ADQI 10 Figures

Original citation: Acute Dialysis Quality Initiative 10

Figure 1. New criteria for AKI diagnosis are displayed. In order to diagnose AKI selecting the worst criterion (function [RIFLE/AKIN] or damage) is recommended. In the appropriate clinical setting, this new damage biomarker criterion will enhance the ability of RIFLE/AKIN to define AKI. There are currently insufficient injury biomarker data to support staging of AKI, however, AKI stages basing on renal function changes are suggested to remain. The semi-quantitative trend for increasing biomarker severity associated with increasing kidney damage is suggested by the literature and is displayed by darkening background color as well as the symbols: +/++/+++. *Adapted from RIFLE/AKIN criteria. AKIN= acute kidney injury Network; sCrea=serum creatinine; UO=urine output; RRT=renal replacement therapy.

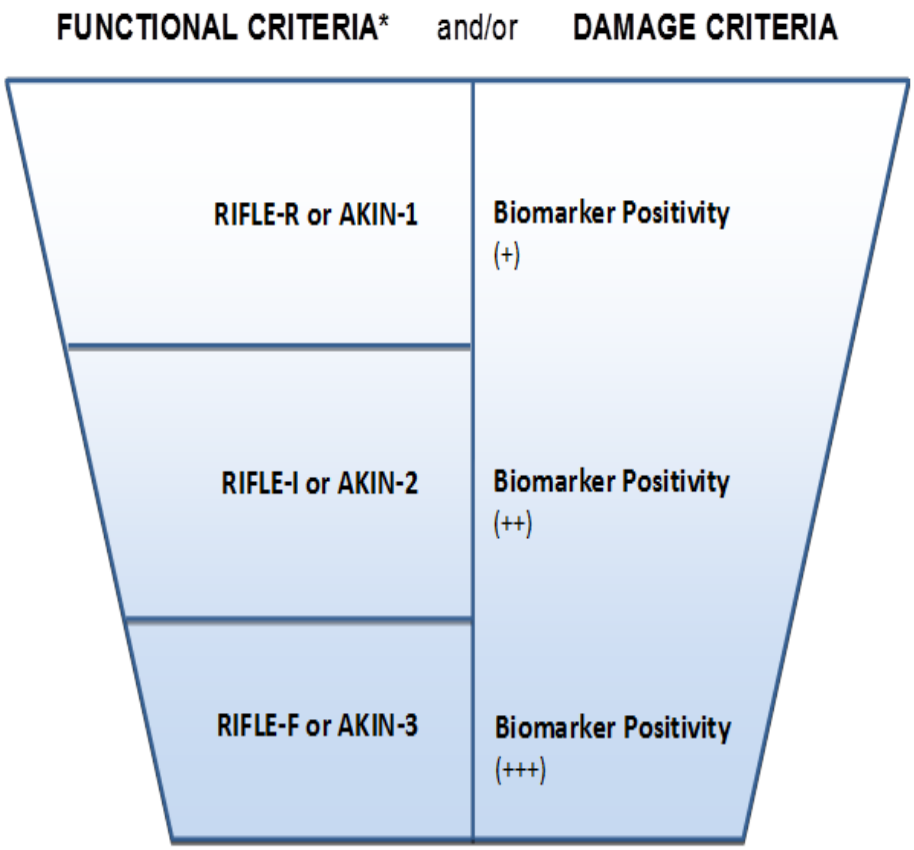


Figure 2. Recognition of AKI and clinical responses. Adapted from KDIGO: Kidney Disease Improving Global Outcomes; Kidney International Supplements (2012) 2, 1; doi: 0.1038/kisup.2012.

