EDITORIAL COMMENT

Percutaneous Coronary Revascularization for Myocardial Infarction in Chronic Kidney Disease

Recognizing the Risk While Seizing the Benefits*

Patricia J. M. Best, MD
Rochester, Minnesota

Chronic kidney disease (CKD) with an estimated glomerular filtration rate <60 ml/min is seen in over 30% of patients undergoing percutaneous coronary intervention (PCI) (1). The prevalence of CKD continues to rise, in part because of increasing diabetes and hypertension (2). In patients undergoing PCI for either stable coronary artery disease or acute myocardial infarction, CKD is one of the strongest risk factors for short- and long-term mortality (1,3).

See page 1002

PCI is an important modality of reperfusion therapy in ST-segment elevation myocardial infarction. When CKD is present, regardless of the increased risk of death from myocardial infarction, PCI is underutilized. Although the reason is not entirely clear, it likely reflects the comorbidities of the population (4) as well as the desire to avoid the consequential and increased risks of PCI complications, such as bleeding and contrast-induced nephropathy. In the Global Registry of Acute Coronary Events study, primary PCI compared with fibrinolysis is associated with a similar reduction in mortality in patients with normal renal function and those with moderate CKD (5). Additional ST-segment elevation myocardial infarction studies in CKD patients have shown mortality reduction with reperfusion by fibrinolysis or PCI (6,7).

In non–ST-segment elevation myocardial infarction, the benefit of revascularization in the CKD patient has not been established. The study by Hanna et al. (8) in this issue of JACC: Cardiovascular Interventions is one of the first to evaluate CKD in non–ST-segment elevation myocardial infarction patients only. Patients were evaluated from the National Cardiovascular Data Registry (NCDR)—Acute Coronary Treatment and Intervention Outcomes Network—Get With The Guidelines (ACTION–GWTG) registry. This study excluded patients not undergoing PCI to decrease the impact of the sickest patients with CKD not being treated with PCI. They found that CKD was a strong predictor of in-hospital mortality, with an odds ratio of 2.0 at Stage 3 CKD compared with those with normal renal function, and increasing to 2.8 in those with Stage 4 CKD. Additionally, as has been seen with other studies (6,9), CKD patients received less standard medical therapy within the first 24 h, including aspirin, clopidogrel, statin therapy, and beta-blockers. In this registry, even with the higher use of bivalirudin in the CKD patients, they still had a 2.8-fold increased risk of major bleeding in the Stage 4 CKD group compared with those with normal renal function.

CKD has been well established as a risk for bleeding complications following PCI and is associated with a graded risk of bleeding over 4 times higher in those with a glomerular filtration rate <30 ml/min compared with those >75 ml/min (10). Given the baseline anemia frequently seen in this population, the ability to tolerate bleeding events may be compromised. In all patients, regardless of renal function, bleeding after PCI is associated with increased mortality (11). Bivalirudin reduces bleeding complications associated with PCI, particularly in those with CKD (12–14). Vascular closure devices or a radial access approach may further reduce bleeding events.

Another important complication associated with CKD is contrast-induced nephropathy. This risk is heightened in patients with an acute myocardial infarction. Contrast-induced nephropathy is a strong and powerful predictor of in-hospital and long-term major adverse cardiovascular events and mortality (15). Although multiple mechanisms to reduce this risk have been tried, hydration and limiting the contrast dose are most effective. Still, the risk of contrast-induced nephropathy may inhibit the appropriate use of coronary angiography and percutaneous coronary revascularization in CKD patients likely to benefit.

Restrained use of PCI in the CKD population has also stemmed from the high restenosis risk seen in the balloon angioplasty era with renal failure patients. Bare-metal stenting in mild and moderate CKD is not associated with increased restenosis (16). With drug-eluting stents in those with nondialysis-dependent CKD, restenosis is not increased compared with those with normal renal function (17,18). However, hemodialysis remains an important risk for restenosis, even using drug-eluting stents. Adding to the complexity in managing the bleeding risk in the CKD patients is that even after a successful PCI, CKD is associated with increased stent thrombosis.

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From the Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota. Dr. Best has reported that she has no relationships relevant to the contents of this paper to disclose.
We will continue to frequently see patients with CKD and an acute myocardial infarction. The paucity of data in this population is quite concerning, given that special characteristics exist, such as altered platelet function leading to increased bleeding and thrombotic risk, and increased inflammation. The study by Hanna et al. (8) is an important initial step into the understanding of outcomes in this population. Further studies will be needed to better define the best adjuvant therapy with PCI and to look for therapies specific and targeted to mechanisms of disease progression associated with CKD.

Reprint requests and correspondence: Dr. Patricia Best, Division of Cardiovascular Diseases, Mayo Clinic, Gonda 5, 200 First Street South West, Rochester, Minnesota 55905. E-mail: best.patricia@mayo.edu.

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