

Managing Stenotic Septal Perforator Branches

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Coronary artery disease of the septal perforator branches can lead to clinical ischemia and conduction abnormalities. Performing interventional procedures in these vessels is frequently impossible because they are small, which makes it difficult to approach them and to select appropriate equipment. Larger septal perforator branches have been treated percutaneously in a few patients; however, the clinical effectiveness and long-term outcomes are not known. We present our experience in managing obstructive septal perforator branch stenosis in 4 patients. (*Tex Heart Inst J* 2017;44(5):361-5)

Obststructive coronary artery disease (CAD) of the septal perforator branches (SPBs) off the left anterior descending coronary artery (LAD) can be expected in association with CAD of the major epicardial vessels.¹ Atherosclerotic occlusion of a relatively large SPB can lead to ischemia, conduction abnormalities, and arrhythmias.²⁻⁵ Revascularization by means of percutaneous coronary intervention (PCI) to the SPBs can be technically challenging when the SPBs have a caliber <2 mm or a sharply angulated takeoff, because it is difficult to deliver devices of appropriate size. Various approaches to PCI, with or without SPB stenting, have been reported^{1-3,6-10}; however, their clinical effectiveness and long-term outcomes are not known.¹ Existing guidelines lack recommendations for interventions in diseased SPBs. We present our experience in managing SPB stenosis in 4 patients.

Case Reports

Patient 1

Subtotal Occlusion of the First Septal Perforator Branch, Treated with Plain Old Balloon Angioplasty (POBA). A 66-year-old man with a history of hypertension and persistent atrial fibrillation presented with non-ST-segment-elevation myocardial infarction (NSTEMI). His cardiac troponin I level was 2.7 ng/mL (normal, ≤0.03 ng/mL). A 12-lead electrocardiogram (ECG) showed rate-controlled atrial fibrillation without acute ischemic changes. A coronary angiogram revealed 99% discrete stenosis at the origin of the first SPB—thought to be the infarct-related artery (Fig. 1A)—and 80% stenosis in the mid right coronary artery (RCA). The LAD and the left circumflex coronary artery (LCx) had nonobstructive disease. The patient reported chest pain despite medical therapy. Myocardial perfusion images revealed moderate ischemia in the basal and mid inferior septum, which seemed to correspond to the territory of the first SPB.

We engaged the left main coronary artery (LMCA) with use of a 6F Amplatz left 2 guide catheter (Merit Medical Systems, Inc.; S. Jordan, Utah) and crossed the first SPB with a Cougar[®] guidewire (Medtronic, Inc.; Minneapolis, Minn). Prolonged inflations of a 2.25 × 15-mm Euphora[™] Semicompliant Balloon Dilatation Catheter (Medtronic) yielded <50% residual ostial stenosis and enabled Thrombolysis in Myocardial Infarction (TIMI) III flow (Fig. 1B). We deployed a drug-eluting stent (DES) in the mid-RCA lesion. At the patient's one-month evaluation, he was asymptomatic.

Patient 2

Acutely Occluded First Septal Perforator Branch after LAD Intervention, Treated with POBA. A 75-year-old woman with CAD presented with NSTEMI. Three years earlier, DESs had been deployed in her mid LAD and mid RCA. Her cardiac troponin I level was 1 ng/mL. A 12-lead ECG showed ST-segment depression in the anterior leads. Coronary angiograms revealed patent stents; however, 90% stenosis of the mid LAD distal to a prior stent involved the takeoff of the first SPB. This 2-mm ves-

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sel had 50% ostial stenosis (Fig. 2A). We engaged the LMCA with use of a 6F Judkins curved left 4 guide catheter (Cordis, a Johnson & Johnson company; Fremont, Calif), then crossed the LAD lesion with a HI-TORQUE Balance Middleweight Universal Guide Wire (Abbott Vascular; Santa Clara, Calif). Predilation with use of a 2 × 12-mm Emerge® Monorail® PTCA Dilatation Catheter (Boston Scientific Corporation; Natick, Mass) preceded the deployment of a 3-mm DES. The first SPB then became acutely occluded (TIMI 0 flow) (Fig. 2B). The patient had severe chest pain, refractory to systemic analgesics and intracoronary vasodilators.

We decided to open the occluded first SPB, crossing the branch with use of a Runthrough® NS guidewire (Terumo Europe NV; Leuven, Belgium) and performing repeated inflations with a 2 × 12-mm Emerge

Monorail Catheter. After final inflation of the LAD with a 3-mm balloon, TIMI III flow was restored in the LAD and first SPB (Fig. 2C). The patient was discharged from the hospital the next day and was asymptomatic through 18 months of monitoring.

