Acute Kidney Injury

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ABSTRACT

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AKI has replaced the term ARF is clinical practice because, the word 'Kidney" is more familiar to public than the word 'renal" and intervention is to be done at the stage of injury to prevent failure. AKI is very common in hospitalized patients (5%), especially in critically ill (25 – 30%). It is an independent risk factor for mortality, associated with a mortality of 50 – 90% when dialysis is required. Recent studies have shown that AKI is associated with an increase long term risk of subsequent development of CVD or CKD and mortality, even after apparent resolution of AKI. AKI is common, harmful and potentially treatable. Even a minor acute reaction in Kidney function has an adverse prognosis.

Keywords: AKI, Causes, Prevention, Management, Prognosis, Biomarkers.

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AKI DEFINITION

Want a new look: Why change of Name to AKI

Acute renal failure (ARF) is not a failure, but an "acute renal success", on the part of nature to achieve self preservation. AKI has replaced the term ARF is clinical practice because, the word 'Kidney" is more familiar to public than the word 'renal" and intervention is to be done at the stage of injury to prevent failure.

Why is AKI important

AKI is very common in hospitalized patients (5%), especially in critically ill (25 – 30%). It is an independent risk factor for mortality, associated with a mortality of 50 – 90% when dialysis is required. Recent studies have shown that AKI is associated with an increase long term risk of subsequent development of CVD or CKD and mortality, even after apparent resolution of AKI. AKI is common, harmful and potentially treatable. Even a minor acute reaction in Kidney function has an adverse prognosis.

AKI: Definition (KDIGO CPG 2012)

AKI is defined as any of the following:[1,2]

1. Increase in Serum Creatinine by e" 0.3 mg/dl in 48 hours
2. Increase in Serum Creatinine by e" 1.5 times in baseline which is known or presumed to have occurred within the prior 7 days
3. Urine volume< 0.5 ml/kg/hr for 6 hours

Kidney failure is defined as GFR <15ml/mt/1.73m2 BSA or requirement of RRT.

Staging of AKI

AKI is staged for severity according to the following criteria.

Stage I: Serum Creatinine 1.5 – 1.9 times baseline a e"0.3mg/dl
Stage 2: Serum Creatinine 2-2.9 times baseline. Urine output <0.5ml/kg/hr for ³12 hrs
Stage 3: Serum Creatinine 3.0 times baseline

OR

Serum Creatinine³ 4.0 OR

Initiation of renal replacement therapy

Urine Output <0.3ml/kg/hr for³ 24hrs OR

Anuria >12 hrs.

Patient is staged according to highest stage, if Serum Creatinine and Urine Output map to different stages. The risk of death and need for RRT increases with increased stage of AKI.

Conceptual model for AKI

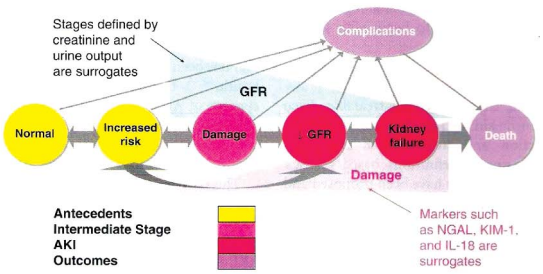

Figure 1. **Conceptual model for AKI**

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Risk factors and causes of AKI

1. Old age
2. Pre existing CKD
3. Sepsis & critical illness
4. IV Vol. depletion - hemorrhage, GI loss, Burns, Capillary leak.
5. Decreased cardiac output- Hypotension and Shock.
6. Systemic Vasodilatation -Anaphylaxis, cirrhosis, drugs.
7. Drugs and toxins- Nephrotoxic antibiotics, NSAIDS Poisonous animals and plant toxins.
8. Obstruction – Bilateral ureteric obstruction or obstruction in a solitary functioning kidney.

Causes of AKI are broadly categorized into:

i. Pre renal AKI: 40-45% of AKI are due to pre-renal causes, in which there is renal hypo perfusion. This occurs in intravascular volume depletion and reduced cardiac output. In Hepatorenal syndrome, there is systemic vasodilatation with intense renal vasoconstriction, leading to a state of pre renal AKI. In pre renal state, kidneys are structurally normal. Correction of cause will improve the kidney function and reverse AKI

ii. Intrinsic AKI: Intrinsic Renal Parenchymal disease leading to AKI

This includes

1. Acute tubular necrosis - ATN
2. Acute Glomerulonephritis- AGN
3. Acute interstitial nephritis- AIN
4. Thrombotic microangiopathy-TMA
5. Acute tubular necrosis – ATN

In ATN, there is apoptosis or necrosis of renal tubular epithelial cells. The most severe injury occur in proximal convoluted tubule (PCT) and Thick Ascending limb of loop of Henle (TALHL) because these two segments of tubule are the metabolically most active segments and lie in the medulla. Normally 99% of glomerular filtrate is to be reabsorbed, which become defective in ATN. So ATN is usually nonoliguric.

