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Acute kidney injury in acute myocardial infarction — A never-ending story?

Yacov Shacham *

Department of Cardiology, Tel-Aviv Sourasky Medical Center affiliated to the Sackler Faculty of Medicine, Tel-Aviv University, Tel-Aviv, Israel

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The heart and the kidneys are linked by complex and bilateral interactions as diseases affecting these two organs share many common and overlapping risk factors.

Among patients with acute myocardial infarction (AMI), especially those undergoing percutaneous coronary intervention (PCI), acute kidney injury (AKI) is frequent and associated with adverse outcomes [1–3]. Over the past few decades, immense progress has been made in both interventional and medical treatment of AMI. In stark contrast, during the same time frame, there have been minimal therapeutic advances in the prevention of AKI. Both prevention and management of AKI can be thus considered as a rate-limiting step to improving outcomes in patients with AMI.

Worsening of renal function throughout hospitalization in AMI patients is multifactorial, though the most important reason is considered contrast induced AKI, related mainly to the amount and type of contrast material and to preprocedural renal function [4]. Growing amount of data now suggests that AKI, in this clinical scenario, has a complex and multifactorial pathogenesis which goes beyond the administration of contrast material during catheterization. These factors include an adverse hemodynamic state resulting in reduced renal perfusion, other metabolic factors such as drugs admitted (especially blockers of the renin-angiotensin axis) as well as the occurrence in parallel of sepsis, bleeding, atheroembolic disease and acute hyperglycemia [5].

Previous studies on AKI in AMI focused mainly on renal dysfunction shortly after PCI, usually within the first 48 to 72. In contrast, only limited data is present on the clinical implications of renal dysfunction persisting beyond hospital discharge. Moreover while mortality was the usual end point assessed following AKI, the actual effect on long term renal function has been somehow negated.

In this issue of the "*International Journal of Cardiology*" Chalikias et al. present data on the long-term prognostic implications of in-hospital AKI in 518 AMI patients [6]. Baseline kidney function was assessed at hospital admission, and AKI was determined based on the rise in the serum creatinine level. The occurrence of AKI was evaluated twice during hospitalization: at 48 h after admission, using the Acute Kidney Injury Network (AKIN) and Risk, Injury and Failure (RIFLE) criteria. In addition, AKI was assessed at discharge using both the RIFLE and the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. The incidence of AKI in the study population ranged from 7% to 16% depending on the time (at 48 h vs. any during hospitalization), and on definition used. Patients were followed up for a median of 5.6 years for the occurrence of death, major adverse cardiovascular events and any deterioration in kidney function.

The results emphasized 2 important points. First, patients with AKI had a three-fold increased mortality risk evident for up to year three of follow-up, independent of possible confounders. A recent report also demonstrated that among patients with AMI even small elevation of serum creatinine (0.1–0.3 mf/dl), not fulfilling the criteria for AKI, was still independently associated with adverse outcomes [7]. Clinical AKI is diagnosed when renal damage and dysfunction reach a threshold sufficient to make serum creatinine rise above 0.3 mg/dl. Such level of renal damage becomes evident only after the structure and function of nephrons that are part of the so-called renal functional reserve are affected. Given these findings, even a minor change in serum creatinine following AMI may be used to identify a group of patients with early stages of renal dysfunction. As serum creatinine is considered to be a relatively late biomarker for AKI, several novel biomarkers have been recently developed to improve the sensitivity and specificity of the early diagnosis of AKI as well. In a recent ADQI consensus, a new perspective has been suggested for the diagnosis of AKI, including a new category of kidney disorders defined by positivity of damage biomarkers and negativity of serum creatinine (kidney attack or subclinical AKI) [8].

The second important point relates to the long term renal outcomes of patients developing AKI following AMI. In the cohort of Chalikias and colleagues, 10% of patients with AKI had further deterioration of renal function during long term follow up. This finding is in line with recent report which demonstrated that among AMI patients who developed AKI nearly 40% failed to normalize renal function at hospital discharge [9]. These patients were likely to develop new chronic kidney disease (CKD)/worsening of present CKD.

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* Department of Cardiology, Tel Aviv Sourasky Medical Center, 6 Weizman Street, Tel Aviv 64239, Israel.
E-mail address: kobyshacham@gmail.com.

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The relation between AKI and potential progression to CKD has been previously suggested [10]. This association is complex in a way that new CKD may represent non-resolution from previous AKI, gradual development of CKD following resolution from AKI or progression of pre-existing CKD. Risk factors for lack of recovery after AKI include advanced age, higher AKI stage, baseline CKD and need for renal replacement therapy. It appears that the occurrence of AKI, especially with lack of recovery at hospital discharge, identifies population at high risk for adverse long term renal outcomes. Defining optimal follow up care for these patients is therefore essential, especially after hospital discharge, when recovery from AKI and its underlying precipitants might have not resolved completely. Questions may also rise regarding the routine administration of Renin/Angiotensin blockers in AMI patients with AKI. This highlights that patients with AKI probably warrant referral for a nephrologist follow- up and frequent assessment of renal function.

A few limitations of the present study require mention. Long-term follow-up was performed in only about 2/3 of the initial study population, which may have influenced the results. No information was present on serum creatinine levels post discharge, and as such assessment of long term deterioration in kidney function could have not been assessed accurately. Similarly the consensus definition of CKD (admission eGFR ≤60 ml/min/1.73 m^2) was not utilized, thus true presence of CKD at admission could have not been assessed.

In conclusion, we should congratulate Chalikias et al. for pointing an important, yet often forgotten complication. The challenge for future physicians will be to address the need for appropriate involvement of renal services during index hospitalization of such patients and appropriately targeted follow-up.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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