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Septal branch in percutaneous coronary intervention: A strange and rare brew.

Author's reply

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We wish to express our gratitude to Yalta et al. [1] for showing the interest for our case report depicting important consequences of septal branch (SB) occlusion during percutaneous coronary intervention (PCI) on left anterior descending (LAD) coronary artery [2]. As Yalta et al. described, several conduction abnormalities can ensue following SB occlusion. We observed newly developed complete right-bundle branch block progressing in a matter of minutes into a complete atrio-ventricular (AV) block with wide QRS ventricular rhythm (38/min). In such circumstances, hemodynamic support provided by iVAC 2L is diminished. Indeed, we observed a drop in blood pressure to a 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, an urgent temporary electrostimulation was established via right femoral venous access. As described, restoring SB flow resulted in resolution of conduction abnormalities and recurrence of narrow QRS with regular AV conduction.

Sizing 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 accompanying video material, abundant 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, patient referred 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 in the pursuit to avoid deleterious hemodynamic effects of complete bundle branch block and the need for permanent electrostimulation (resynchronisation therapy in this case).

Yalta et al. question whether safeguarding SB with a stiff wire may facilitate further SB intervention in the event of occlusion. We argue against routine SB wiring during LAD PCI. As commented in the case report, favourable take-off angle (approaching 90°), collateral network, and small calibres render SB protection unnecessary. Scarcity of similar cases and high rate of clinically silent SB occlusion also advocate more conservative approach. Avoiding double layer stenting over SB ostia may be a prudent strategy as well (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 interventional armament. Main focus of the PCI should be long term result on main branch, a fact easily disrupted by a stiff wire in steep angled side branch. If side branch balloon dilatation is sufficient to restore the SB flow, we suggest against routine balloon kissing dilatation. In any case, and in particular if kissing balloon dilatation is inevitable, we strongly suggest to employ abundant balloon postdilatation to optimize 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 potential implications of such event, reaching much further than the occlusion of any other similarly sized side branch. As for the protective strategies, we suggest “the less is more” principle with the focus on perfecting the main branch stent scaffolding.

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