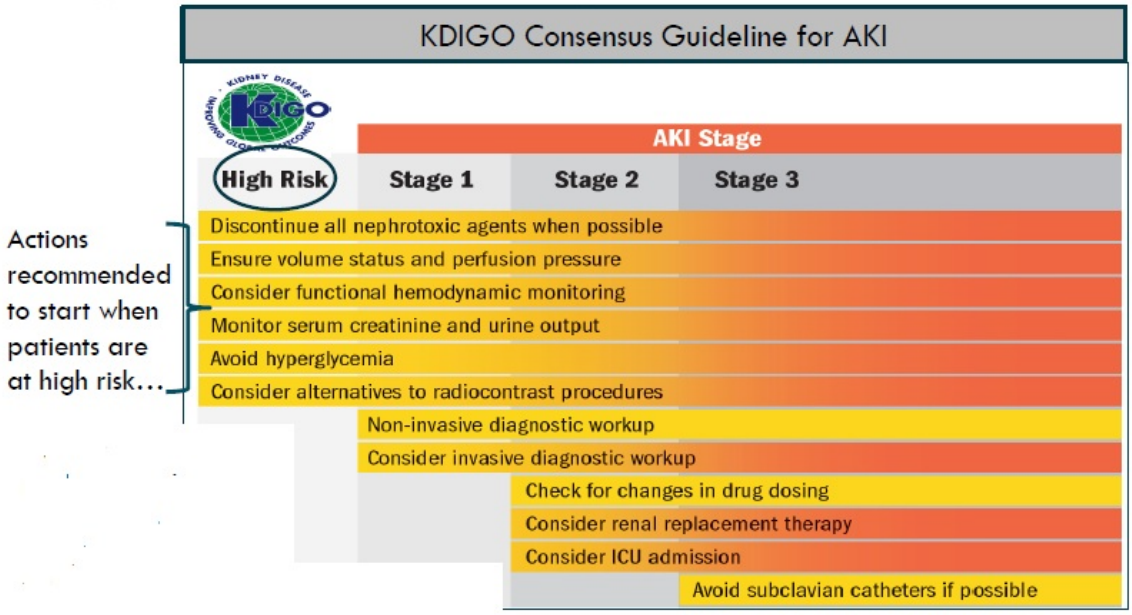


Figure 3. Acute kidney injury continuum and biomarker-based assessment.

