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Paulette S. Wehner

Marshall University, wehner@marshall.edu

William A. Nitardy

Marshall University, nitardy1@marshall.edu

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Chronic Kidney Disease and Acute Myocardial Infarction: The Story After 1 Year

Paulette Wehner, MD, FACC, FCCP, FACP, FAHA; William Nitardy, MD

When chronic kidney disease (CKD) is part of the clinical history for a patient with acute myocardial infarction, the interventional cardiologist experiences an increased anxiety level. An acute myocardial infarction with renal disease requires more attention to dye load and fluid status,¹ and general opinion exists regarding the negative outcomes of these “sicker” patients.²

In this issue of the *Journal of the American Heart Association*, Mark Navarro and colleagues³ completed a thorough study from a different angle. We know the interventionists’ concerns are justified acutely as these CKD patients have a higher level of inpatient complications,⁴ but these authors chose to look at the patients 1 year from their event to determine if a relationship existed between CKD and the patients’ health status. In the current climate of patient-centered care and outcomes, the study is very timely.

The investigators chose to use the Seattle Angina Questionnaire and Short Form-12 to collect data in a prospective manner.³ The Seattle Angina Questionnaire assesses 5 domains of coronary artery disease health status. The quality of life and angina frequency were used in the analysis. The Short Form-12 is a 12-item survey of physical and mental functioning. These score differences are related to the age-specific mean score, with higher scores indicating better health status. The cohort was 3617 patients with 16% of these having CKD. In summary, the investigators looked at angina frequency, quality of life, and overall functional status

in CKD and non-CKD patients 1 year from their coronary event.³

Briefly, the investigators used the Translational Research Investigating Underlying disparities in acute Myocardial infarction Patients’ Health status registry⁵ and had the patients complete the surveys at 1, 6, and 12 months. The analysis was completed by using patients with an initial glomerular filtration rate assessment, a baseline health status assessment, and ≥ 1 follow-up assessment. The highest glomerular filtration rate was used to define CKD, which would bring one to the possibility of the acute event contributing to a high glomerular filtration rate, not truly meeting a criterion for CKD, but this should not negate the outcomes.

The results are somewhat contradictory to what has been reported previously. In the *American Heart Journal* article “The prognostic importance of worsening renal function during an acute myocardial infarction on long-term mortality” by Amin and colleagues,⁶ worsening renal function was found to be independently associated with an adverse long-term prognosis. These patients were followed for 4 years and they followed patients with worsening of renal failure during the acute event. Worsening of renal failure was found to be independently associated with a higher risk of death.

In the study by Navarro, CKD does not seem to impact the ultimate quality of life 1 year removed from the myocardial infarction. Specifically, angina was not more frequent in the CKD patients, mental functioning was similar regardless of whether CKD existed, and the difference in physical functioning was not statistically significant.³

In the studied cohort, there is some positive data regarding CKD patients 1 year removed from their myocardial infarction. The study is important clinically because these patients should not be denied such proven interventions as cardiac rehabilitation, cardiac support groups, and dietary counseling. The CKD patient now should not be considered “too sick” to benefit from these critical interventions. The authors are to be applauded for changing, we hope, how the patient with CKD is approached post myocardial infarction!

Now for the bad news. Why would this patient cohort have lower discharge medication rates of aspirin, statin medications, and angiotensin-converting enzyme inhibitors? Previous studies have shown the underuse of guideline-directed

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From the Departments of Cardiovascular Medicine (P.W., W.N.) and Internal Medicine (P.W., W.N.), Marshall University Joan C. Edwards School of Medicine, Huntington, WV.

Correspondence to: Paulette Wehner, MD, FACC, FCCP, FACP, FAHA, Marshall University Joan C. Edwards School of Medicine, Cardiovascular Medicine/Office of GME, 1600 Medical Center Dr, Suite 2582, Huntington, WV 25701. E-mail: wehner@marshall.edu

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therapy in the CKD patient^{7,8}; sadly, this was confirmed. The concern of the elevated creatinine being caused by the acute event comes into play again. Physicians may be hesitant to start the angiotensin-converting enzyme inhibitor if they perceive the renal injury is acute. The high-dose statin is recommended for secondary prevention with no caveat regarding CKD. In a meta-analysis by Nikolic,⁹ a conclusion was proposed that statins may actually be protective to the renal function in CKD patients not receiving dialysis, but the benefit seemed to depend on duration of treatment. Finally, the lack of use of aspirin is truly a bewilderment. Platelet dysfunction in the uremic patient is a well-known clinical syndrome but should not be a contributor to the lack of aspirin use in a post-myocardial infarction patient. Many of these patients should actually be taking dual antiplatelet therapy with no contraindications in the CKD patients.¹⁰ To all who read this, medical students, residents, fellows, and attending physicians, the message is clear. Follow the guidelines!¹

In summary, the study's authors should be commended for looking at the entire picture of the CKD patient with acute myocardial infarction and the clinical status 1 year post myocardial infarction. These patients did as well as their non-CKD counterparts from an angina and quality of life standpoint. The outcomes of the study are significant in how these patients are perceived and, we hope, treated. These data reinforce the need to follow guideline-directed therapy in these patients. The burning question remains: Why the difference in aspirin, statin, and angiotensin-converting enzyme inhibitor use in the CKD patient post myocardial infarction? The lack of use of these therapies did not make sense before this study and certainly even less so now.

Disclosures

None.

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