



Acute kidney success versus acute kidney injury

Hamid Nasri*

Acute kidney injury is considered by a sudden regression in kidney function, following an incapability to secrete waste products and to keep electrolyte and water balance (1,2). This condition is related to high risks of morbidity and death. Kidney improvement was described as a decrease in a serum creatinine level to the normal value. Acute kidney injury or acute renal failure necessitates to be distinguished early and primary triggers treated or eradicated (1-3). Acute renal failure is frequently multifactorial. Several factors like hemodynamic instability, major surgeries (i.e. cardiac surgery), sepsis, trauma, drug toxicity, radiocontrast agents and hypovolemia are the most popular causes of this condition, however, patients with underlying diseases have increased liability. Regardless of most dissimilar etiologies, pathophysiologic factors that happen concurrently in series, are endothelial dysfunction, modification of the microcirculation and renal tubular cell damage, then venous congestion happened and finally interstitial inflammation. Even milder forms of acute kidney injury have enhanced probability of morbidity and mortality (2-5). Acute renal failure complicates around 20% of hospitalizations, getting rates up to 40% in critically ill individuals. Numerous large observational investigations have detected link of acute renal failure with mortality in hospitalized individuals. Importantly, mortality has been connected to increase the severity of acute renal failure (1-6). Main risk factors for acute kidney injury at the patient's situation, are older age, the presence of chronic kidney disease, diabetes, hypertension, cardiovascular disease, hepatic problems, and lung disease. Regarding the large proportion of patients affected by acute kidney injury, the sequelae of acute renal failure may be a very large public health problem. Additionally, there are growing evidence, that acute kidney injury is an pivotal factor for chronic renal failure. Various investigations have detected a link between acute renal failure and long-term mortality and particularly progress of chronic renal failure and even development of end-stage kidney failure (3-6). In the recent decade, a tight link between acute kidney injury and progression to chronic renal failure has obtained increasing consideration by numerous large epidemiological investigations. In fact, even a complete

■ Implication for health policy/practice/research/medical education

Acute kidney injury is considered by a sudden regression in kidney function, following an incapability to secrete waste products and to keep electrolyte and water balance. This condition is related to high risks of morbidity and death. Kidney improvement was described as a decrease in a serum creatinine level to the normal value. The renin-angiotensin system inhibition will innately diminish excretory function in situations that intrarenal perfusion pressure is disturbed. Surprisingly, such a lessening in excretory work does not essentially accompany by any further injury, while lower intra-glomerular capillary pressures has not been associated with any particular morphological lesion. Pre-renal success has been designated for this contradistinction condition to the countering the diagnosis of 'pre-renal insufficiency'. In fact the term of 'pre-renal' notifies that the enhanced serum creatinine follows from hemodynamic derangements, and the term 'success' emphasizes the fact that the long-standing outcome is approximately suitable.

Keywords: Acute kidney injury, Chronic kidney disease, Renin-angiotensin system, Diabetes, Hypertension, Cardiovascular disease

recovery from acute kidney injury has been proposed to predispose to long-term harmful outcomes, while absent or only partial recovery further provokes this predisposition (2,4-7).

It should be noted that 20% of the cardiac output belongs to kidneys. Blood vessels ended to glomerular afferent arterioles and form a capillary network named glomerular tuft responsible for glomerular filtration. Then, efferent arterioles give rise to a dense network of capillaries which run alongside the proximal and distal convoluted renal tubules or form the vasa recta, which go parallel to the loops of Henle. Numerous investigations detected that angiotensin II independently or synergistically with other substances influences the mediators of glomerular filtration. Additionally, tubular damage can activate prerenal mechanisms through the tubular-glomerular response organization. The impact of angiotensin II is modulated by engagements of nitric oxide and also prostaglandins throughout the renal vascular system. Hence a bidirectional controlling happens amongst the

Received: 20 May 2018, Accepted: 9 July 2018, ePublished: 17 July 2018

Department of Nephrology, Division of Nephropathology, Isfahan University of Medical Sciences, Isfahan, Iran.

