

CKD after AKI: time to be alert

DRC após IRA: momento para estar alerta

Authors

Miguel Luis Graciano ¹

¹ Faculdade de Medicina,
Universidade Federal
Fluminense.

Along decades Acute Renal Injury (AKI) epidemiology has been evolving steadily from a syndrome related to renal perfusion in younger patients to the current stage of renal compromise commonly triggered by inflammation in the elderly. Presently, the typical AKI patient harbors multiple morbidities and reduced renal functional reserve conferred by several different chronic insults like diabetes, hypertension, age and ischemia.¹

It is now established knowledge that renal damage caused by AKI might result in chronic renal functional loss or acceleration of this process if it is already in course.^{2,3} Accordingly, AKI should not be viewed anymore as a benign clinical entity that brings with clinical resolution full restoration of renal capabilities. The fact that now many survive this still lethal disease, despite the present intensive care environment of complex and severe medical and surgical conditions where it occurs, is for sure a reason for celebration. However, we must deal with the consequences of the acute renal aggression on the long term renal outcome in the survivors.

It is more than welcome, therefore, the paper entitled “*Risk factors for the progression of chronic kidney disease after acute kidney injury*” published in this edition of *the Brazilian Journal of Nephrology* by Pereira *et al.*⁴ The authors assessed a databank containing information on 1200 patients treated in a public hospital in São Paulo focusing on the survivors of AKI episodes and retrospectively evaluating clinical risk factors associated with renal outcome and death after discharge.

It should be noted that from the original population of 1200 patients, 993 (83%) were excluded for meeting the exclusion criteria (death before discharge or CKD stage 5). The observation that patients admitted to hospital wards progress more than those admitted to the ICU should be viewed in this context. Accordingly, only healthier patients with milder forms of systemic disease may have survived the admission to the ICU and the others have never had the opportunity to survive and progress to chronic kidney disease (CKD). This may constitute a prevalence/incidence (Neyman) bias that was properly recognized by the authors themselves.

The authors depicted that mechanical ventilation, need for dialysis and septic shock had no correlation with progression of CKD. However, they observed that progression was more common in patients with sepsis, cancer or urinary tract obstruction. The observation that sepsis but not septic shock correlates with CKD progression might again be related to a high mortality rate in the ICU. Unfortunately, the impact of previous renal dysfunction on progression was not mentioned.

The observation that urinary obstruction is related to CKD progression is interesting and has been observed both in the clinical and experimental settings.⁵ The intense interstitial fibrosis conferred by the obstruction may provide the pathophysiological link between AKI and CKD. Indeed, early signs of fibrosis and collagen deposition has been detected in other models of AKI.⁶ More recently, experimental results bring the possibility of

Submitted on: 6/14/2017.
Approved on: 6/14/2017.

Correspondence to:

Miguel Luis Graciano.
Faculdade de Medicina
Universidade Federal
Fluminense.
Av. Atlantica, 3170, Rio de
Janeiro, RJ, Brazil.
CEP: 22070-000
E-mail: mgraciano@id.uff.br

DOI: 10.5935/0101-2800.20170045

different mechanisms linking acute and chronic renal events, such as mitochondrial damage which mediates and amplifies innate immune responses.⁷

Finally, Pereira *et al.* described the switching among different CKD stages after discharge that clearly shows the need of nephrology consultation during the follow up of AKI survivors. This is a very important message that calls for our attention concerning the care of AKI patients after AKI itself. In the era of electronic alerts designed for real time identification of AKI episodes the recognition of the impact of such episodes on the course of CKD may be even more important. Accordingly, it has been shown recently that AKI diagnosed by such E-alerts may correlate with mortality and renal outcome.⁸

REFERENCES

1. Cerdá J, Lameire N, Eggers P, Pannu N, Uchino S, Wang H, et al. Epidemiology of acute kidney injury. *Clin J Am Soc Nephrol* 2008;3:881-6. DOI: <http://dx.doi.org/10.2215/CJN.04961107>
2. Coca SG, Singanamala S, Parikh CR. Chronic kidney disease after acute kidney injury: a systematic review and meta-analysis. *Kidney Int* 2012;81:442-8.
3. Hsu CY, Ordoñez JD, Chertow GM, Fan D, McCulloch CE, Go AS. The risk of acute renal failure in patients with chronic kidney disease. *Kidney Int* 2008;74:101-7.
4. Pereira BJ, Barreto S, Gentil T, Assis LS, Soeiro EMD, Isac de Castro I, et al. Risk factors for the progression of chronic kidney disease after acute kidney injury. *Braz J Nephrol* 2017;39:230-1.
5. Gonçalves RG, Gabrich L, Rosário A Jr, Takiya CM, Ferreira ML, Chiarini LB, et al. The role of purinergic P2X7 receptors in the inflammation and fibrosis of unilateral ureteral obstruction in mice. *Kidney Int* 2006;70:1599-606. PMID: 16969386 DOI: <http://dx.doi.org/10.1038/sj.ki.5001804>
6. Bonventre JV, Yang L. Cellular pathophysiology of ischemic acute kidney injury. *J Clin Invest* 2011;121:4210-21. PMID: 22045571 DOI: <http://dx.doi.org/10.1172/JCI45161>
7. Szeto HH, Liu S, Soong Y, Seshan SV, Cohen-Gould L, Manichev V, et al. Mitochondria Protection after Acute Ischemia Prevents Prolonged Upregulation of IL-1 β and IL-18 and Arrests CKD. *J Am Soc Nephrol* 2017;28:1437-49. DOI: <http://dx.doi.org/10.1681/ASN.2016070761>
8. Holmes J, Rainer T, Geen J, Roberts G, May K, Wilson N, et al. Acute Kidney Injury in the Era of the AKI E-Alert. *Clin J Am Soc Nephrol* 2016;11:2123-31. DOI: <http://dx.doi.org/10.2215/CJN.05170516>