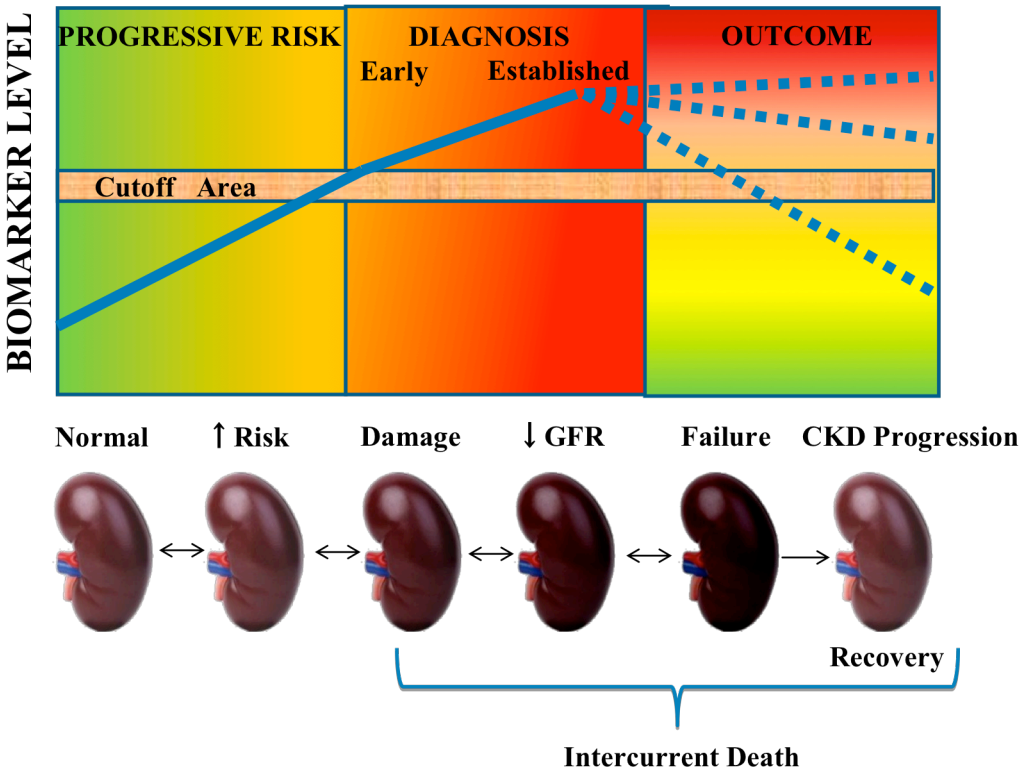


Figure 4 New spectrum of AKI based on combination of functional and damage biomarkers. As illustrated in this figure the combination of functional and damage biomarkers allows the clinician to differentiate a normal state of kidney function from abnormal to diagnose AKI. The current criteria for diagnosis include the lower two quadrants. This new spectrum enables the recognition of four subgroups of patients according to their AKI state. Patients negative for functional and kidney damage markers are considered to have no AKI (upper left quadrant). The ability to detect a state of damage alone (right upper quadrant) allows an expanded criteria for diagnosis of AKI. This may represent a "subclinical " state in which loss of function might develop several days after detection of kidney damage or not at all, however maybe associated with impaired outcomes. The bottom left quadrant indicates a dynamic change in renal filtration of serum creatinine but without detectable kidney damage that may be physiologic such as seen in patients with dehydration. The right lower quadrant represents patients with functional and damage criteria of AKI associated with the worst prognosis. It is expected that the process is dynamic and patients will move form one phase to another during the course of their illness. Currently there is limited information on what thresholds will be applicable for each of the damage biomarkers for the best diagnostic and staging criteria based on damage criteria alone. This will need to be defined in future studies.

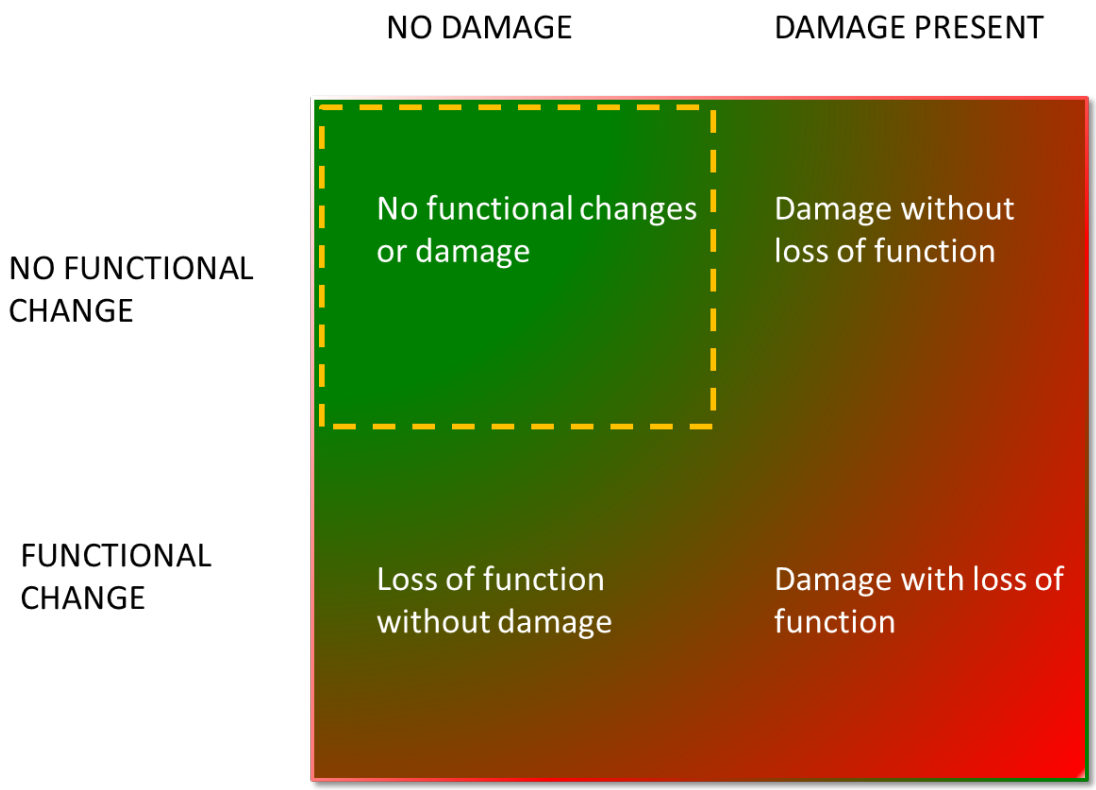


Figure 5. As shown in this figure the combination of functional and damage biomarkers allows categorziation of patients into a specific category after the diagnosis of AKI is made. Currently this is based on diagnosis with standard creatinine and urine output criteria, however in future could include a diagnosis based on damage biomarker criteria. These findings would need to be combined with the clinical and laboratory information to determine the underlying pathophysiology and the contributory factors. Sequential assessments could provide information on which of the factors is prevalent for ongoing injury or resolution and offer opportunities for targeted intervention.

