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Acute Kidney Injury

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UNIWERSYTET MEDYCZNY W ŁODZI

- a rapid loss of kidney function.
- It affects some 3-7% of patients admitted to the hospital and approximately 25-30% of patients in the ICU

Introduced by the Acute Kidney Injury Network (AKIN), specific criteria for the diagnosis of AKI:

- Rapid time course (**less than 48 hours**)
- Reduction of kidney function

  Absolute increase in **serum creatinine of ≥0.3 mg/dl** (≥26.4 µmol/l) or percentage increase in serum creatinine of **≥50%**
- Reduction in urine output, defined as **<0.5 ml/kg/hr** for more than 6 hours

no more GFR

Stage	SCr	Urine output
1	≥1.5-1.9 times baseline OR 0.3 mg/dl increase	<0.5 ml/kg/h for 6-12 hours
2	≥2.0-2.9 times baseline	<0.5 ml/kg/h for ≥12 hours
3	≥3.0 times baseline OR increase in SCr to ≥4.0 mg/dl OR RRT In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2	<0.3 ml/kg/h for ≥24 hours OR Anuria for ≥12 hours

Risk, Injury, Failure, Loss, and End-stage Kidney (RIFLE) classification

Class	Glomerular filtration rate criteria	Urine output criteria
Risk	Serum creatinine × 1.5	< 0.5 ml/kg/hour × 6 hours
Injury	Serum creatinine × 2	< 0.5 ml/kg/hour × 12 hours
Failure	Serum creatinine × 3, or serum creatinine ≥ 4 mg/dl with an acute rise > 0.5 mg/dl	< 0.3 ml/kg/hour × 24 hours, or anuria × 12 hours
Loss	Persistent acute renal failure = complete loss of kidney function > 4 weeks	
End-stage kidney disease	End-stage kidney disease > 3 months	

Table 4. Comparison of RIFLE and AKIN criteria for diagnosis and classification of AKI.

AKI Staging		RIFLE	
Serum Creatinine	Urine Output (Common to Both)	Class	Serum Creatinine or GFR
Stage 1 Increase of more than or equal to 0.3 mg/dL (≥ 26.4 μmol/L) or increase to more than or equal to 150% to 200% (1.5- to 2-fold) from baseline	Less than 0.5 mL/kg/h for more than 6 hours	**R**isk	Increase in serum creatinine x 1.5 or GFR decrease >25%
Stage 2 Increased to more than 200% to 300% (> 2- to 3-fold) from baseline	Less than 0.5 mL/kg per hour for more than 12 hours	**I**njury	Serum creatinine x 2 or GFR decreased >50%
Stage 3 Increased to more than 300% (>3-fold) from baseline, or more than or equal to 4.0 mg/dL (≥354 μmol/L) with an acute increase of at least 0.5 mg/dL (44 μmol/L) or on RRT	Less than 0.3 mL/kg/h for 24 hours or anuria for 12 hours	**F**ailure	Serum creatinine x 3, or serum creatinine >354 μmol/L (4 mg/dL) with an acute rise >44 μmol/L (0.5 mg/dL) or GFR decreased >75%
		Loss	Persistent acute renal failure = complete loss of kidney function >4 weeks
		End-stage kidney disease	End-stage kidney disease >3 months

Prerenal causes of AKI – leading to decrease effective blood flow to the kidney.

- systemic causes,
  - low blood volume
  - low blood pressure
  - heart failure,

local changes to the blood vessels supplying the kidney.

- renal artery stenosis
- renal vein thrombosis

- Intrinsic AKI due to:
- damage to the glomeruli,
- Damage to the renal tubules,
- Damage to the interstitium.

- Common causes:
- glomerulonephritis,
- acute tubular necrosis (ATN)
- acute interstitial nephritis (AIN)

- *Postrenal* AKI is a consequence of urinary tract obstruction.
- may be related to
  - benign prostatic hyperplasia,
  - kidney stones
  - obstructed urinary catheter.

BUN:Cr	Cr:Urea	Location of disorder	Mechanism
>20:1	<10:1	Prerenal (before the kidney)	Reduced bloodflow causes elevated creatinine and BUN due to decreased glomerular filtration rate (GFR). Additionally, BUN reabsorption is increased because of the lower flow; BUN is disproportionately elevated relative to creatinine.
10-20:1	10-25:1	Normal or Postrenal (after the kidney)	Normal range. Can also be postrenal disease, as backflow from obstruction (e.g., in the ureters or urethra) causes elevated BUN reabsorption within kidney; creatinine is not reabsorbed, therefore BUN:Cr ratio increases.
<10:1	>25:1	Intrarenal (within kidney)	Renal damage causes reduced reabsorption of BUN, therefore lowering the Bun:Cr ratio.

prevention

Table 6. Causes of AKI: Exposures and susceptibilities

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

Abbreviations: CKD, chronic kidney disease; CPB, cardiopulmonary bypass.

treatment

- Improve cardiac output:
  - intravenous fluids
  - inotropes
  - dopamine **(„renal dose" doesn't exist, no benefit, may be harmful)**
- Toxin-induced prerenal AKI – discontinue the agent (aminoglycoside, penicillin, NSAIDs, or acetaminophen).
- Remove the obstruction of the urinary tract

- Loop diuretics, sometimes convenient in ameliorating fluid overload
- do not reduce the risk of complications or death.
- Managemnt of Metabolic acidosis, hyperkalemia or pulmonary edema if present

- Renal replacement therapy,
- Hemodialysis, sometimes continuous venovenous hemofiltration (CVVH)
- However no difference in outcomes between the use of intermittent hemodialysis and CVVH