Right coronary artery patency as a modulator for unprotected left main PCI risk: myth or reality?

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see p. 631

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According to the evolving definition for high-risk percutaneous coronary intervention (PCI) [1], unprotected left-main (ULM) disease is recognized as one of the unfavorable features helping to identify high-risk PCI patients. Randomized trials made it possible to highlight the overall coronary artery tree involvement as a potent modulator of PCI efficacy [2] so that only patients with ULM disease and low (≤22) Synergy Between PCI With Taxus and Cardiac Surgery (SYNTAX) score have a class I recommendation for PCI. Nevertheless, remarkable progress in PCI techniques and devices facilitated offering PCI to many ULM patients with a wide spectrum of anatomic complexity [3].

The experience of interventionalists early started to consider the possible "protective" value of right coronary artery (RCA) flow during ULM PCI. Indeed, considering the large region of jeopardized myocardium in the absence of a patent RCA concern about the possible occurrence of hemodynamic deterioration during ULM PCI is justified.

The SYNTAX score provided numbers (proven to be useful for ULM patients) that are influenced by RCA dominance, RCA patency, and RCA disease pattern. For instance, RCA patency disease by itself does not imply a strong SYNTAX score rise. On the opposite, a long, calcified, chronic total occlusion (CTO) dominant RCA lesion has a major impact on the SYNTAX score.

When assessing the impact of RCA features on ULM PCI, the major focus of the past investigations was on RCA CTO. Of note, complex anatomy and CTO are the most frequent reasons for referring patients to CABG [4]. According to the early publication of Capodanno et al. [5], patients with concomitant LM and RCA disease had higher cardiac mortality after LM PCI (17.7% vs 6.7%; $P = 0.056$) than those without RCA disease. Importantly, mortality in patients with RCA CTO was extremely higher (30% vs 6.7%; $P = 0.015$) in comparison to the patients without RCA CTO. Similarly, Migliorini et al. [6] noticed significantly higher 6-month (12.8% vs 3.6%; $P < 0.02$) and 3-year mortality rates (23.6% vs 10.3%; $P < 0.03$) in patients with RCA CTO than in those without RCA CTO. Moreover, RCA CTO was recognized as an independent predictor of 3-year cardiac mortality (HR, 2.15 [1.02–4.05]; $P = 0.043$). In line with these results, Takagi et al. [7] reported that in patients undergoing ULM PCI, cardiac death rate was higher in the presence of residual RCA CTO (HR, 2.163 [1.018–4.597]; $P = 0.045$) at 1466 days of follow-up. Additionally, they showed that recanalization of RCA CTO significantly improves long-term survival ($P = 0.010$).

In such a context, Skorupski et al. [8] assessed the impact of the absent functional RCA support on prognosis of patients undergoing ULM PCI. They applied an original definition of no "RCA support" which included a broader spectrum of patients, not only with RCA CTO but also with significant stenosis or minor RCA. They concluded that long-term all-cause mortality at a median follow-up of 1149 days did not differ among the groups (23% vs 20%;

$P = 0.37$ in patients without and with RCA support, respectively). Moreover, RCA CTO (found in 14.3% of patients) did not increase all-cause mortality.

How to explain these conflicting results of Skorupski et al. [8] in comparison to the previous retrospective trials/ /registries?

A logical explanation might come from differences in the characteristics of the study population investigated in different studies. Indeed, the relevance of RCA support during ULM PCI is strongly modulated by:

- left ventricular ejection fraction;
- technical complexity of PCI on the left system;
- clinical conditions.

In these regards, the study population enrolled by Skorupski et al. [8] was characterized by a favorable combination of high ejection fraction (mean value around 55%), low SYNTAX score (mean value 21), and low incidence of 3-vessel disease (7.5%). Furthermore, most of the patients were stable and EuroSCORE II (a strong predictor of adverse clinical outcome after PCI as previously reported) [9] was as low as 1.45%. In other words, RCA support failed to impact the outcome of PCI in a "selected" subgroup of ULM patients exhibiting low risk of both acute hemodynamic compromise and late adverse outcome, as compared with other studies. Consequently, the reported findings cannot translate to other patient subsets.

Another important issue is related to the size of RCA (ranging between super-dominant and recessive) and the type of eventually present coronary lesions (ranging between plaques with borderline hemodynamic significance to sub-occlusions and collateralized chronic total occlusions). Skorupski et al. [8] tried to address this issue but the three categories they applied to RCA lesions (recessive, significant stenosis and CTO) cannot entirely describe the relevance of hemodynamic support provided during ULM PCI. According to a recent study [10], in patients with ULM PCI, the performance of PCI on significant (>70%) RCA stenosis during the same hospitalization might reduce 30-day cardiovascular death. All together, these observations call for patient-to-patient decisions which should take into account the feasibility of achieving reasonable levels of revascularization completeness (not to leave unrevascularized stenoses supplying large areas of ischemic myocardium [11]).

As a final remark, it is crucial to highlight the possibility to deal with patients exhibiting extreme challenges like complex, calcific ULM bifurcation disease and very low ejection fraction. In these circumstances:

- a patent RCA can provide minor support so that mechanical circulatory support can be considered to increase procedure safety (moving from high risk to "protected" PCI [12]);
- untreated proximal lesion in a large RCA may imply large residual jeopardized myocardium resulting in impaired late outcome despite successful protected PCI [13].

Article information

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