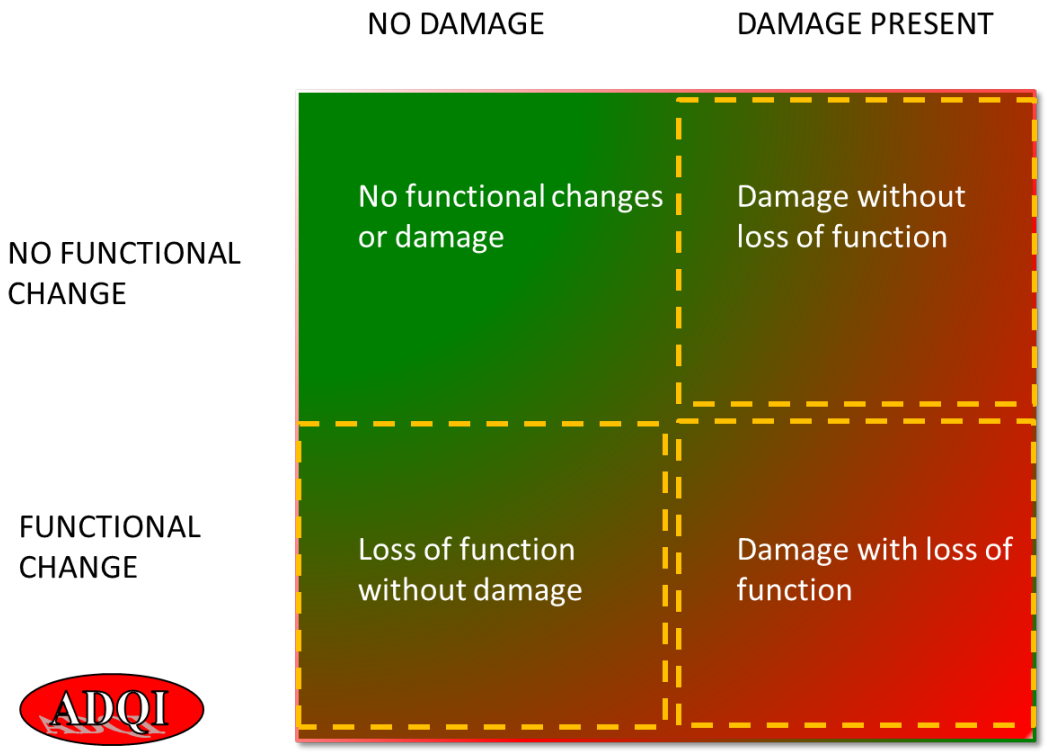
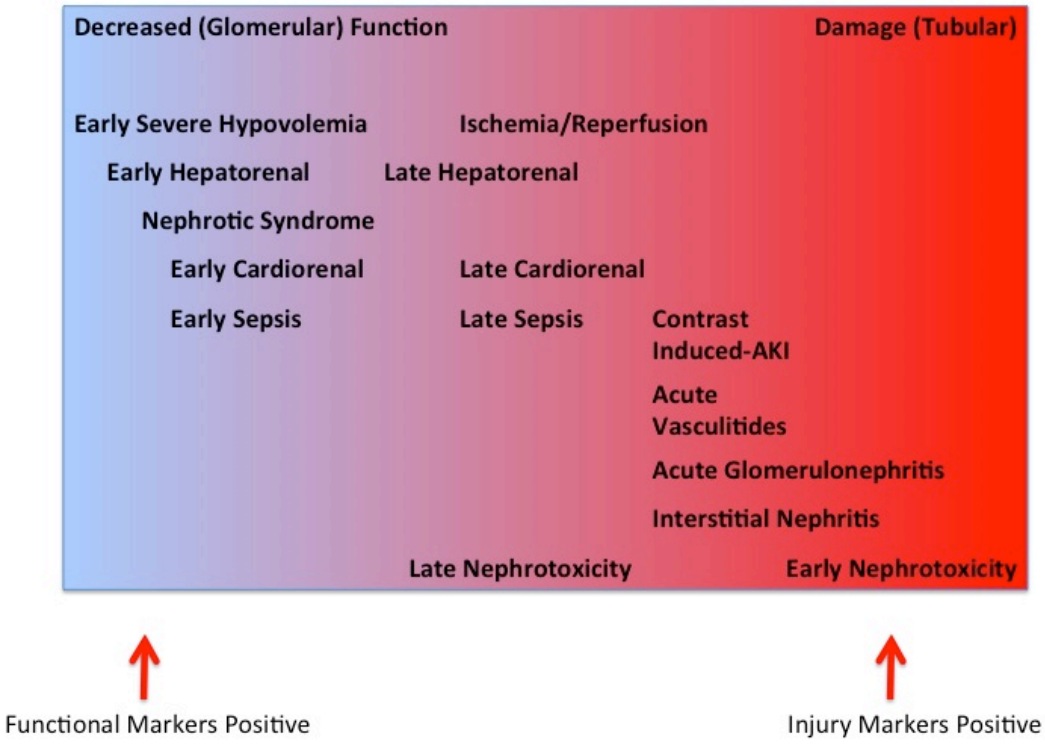