Patient 3

First Septal Perforator Branch after Unsuccessful PCI to the LAD, Treated with Stenting. A 72-year-old man presented with acute-on-chronic systolic heart failure. He had a medical history of myocardial infarction 12 years earlier, associated with ischemic cardiomyopathy and a reduced left ventricular ejection fraction (LVEF) (0.30). Upon presentation, his LVEF was 0.10. Coronary angiograms revealed chronic occlusion of the LAD distal to the origin of the first SPB (Fig. 3A). The distal LAD

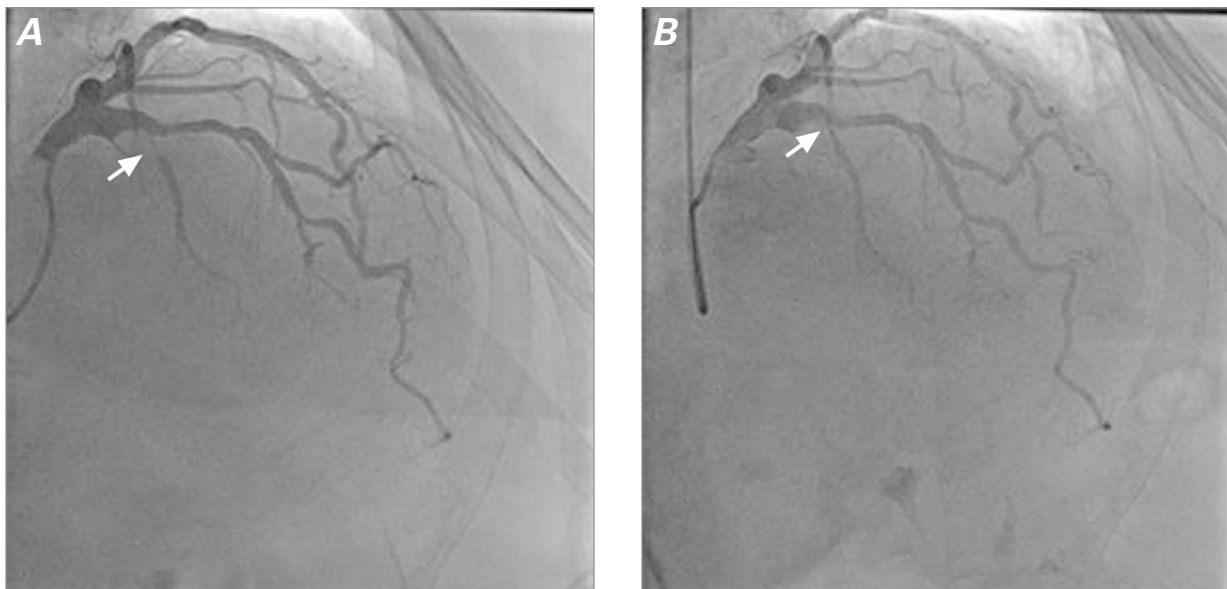


Fig. 1 Patient 1. Coronary angiograms (right anterior oblique views) show **A**) critical ostial stenosis in the first septal perforator branch, and **B**) resolution after balloon angioplasty.

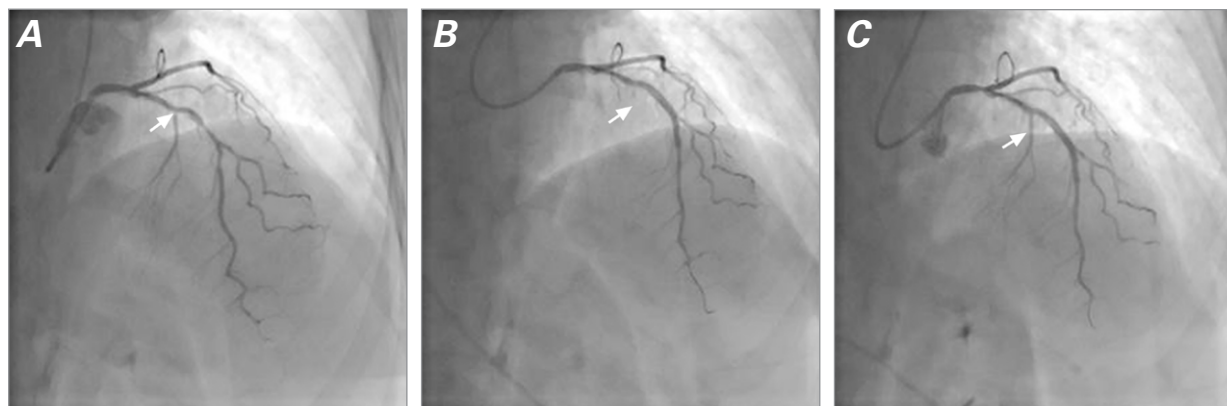


Fig. 2 Patient 2. Coronary angiograms (right anterior oblique views) show **A**) severe stenosis in the mid left anterior descending coronary artery (LAD) and 50% ostial stenosis in the first septal perforator branch (arrow), **B**) acute occlusion of the branch after LAD intervention, and **C**) resolution after balloon angioplasty.