The two most common causes leading to ATN are ischaemia and toxins. Ischaemic ATN occurs in the setting of prolonged hypotension and renal hypo perfusion due to IV volume depletion. This is the usual cause of AKI in the post operative period, contributed by NSAIDS or amino glycosides.

Toxic ATN occur in the setting of snake envenomation, wasp bite, NSAIDS, Cisplatin, contrast media, aminoglycosides etc.

Sepsis is another important cause of AKI. AKI occur in 65% of septic shock. Kidney involvement is common in sepsis because

- Intrarenal hemodynamic changes
- Endothelial dysfunction
- Infiltration of inflammatory cells is renal Parenchyma
- Intraglomerular thrombosis
- Obstruction of tubules by necrotic debri

Early diagnosis

Patients with high risk of developing AKI should be monitored closely; necessary modification should be done to prevent the development of AKI. For eg:

- Maintain normal IV fluid volume status
- Maintenance of BP
- Improve cardiac function
- Avoid n NSAIDS and nephrotoxic antibiotics wherever possible
- Take necessary precautions to prevent CI-AKI before contrast media administration

Using Serum Creatinine as a measure of GFR to define or stage AKI has several shortcomings :-

- Different values from different labs
- Coefficient variation between different testing methods
- Daily variation in Serum Creatinine due to difference in diet and activity.
- Endogenous chromogens (bilirubin, vitamin C, uric acid) and drugs (eg: cephalosporins, trimethoprim, cimetidine)

So new Biomarkers of AKI may help in making an early and precise diagnosis of AKI. Unlike in acute myocardial infarction where the new molecules like CPK, CPK-MB, Troponins have come into clinical practice over the last 30 years, AKI biomarker are not that popular.

The Biomarkers in AKI are

1. Cystatin C
2. NGAL
3. IL-18

4. KIM-1
5. L-FABP
6. Netrin-1

Of these different biomarkers, Serum Cystatin C and NGAL in Serum or Urine are available for clinical use.

Prevention and Management

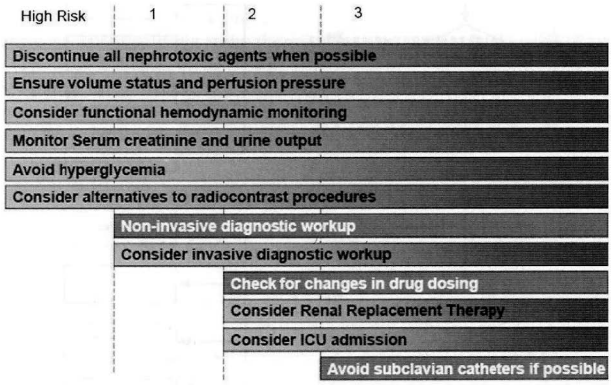

Figure 2. **Prevention and management**

Patients with increased risk of AKI require careful attention to their hemodynamic status. Management of BP and Cardiac output require careful titration of fluids and vasoactive medications. The fluid of choice on the initial management for expansion of intravascular volume in patients with risk of AKI or with AKI is crystalloids rather than colloids. Vasopressors may be added in conjunction with fluids in patients with vasomotor shock. Norepinephrine and vasopressin are more commonly used now. Low due dopamine, Fenoldopam and ANP are not recommended to treat or prevent AKI.

Diuretics are recommended only in the management of fluid over load. Its use is not recommended to prevent AKI. Mannitol also is not scientifically justified in the prevention of AKI. A single dose of Theophylline may be given in neonates with severe prenatal asphyxia, who are at risk of AKI.

The recommendations to prevent AKI due to amino glycoside treatment are

1. Don't use amino glycoside for treatment of infection unless no suitable, less nephrotoxic therapeutic alternates are available.
2. Administer amino glycosides as a single daily dose rather than multiple dose daily treatment regimens.
3. Monitor drug level if possible if the drug is used for > 48 hrs.
4. Necessary dose modification may be done according to GFR.

To Prevent CI-AKI:

- Use lowest possible dose of contrast medium in patients at risk for CI-AKI
- Use either iso-osmolar or low osmolar iodinated contrast media, rather than high osmolar iodinated contrast media in patients at increased risk of CI-AKI
- Intravascular volume expansion with either isotonic saline or sodium bicarbonate solutions should be given 12 hr prior and 12 hr after the contrast exposure, at a rate of 1 ml/kg/hr.
- Oral N-acetyl cysteine 600mg twice/day may be given 48 hrs, along with Normal Saline
- Theophylline, Fenoldopam etc are not recommended to prevent CI – AKI
- Prophylactic hemodialysis or hemofiltration for contrast media removal in not recommended in patients at increased risk of CI-AKI

Renal replacement therapy,(RRT) may have to be started when life-threatening changes in fluid, electrolytes and acid- base balance exist.[1,2] When making the decision to start RRT, broader clinical context should be taken in to account, rather than a single BUN and Creatinine value. Both CRRT and intermittent Hemodialysis can be used an complementary therapies, but CRRT is preferred for hemodynamically unstable patients.

END NOTE

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