*Corresponding Author: Professor Hamid Nasri, Email: hamidnasri@med.mu.ac.ir

vasodilatory and vasoconstrictor hormonal structures. The administration of renin–angiotensin system inhibitors in various diseases necessitates a balance between longstanding benefits to renal survival and a primary reduction in excretory function that happen in the few days after starting renin–angiotensin system inhibitors. In fact, few amount of functional decrease subsequent renin–angiotensin system inhibition is mostly deemed tolerable in looking forward to the longer-term protection of renal system (1-5). Sometimes, some physicians diagnose an initial decline in GFR following renin–angiotensin system inhibition as ‘pre-renal insufficiency’. This entity is general and not restricted to general prescription. This condition demonstrates when blood pressure in chronic renal failure is dropped even by non-dropped medications. For these explanations, the expression of ‘pre-renal success’ has been recommended for these conditions (2-6).

In some clinical backgrounds, like pre-dialysis conditions, it may be more imperative to obtain maximization of renal excretory function by avoiding renin–angiotensin system inhibition. When acute renal failure happens, renin–angiotensin system inhibition is noticed to be a promoting factor to further intensify the acute kidney injury. This condition with conducted through specially vasodilating the efferent arterioles and eliminating the angiotensin II- facilitated keeping of internal glomerular pressure. Thus renin–angiotensin system inhibition will innately diminish excretory function in situations that intrarenal perfusion pressure is disturbed (4-7). Surprisingly, such a lessening in excretory work does not essentially accompany by any further injury, while lower intraglomerular capillary pressures have not been associated with any particular morphological lesion. Pre-renal success has been designated for this contradistinction condition to the countering the diagnosis of ‘pre-renal insufficiency’. In fact the term of ‘pre-renal’ notifies that the enhanced serum creatinine follows from hemodynamic derangements, and the term ‘success’ emphasizes the fact

that the long-standing outcome is approximately suitable. Thus, such treatment should be conserved, not changed. Hence, ‘Pre-renal success’ is a variation of the phrase ‘acute renal success’ (3-7).

Author's contribution

HN was wrote the paper lonely.

Conflicts of interest

The author declare no conflict of interest.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Funding/Support

None.

References

1. Condon CJ. Acute renal failure: success and failure of treatment. *J Iowa Med Soc.* 1973; 63:64-8.
2. Roshan B. Pre-renal or pre-tubular? *J Renal Inj Prev.* 2015;4:107-108. doi: 10.12861/jrip.2015.22
3. Arora P, Rajagopalam S, Ranjan R, Kolli H, Singh M, Venuto R, Lohr J. Preoperative use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers is associated with increased risk for acute kidney injury after cardiovascular surgery. *Clin J Am Soc Nephrol.* 2008;3:1266-73. doi: 10.2215/CJN.05271107.
4. Thureau K, Boylan JW. Acute renal success. The unexpected logic of oliguria in acute renal failure. *Am J Med.* 1976;61:308-15.
5. Redfors B, Bragadottir G, Sellgren J, Swärd K, Ricksten SE. Acute renal failure is NOT an “acute renal success”--a clinical study on the renal oxygen supply/demand relationship in acute kidney injury. *Crit Care Med.* 2010;38:1695-701. doi: 10.1097/CCM.0b013e3181e61911.
6. Romundstad S, Holmen J, Kvenild K, Hallan H, Ellekjaer H. Microalbuminuria and all-cause mortality in 2,089 apparently healthy individuals: a 4.4-year follow-up study. The Nord-Trøndelag Health Study (HUNT), Norway. *Am J Kidney Dis.* 2003;42:466-73.
7. Wolf G. Renal injury due to renin-angiotensin-aldosterone system activation of the transforming growth factor-beta pathway. *Kidney Int.* 2006;70:1914-9.

Citation: Nasri H. Acute kidney success versus acute kidney injury. *J Renal Endocrinol.* 2019;5:e01.

Copyright © 2019 The Author(s); Published by Nickan Research Institute. This is an open-access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.