Septal branch in percutaneous coronary intervention: A strange and rare brew. Author's reply

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DOI: 10.33963/v.kp.97650

Received:
September 25, 2023

Accepted:
September 28, 2023

Early publication date:
September 28, 2023

We wish to express our gratitude to Yalta et al. [1] for showing interest in our case report depicting important consequences of septal branch (SB) occlusion during percutaneous coronary intervention (PCI) on the left anterior descending (LAD) coronary artery [2]. As Yalta et al. remarked, several conduction abnormalities can ensue following SB occlusion. We observed a newly developed complete right-bundle branch block progressing within minutes into a complete atrioventricular (AV) block with wide QRS ventricular rhythm (38/min). In such circumstances, hemodynamic support provided by iVAC 2L is diminished. Indeed, blood pressure dropped to the level of 80/30 mm Hg. In addition, the system provides no support in the event of malignant ventricular arrhythmia, a complication potentially triggered by acute ischemia, bradycardia, and ventricular premature beats. To overcome this, urgent temporary electrostimulation was established via right femoral venous access. As described, restoring SB flow resulted in the resolution of conduction abnormalities and recurrence of narrow QRS with regular AV conduction.

The size of potential myocardial infarction (MI) following SB occlusion is not negligible; however, it was not the main indication for pursuing SB reperfusion. As shown in the accompanying video material, the extensive transseptal collateral network supplying chronic total occlusion (CTO) of the right coronary artery, and considerable remaining SBs would possibly render newly developed MI less substantial. During SB occlusion, the patient reported only slight chest discomfort while consistent ST-segment disturbances could not be observed due to conduction abnormalities and electrostimulation. Although MI of any size may have diminished the potential for recovery in this patient, PCI was continued to avoid deleterious hemodynamic effects of complete bundle branch block and the need for permanent electrostimulation (resynchronization therapy in this case).

Yalta et al. imply that safeguarding SB with a stiff wire may have facilitated further SB intervention in the event of occlusion. We argue against routine SB wiring during LAD PCI. As mentioned in the case report, a favorable take-off angle (approaching 90°), collateral network, and small calibers render SB protection unnecessary. The scarcity of similar cases and the high rate of clinically silent SB occlusion also advocate a more conservative approach. Avoiding double-layer stenting over the SB ostia may be also a prudent strategy (as applied in our case). SBs are usually spared from profuse chronic calcific atherosclerosis [3]. Soft atherosclerotic plaque or thrombus shift was the source of the SB occlusion in our case. In such circumstances, accessing SB should not be challenging with contemporary armamentarium. The main focus of the PCI should be long-term results on the main branch, a goal easily disrupted by a stiff wire in the steeply angled side branch. If side branch balloon dilatation is sufficient to restore the SB flow, we advise against routine balloon kissing dilatation. In any case, and, in particular, if kissing balloon dilatation is inevitable, we strongly suggest employing abundant balloon postdilatation to optimize the main branch stent, as performed in our case. One should also bear in mind the possibility of endothelial damage by a "cheese cutting" effect of the wires placed in septal branches (for example, while retrieving a jailed wire), a phenomenon well described in CTO procedures [4].

Although a strange brew, relevant consequences of SB occlusion during LAD PCI are primarily a rare brew. Every interventional cardiologist should, however, be aware of the potential implications of such an event, reaching much further than the occlusion of any other similarly sized side branch. As for protective strategies, we suggest the "less-is-more" principle with a focus on perfecting the main branch stent scaffolding.

Article information

Conflict of interest: None declared.

Funding: None.

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