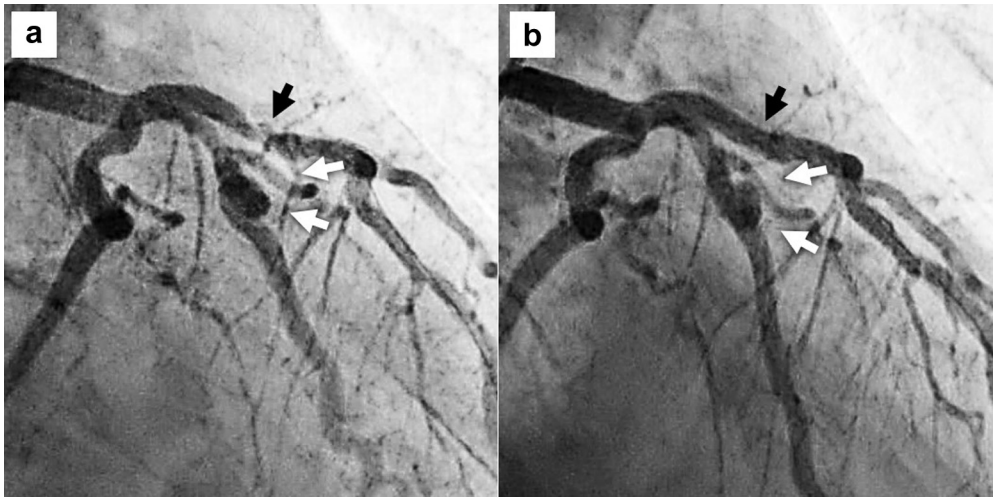
TTE = transthoracic echocardiography

FIGURE 1 Echocardiographic Findings Before and After Stenting PCI to Proximal LAD



(Top row) Pre-percutaneous coronary intervention (PCI) transthoracic echocardiography (TTE) shows significant calcification and stenosis of the aortic valve in the short-axis view **(a1)**, asymmetrical septal hypertrophy and systolic anterior motion of the mitral valve in the parasternal long-axis 2-dimensional view **(a2)**, and M-mode **(a3)**; a dagger-shaped late-peaking pressure gradient (PG) of 132 mm Hg on continuous-wave Doppler **(b)**; and reduced global longitudinal strain (GLS) of −11.6% **(c)**. **(Middle row)** Post-PCI TTE shows left ventricular outflow tract (LVOT) peak PG reduction to 13 mm Hg on pulsed-wave Doppler **(a)**, a residual peak PG of 50 mm Hg on continuous-wave Doppler due to the concomitant aortic valve stenosis with no late-peaking velocity **(b)**, and GLS improvement to −15.6% **(c)**. **(Bottom row)** Four-month follow-up TTE shows a persistent reduction in LVOT obstruction **(a)**, a stable residual peak PG on continuous-wave Doppler due to the aortic valve stenosis **(b)**, and additional improvement in GLS to −18.1% **(c)**.

FIGURE 2 Septal Branch Occlusion Following Proximal LAD Stenting



Angiograms before **(a)** and after **(b)** percutaneous coronary intervention to the proximal left anterior descending coronary artery (LAD) **(black arrows)** show septal branch occlusion of the first septal unit **(white arrows)**.

patients with isolated HOCM (5). In our patient, the significant improvement in GLS of the left ventricle after PCI implies that the poor initial GLS could be explained mainly by the LVOT obstruction by itself and that the LV hypertrophy could not be explained mainly by pure primary myocardial disease but rather by the coexistence of AVS and hypertension. However, because of the diminished symptoms, we did not perform cardiac magnetic resonance imaging or cardiac biopsy, so the exact etiology of the HCM is still unknown.

Concomitant AVS and LVOT obstruction is commonly observed in clinical practice (8). However, it can be challenging to assess AVS severity using the continuity equation because of the LVOT obstruction. In such cases, measuring the subaortic pressure and flow passing through the aortic valve accurately using catheterization can be useful (9). In the present case, the significant improvement of the LVOT obstruction allowed us to determine the severity of the accompanying AVS without the need to perform a hemodynamic study.

CONCLUSIONS

To the best of our knowledge, this is the first report of a patient with HOCM in whom marked regression of LVOT obstruction and improvement of LV function occurred after PCI to the LAD, unmasking the severity of accompanying AVS.

This case report suggests that patients with HOCM and AVS may be more likely to have coronary atherosclerosis, requiring further attention and earlier detection.

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KEY WORDS cardiomyopathy, myocardial infarction, percutaneous coronary intervention

APPENDIX For supplemental videos, please see the online version of this paper.