Figure 6. Differential Diagnosis of AKI. The arrows indicate the most likely type of biomarker to be positive during initial injury..



Fugure 7. Biomarker utility in differential diagnosis of established AKI and in the identification of injury pathways.

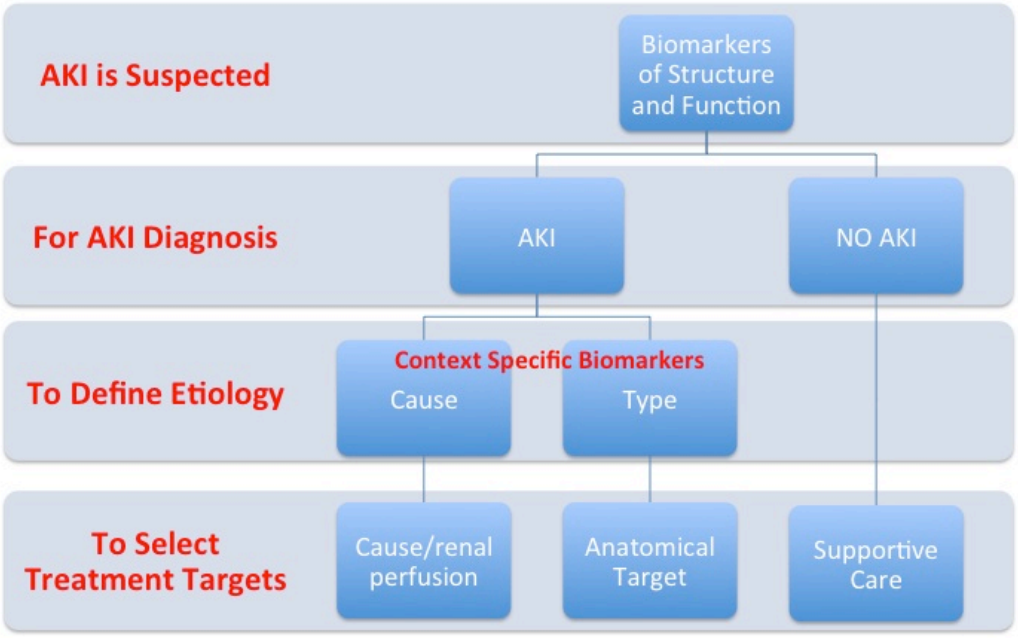


Figure 8. Biomarker Context Specific Thresholds

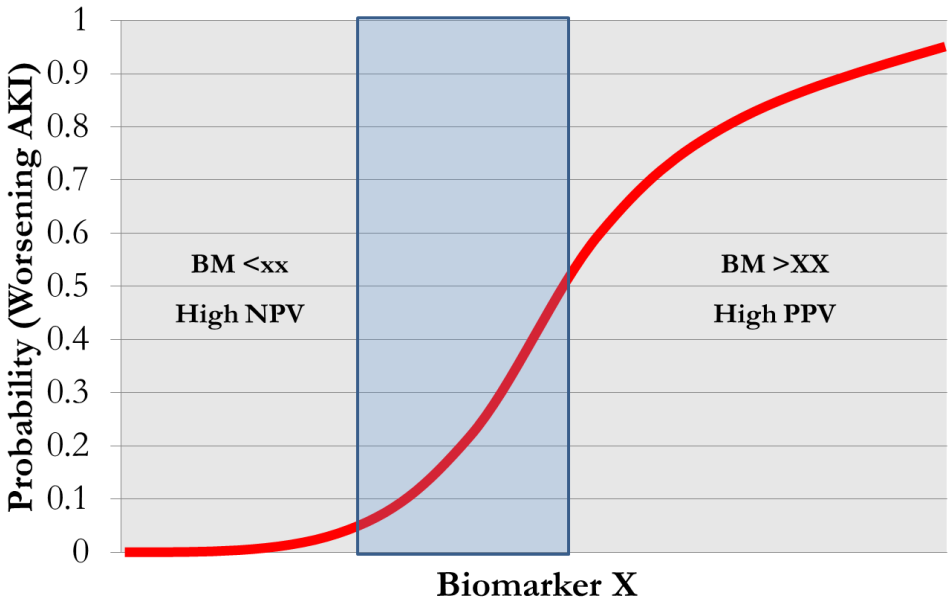


Figure 9. Utilization of functional and damage biomarkers for prognosis in AKI

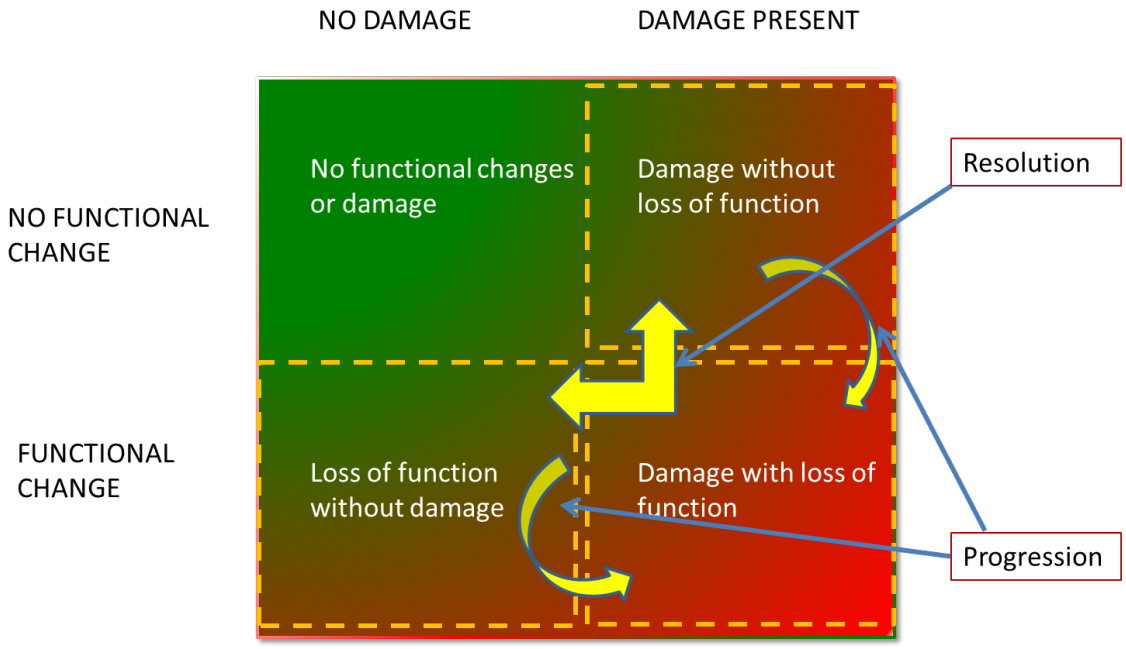


Figure 10. Physiological Phases of Acute Kidney Injury. The conceptual frame work of physiological biomarkers is superimposed upon the previously established concept of clinical phases of acute kidney injury.[1] This figure illustrates progression from risk to prerenal AKI and represents experimental data from ischemia reperfusion but not necessarily other forms of AKI such as drug-induced direct nephrotoxicity. Thus physiological biomarkers are not only needed in the early phase of AKI but throughout the continuum of AKI. The ability to measure these physiological variables may lead to identification of patients at risk for AKI, early diagnosis of AKI and may lead to variables, which may inform therapeutic decisions. These physiological processes represent an integrative environment for the interaction of inflammatory mediators, imbalance in the homeostasis of oxygen, nitric oxide and oxygen radicals causing microcirculatory dysfunction and impaired tissue oxygenation leading to AKI.