was filled retrograde via left-to-left collateral circulation. The LCx and RCA were also occluded.

The patient declined coronary artery bypass grafting in favor of multivessel PCI. Given the high risk, we used an Impella 2.5® heart pump (ABIOMED Inc.; Danvers, Mass) for circulatory support. We engaged the LMCA by using a 6F Extra Back-Up 3.5 catheter (Medtronic). However, the LAD lesion could not be crossed, and LCx intervention also failed because the proximal vessel was tortuous. We found 80% ostial stenosis in the first SPB, a relatively large vessel that supplied much of the anterior septum and provided substantial collateral circulation to the LAD and LCx. We used a Runthrough

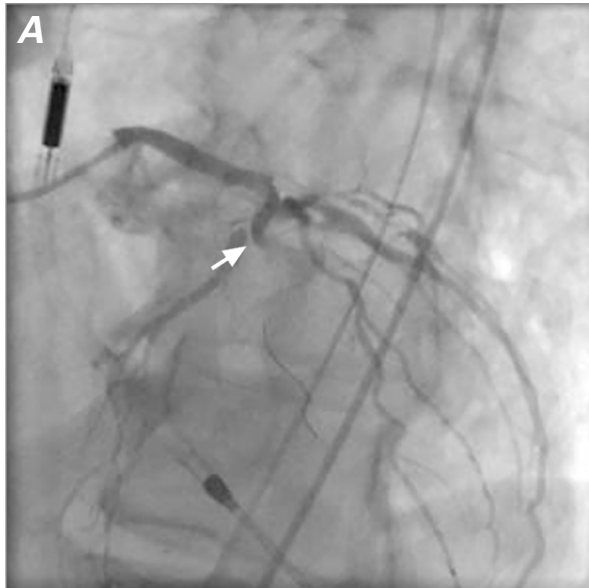


Fig. 3 Patient 3. Coronary angiograms (left anterior oblique views) show **A**) substantial ostial stenosis of the first septal perforator branch, and **B**) resolution after balloon angioplasty.

wire to cross the SPB lesion, a 2 × 20-mm Emerge Catheter for predilation, and a 2.5 × 8-mm Resolute Integrity® stent (Medtronic) to establish TIMI III flow (Fig. 3B). At the patient's 3-month evaluation, his LVEF was 0.30. He remained asymptomatic through one year of monitoring.

Patient 4

Subtotal Ostial Occlusion of the First Septal Perforator Branch, Managed Medically. A 39-year-old man with hyperlipidemia presented with NSTEMI. His cardiac troponin I level was 5.8 ng/mL. No ischemic changes were noted on a 12-lead ECG. A transthoracic echocardiogram revealed a normal LVEF with basal and mid-inferior septal hypokinesia. Coronary angiograms revealed thrombotic subtotal occlusion of the first SPB at the ostium, and TIMI II flow suggested ruptured plaque (Fig. 4). The patient was asymptomatic, so we decided upon evidence-based medical therapy. The patient reported no symptoms during the 2-month monitoring period.

Discussion

The first SPB supplies blood to the atrioventricular node and bundle of His in 50% of the population and is therefore a clinically important vessel.¹¹ Septal perforator branches can harbor collateral circulation for territories of occluded vessels, thereby justifying revascularization of SPBs in selected patients. Percutaneous revascularization of the SPB is rarely performed, because of a lack of supporting evidence. Technically, PCI can be challenging, unless the SPB is of sufficient caliber (>2 mm)

