

# The simpler, the better: culprit-only intervention is beneficial in patients with chronic kidney disease with concurrent acute myocardial infarction and multivessel disease

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Multivessel disease (MVD) in patients with ST-elevation acute myocardial infarction (STEMI) is not infrequent, occurring in up to 65% of such patients and leading to poor clinical outcomes [1,2]. Current guidelines recommend that primary percutaneous coronary intervention (PCI) should be performed solely for the culprit vessels and not in non-infarct-related arteries if the patient is hemodynamically stable [3,4]. Recent meta-analyses support these guidelines [5,6].

The rationale for these guidelines is that patients with acute myocardial infarction (AMI) are at the center of a full-blown inflammatory, thrombotic milieu and have a high likelihood of dehydration and renal dysfunction. Moreover, they are at risk of suppurative failure and complications during PCI of non-culprit coronary arteries.

The article by Park et al. [7] in this issue extends this thought to patients with concurrent AMI and MVD in the setting of renal insufficiency (RI). As stated by the authors, few studies have addressed this specific group of patients. This is surprising and interesting when we consider that patients

with renal impairment have the highest risk of cardiovascular morbidity and mortality.

The authors demonstrate that total revascularization in patients with AMI and MVD plus RI during the index hospitalization confers no benefit; rather, it may induce harm in the short term. These results are in concordance with previous studies of patients with concurrent AMI and MVD irrespective of their kidney function.

These results are easily anticipated, but, as mentioned above, few studies have addressed this issue. Therefore, this paper has great clinical value and implications when we consider the high prevalence of renal failure in patients with AMI.

As noted by the authors, their study has some limitations. First, this study may be biased by the selection of sicker patients in the MVD group despite the subgroup analysis according to shock status. Propensity matching for unbalanced baseline parameters could have been a partial solution. The second limitation is that the time point of non-culprit revascularization was not definitively addressed in the article. The authors defined multivessel revascularization as performance of

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this procedure during hospitalization with no detailed classification according to time points (i.e., during the index procedure versus delayed PCI after the index procedure during admission). Third, the authors did not clearly classify AMI by ST-elevation status. Most previous studies of culprit-only versus total revascularization in patients with MVD involved patients with STEMI. No studies have involved patients with non-ST-elevation AMI. Therefore, the patients in this article are heterogeneous with respect to the mixing of patients with and without STEMI. The use of classified reports might have raised the quality of this article. Finally, the follow-up period is relatively short. Long-term data could have altered the results.

Most of these limitations are inevitable when we consider that the study results were obtained from retrospective data. Although it has some shortcomings, this study has value in its first look at this subset of fragile patients with renal impairment.

**Conflict of interest**

No potential conflict of interest relevant to this article was reported.

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