Acute Kidney Injury (AKI)

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13.1 Definition

Acute kidney injury (formerly known as acute renal failure) is a complex disorder with many underling conditions. It is seen in SIRS/sepsis and associated with a mortality of 60 % [1]. In 2007, a uniform definition was proposed which now replaces the more than 30 definitions that existed previously [2]. The diagnostic criteria for AKI were proposed based on acute alterations in serum creatinine or urine output. The Acute Kidney Injury Network (AKIN) criteria (Table 13.1) are based on epidemiological studies. Those studies indicated that modest changes in serum creatinine were significantly associated with mortality, hospital length of stay, and costs [3]. The old classification of prerenal, renal, and postrenal AKI is becoming less important as more than 90 % of the AKI patients suffer from a combination of prerenal and renal AKI.

The incidence of AKI is increasing worldwide. From 2000 to 2009, the incidence of dialysis-requiring AKI in the USA increased about 10 % per year [4]. This increase in incidence was evident in all age, sex, and race subgroups examined. The total number of deaths associated with dialysis-requiring AKI rose from 18,000 in 2000 to nearly 39,000 in 2009 [4]. Old age, higher degree of baseline renal impairment, and advanced diagnostic and therapeutic procedures epitomized by high-dose chemotherapy or implantation of cardiac assist devices are factors associated with this increase.

There is no specific treatment for AKI! Dozens of compounds that were tested to be effective in preclinical studies failed in the clinical setting; hence preventive measures are of importance. Prevention starts by identifying patients at risk. Table 13.2 summarizes risk factors.

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A.S. Merseburger et al. (eds.), *Urology at a Glance*,
DOI 10.1007/978-3-642-54859-8_13, © Springer-Verlag Berlin Heidelberg 2014

Table 13.1 AKIN criteria for the diagnosis of AKI

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR >0.3 mg/dl (>26.5 µmol/l) increase	<0.5 ml/kg/h for 6–12 h
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ≥12 h
3	3.0 times baseline OR Increase in serum creatinine to >4.0 mg/dl (>353.6 µmol/l) OR Initiation of renal replacement therapy OR in patients <18 years, decrease in eGFR to <35 ml/min/1.73 m^2	<0.3 ml/kg/h for >24 h OR Anuria for >12 h

Table 13.2 Causes of AKI: exposures and susceptibilities for nonspecific AKI

Exposures	Susceptibilities
Sepsis	Dehydration or volume depletion
Critical illness	Advanced age
Circulatory shock	Female gender
Cardiac surgery (especially with CPB)	Chronic diseases (heart, lung, liver)
Trauma	Diabetes mellitus
Major noncardiac surgery	Anemia
Nephrotoxic drugs	Cancer
Radiocontrast agents	CKD

Adapted from Kellum and Lameire [5]

There are currently no studies supporting the use of specific drugs for the prevention of AKI. This holds true for the infamous "renal dose dopa" [6] as well as for the use of diuretics which have been shown to have a detrimental effect on renal function and mortality [7, 8]. However, the early consultation of a nephrologist is associated with a better outcome of AKI patients [9]. In contrast to the disappointing results of pharmacological interventions, volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions has repeatedly shown to ameliorate or prevent AKI, especially in the setting of radio contrast exposure. Care must be taken that volume replacement does not lead to prolonged fluid accumulation, which has been shown to be associated with adverse outcome [10].

Although there is consensus that renal replacement therapy (RRT) needs to be emergently initiated if life-threatening changes in fluid, electrolyte, and acid-base balance exist or the patients are intoxicated with a dialyzable toxin, the timing of RRT initiation in general is more complex. There is no specific urea/BUN that should trigger the start of RRT, but rather the broader clinical context and the presence of conditions that can be modified with RRT have to be integrated into the decision making.

In contrast to previous assumptions that AKI, once survived, will not have long-term sequela, we now know that this is not true. In a large, community-based cohort

Table 13.3 The AKI ABC

A	Address drugs	Look for/discontinue NSAIDs, inhibitors of renin–angiotensin–aldosterone system, nephrotoxic antibiotics
B	Boost blood pressure	Required perfusion pressure for the kidney might be higher in severe atherosclerosis, abdominal compartment syndrome use Doppler sonography Consider functional hemodynamic monitoring and use of pressors
C	Calculate fluid balance	Persistent (>3 days) hypervolemia in AKI is associated with adverse outcome; hence, persistent hypervolemia should be avoided
D	Dip urine	Urinary sediment analysis to diagnose acute glomerulonephritis, vasculitis, interstitial nephritis, thrombotic Microangiopathy
E	Exclude obstruction	Obstruction has to be excluded in every patient with AKI by sonography

of patients with CKD, an episode of superimposed dialysis-requiring AKI was associated with very high risk for non-recovery of renal function. Dialysis-requiring AKI also seemed to be an independent risk factor for long-term risk for death or chronic dialysis dependency [11] (Table 13.3).

References

1. Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, Schetz M, Tan I, Bouman C, Macedo E, Gibney N, Tolwani A, Ronco C. Acute renal failure in critically ill patients: a multinational, multicenter study. JAMA. 2005;294:813–8.
2. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A. Acute Kidney Injury Network (AKIN): report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11:R31.
3. Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. J Am Soc Nephrol. 2005;16:3365–70.
4. Hsu RK, McCulloch CE, Dudley RA, Lo LJ, Hsu CY. Temporal changes in incidence of dialysis-requiring AKI. J Am Soc Nephrol. 2013;24:37–42.
5. Kellum JA, Lameire N. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (part 1). Crit Care. 2013;17:204.
6. Friedrich JO, Adhikari N, Herridge MS, Beyene J. Meta-analysis: low-dose dopamine increases urine output but does not prevent renal dysfunction or death. Ann Intern Med. 2005;142:510–24.
7. Lassnigg A, Donner E, Grubhofer G, Presterl E, Druml W, Hiesmayr M. Lack of renoprotective effects of dopamine and furosemide during cardiac surgery. J Am Soc Nephrol. 2000;11:97–104.
8. Mehta RL, Pascual MT, Soroko S, Chertow GM. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. JAMA. 2002;288:2547–53.
9. Mehta RL, McDonald B, Gabbai F, Pahl M, Farkas A, Pascual MT, Zhuang S, Kaplan RM, Chertow GM. Nephrology consultation in acute renal failure: does timing matter? Am J Med. 2002;113:456–61.
10. Bouchard J, Soroko SB, Chertow GM, Himmelfarb J, Ikizler TA, Paganini EP, Mehta RL. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. Kidney Int. 2009;76:422–7.
11. Hsu CY, Chertow GM, McCulloch CE, Fan D, Ordonez JD, Go AS. Nonrecovery of kidney function and death after acute on chronic renal failure. Clin J Am Soc Nephrol. 2009;4:891–8.