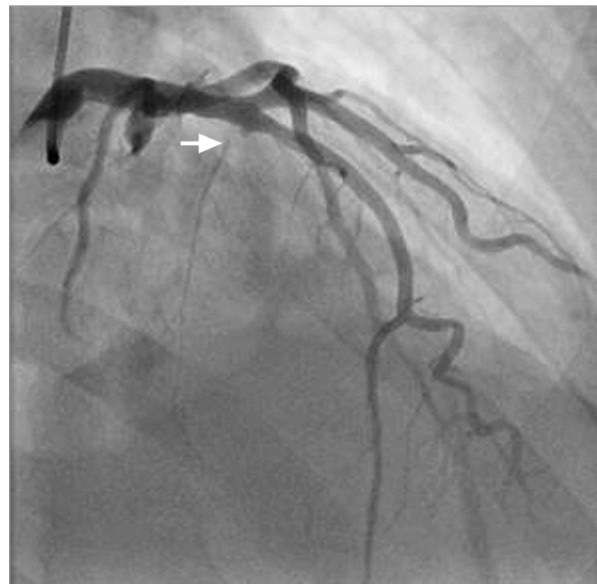


Fig. 4 Patient 4. Coronary angiogram (right anterior oblique view) shows subtotal occlusion of the first septal perforator branch, which exhibits Thrombolysis in Myocardial Infarction II flow.

and has a wide takeoff angle to enable device delivery. Risks of stenting include a high restenosis rate related to small stent size, and stent fracture or myocardial rupture consequent to intramural stenting.

When substantial SPB stenosis is found, several approaches are possible.

Revascularization by Means of POBA. One approach is revascularization by means of POBA.^{1,6-9,12} In the largest relevant study (21 patients),¹ POBA of a large SPB had a 95% success rate. However, other investigators reported that intervention in the SPB can lead to acute vessel occlusion and complete heart block, either immediately or later.^{12,16} Furthermore, SPB atherosclerotic lesions are most often ostial, and treatment solely by means of POBA is associated with restenosis,¹⁷ presumably secondary to elastic recoil.¹⁸ To overcome this risk, a rotational atherectomy technique⁷ can reportedly facilitate angioplasty by debulking the atheromatous plaque and minimizing the effect of elastic recoil. However, there is a risk of slow flow or no reflow, and it can be used only large-caliber SPBs. In our Patient 1, we decided on POBA for 2 reasons: the vessel size precluded our using the smallest available DES (2.25 mm), and substituting a 2-mm bare-metal stent posed an unacceptable risk of restenosis. In Patient 2, we intervened because PCI to the LAD led to acute occlusion of the first SPB and associated severe symptoms.

Revascularization by Means of PCI and Stenting. Another approach, PCI with stenting, prevents immediate elastic recoil and thereby minimizes restenosis. To our knowledge, PCI with SPB stenting has been described only 3 times. In the case of a 61-year-old man who had stable angina,¹⁰ a coronary angiogram showed a high-grade lesion in the mid portion of the 2nd SPB, which supplies much of the anterior intraventricular septum. Deployment of a 2-mm stent established TIMI III flow. In a 42-year-old man with a history of coronary artery bypass grafting who had unstable angina,¹¹ a substantial tandem lesion in a 2.5- to 3-mm first SPB extended to the body of the LAD, which was occluded distal to the origin of the first SPB. A 3-mm stent was deployed. A 52-year-old man with refractory chest pain 48 hours after an acute anteroseptal myocardial infarction was given thrombolytic therapy.³ A coronary angiogram showed isolated 90% stenosis in the proximal portion of a large-caliber first SPB. A 3-mm stent was placed, and the patient was asymptomatic at one year. In our Patient 3, we decided to stent the first SPB after PCI to the LAD had failed. Optimizing the flow in the first SPB was crucial, because it was a source of collateral circulation to the obstructed LAD. Of importance, this approach carries the risk of occluding the LAD during SPB stenting. (Our patient's LAD was already occluded, so this risk did not apply in his case.)

Revascularization by Means of Aspiration Thrombectomy Alone. A third approach, revascularization by means

of aspiration thrombectomy, is rarely used by itself to treat atherosclerotic occlusion of the SPBs. In the case of a 78-year-old woman who had chest pain and ST-segment elevation in leads V₁ and V₂, a coronary angiogram showed acute thrombotic occlusion of a large-caliber first SPB.² Aspiration thrombectomy left 10% to 20% residual stenosis. Of note, neither stenting nor angioplasty was performed, because the patient's chest pain abated, the ST-segment elevation resolved, and TIMI III flow was restored.

Medical Management Only. A fourth approach, medical management alone, can be considered as the first option in selected patients. In our Patient 4, the culprit lesion was an isolated subtotal occlusion of the first SPB. However, given the vessel's small caliber, the absence of symptoms, and no hemodynamic or electrical instability, we anticipated clinical improvement from medical therapy alone.

Conclusion. Despite the lack of evidence to support intervention in the SPBs, we think that PCI—with or without stenting—is feasible in selected patients. Large studies are needed to validate our results and to investigate the impact on long-term outcomes.

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