With the invention of current medical tools and machineries the utilization of clinical judgment sometimes takes a back seat in our day to days practice. We as a clinician need to know thoroughly what tools to utilize and when not to take their help. Nephrology practice has to be followed on this concept as well. Before going into the topic, it would be worthwhile explaining some of the commonly used nephrology terms to which most of us would be already aware of.

AKI is defined as any of the following:

1. Increase in serum creatnine by 0.3 mg/dl within 48 hours; or
2. Increase in serum creatinine to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
3. Urine volume

Table1: Staging 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 hours
2	2.0–2.9 times baseline	< 0.5 ml/kg/h for ≥ 12 hours
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 per 1.73 m^2	< 0.3 ml/kg/h for ≥ 24 hours OR Anuria for ≥ 12 hours

* *Kidney International Supplements (2012) 2, 19–36; doi:10.1038/kisup.2011.32*

CKD is defined as abnormalities of kidney structure or function, present for >3 months, with implications for health.

Table 2: Criteria for CKD*

Markers of kidney damage (one or more)	Albuminuria (AER ≥30 mg/24 hours; ACR ≥30 mg/g [≥3 mg/mmol]) Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging History of kidney transplantation
Decreased GFR	GFR <60 ml/min/1.73 m^2 (GFR categories G3a-G5)

Abbreviations: CKD, chronic kidney disease; GFR, glomerular filtration rate.

* *Kidney International Supplements (2012) 2, 19–36; doi:10.1038/kisup.2011.32*

It is recommended that CKD be classified based on cause, GFR category, and albuminuria category (CGA). Cause of CKD has to be assigned based on the presence or absence of systemic diseases and the location within the kidney of observed or presumed pathologic-anatomic findings.

GFR categories has to be assigned as follows:

Table 3. GFR Categories*

GFR category	GFR (ml/min/1.73 m^2)	Terms
G1	≥ 90	Normal or high
G2	60-89	Mildly decreased*
G3a	45-59	Mildly to moderately decreased
G3b	30-44	Moderately to severely decreased
G4	15-29	Severely decreased
G5	< 15	Kidney failure

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Albuminuria categories have to be assigned as follows:

Table 4: Albuminuria Categories*

Category	AER (mg/24 hours)	ACR (approximate equivalent) (mg/mmol)	(mg/g)	Terms
A1	<30	<3	<30	Normal to mildly increased
A2	30-300	3-30	30-300	Moderately increased*
A3	>300	>30	>300	Severely increased**

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Acute Kidney Injury:

The cause of AKI should be determined whenever possible. Patients should be stratified for risk of AKI according to their susceptibilities and exposures and should be managed accordingly. They should be evaluated to determine the cause of AKI, with special attention to reversible causes. Regular monitoring of these patients with measurements of serum creatinine and urine output should be done. Once AKI has resolved the patient should be re-evaluated 3 months for new onset, or worsening of pre-existing CKD. If these patients have CKD, they should be managed as per CKD guidelines. However, if patients do not have CKD, still consider them to be at increased risk for CKD and regular follow up of these patients should be done.

Prevention and Treatment of AKI:

In the absence of hemorrhagic shock, isotonic crystalloids rather than colloids (albumin or starches) should be used for initial management for expansion of intravascular volume in patients at risk for AKI or with AKI. Vasopressors should also be added in conjunction with fluids in patients with vasomotor shock with, or at risk for AKI.[8] In critically ill patients, insulin therapy has to be started with target plasma glucose of 110–149mg/dl.[9-11] Dietary considerations has to be given as well in the management of AKI. A total energy intake of 20–30 kcal/kg/day has to be provided in all patients with any stage of AKI. Protein intake should not be curtailed with the aim of preventing or delaying initiation of RRT. The recommended allowance is 0.8–1.0 g/kg/day of protein in noncatabolic AKI patients without need for dialysis, 1.0–1.5 g/kg/day in patients with AKI on RRT, and up to a maximum of 1.7 g/kg/day in patients on continuous renal replacement therapy (CRRT) and in hypercatabolic patients. Nutrition preferentially should be administered via the enteral route in patients with AKI.

Following is the list of Dos ans Don’ts in the management of AKI:

- Do not use diuretics to prevent AKI. Do not use diuretics to treat AKI, except in the management of volume overload.[18,19]
- Do not use low-dose dopamine to prevent or treat AKI.[20,22]
- Do not use fenoldopam to prevent or treat AKI. [23,24]
- Do not use atrial natriuretic peptide (ANP) to prevent or treat AKI.[25-27]
- Do not use aminoglycosides for the treatment of infections unless no suitable, less nephrotoxic,therapeutic alternatives are available. In patients with normal kidney function in steady state, aminoglycosides should be administered as a single dose daily rather than as multiple-dose daily treatment regimen. Topical or local applications of aminoglycosides (e.g., respiratory aerosols, instilled antibiotic beads), rather than i.v. application, can be employed when feasible and suitable.[28-34]
- Do not use NAC to prevent AKI in critically ill patients with hypotension. [35,36]

- Do not use oral or i.v. NAC for prevention of postsurgical AKI. [37]
- A single dose of theophylline may be given in neonates with severe perinatal asphyxia, who are at high risk of AKI.
- Lipid formulations of amphotericin B rather than conventional formulations of amphotericin B should be used. In the treatment of systemic mycoses, azole antifungal agents and/or the Echinocandins should be used rather than conventional amphotericin B, if equal therapeutic efficacy can be assumed.

Contrast-induced (CI) AKI:

In individuals who develop changes in kidney function after administration of intravascular contrast media, evaluation for CI-AKI as well as for other possible causes of AKI should be done. Assessment for the risk for CI-AKI and, in particular, screening for pre-existing impairment of kidney function should be done in all patients who are considered for a procedure that requires intravascular (i.v. or i.a.) administration of iodinated contrast medium. [47]

Dos ans Don’ts:

- Alternative imaging methods should be considered in patients at increased risk for CI-AKI. Lowest possible dose of contrast medium has to be used in patients at risk for CI-AKI. [48]
- Either iso-osmolar or low-osmolar iodinated contrast media should be used, rather than high-osmolar iodinated contrast media in patients at increased risk of CI-AKI.[49,50]
- I.v. volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions should always be done prior to i.v. contrast administration. Do not use oral fluids alone in patients at increased risk of CI-AKI. [51]
- Oral NAC, together with i.v. isotonic crystalloids can be administered, in patients at increased risk of CI-AKI.[52,53]
- Do not use theophylline to prevent CI-AKI. [54,55]
- Do not use fenoldopam to prevent CI-AKI. [56,57]
- Do not use prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI.[58-60]

Dialysis Interventions for Treatment of AKI:

Renal Replacement therapy (RRT) should be initiated emergently when life-threatening changes in fluid, electrolyte, and acid-base balance exist. A broader clinical outlook has to be adopted to visualize the presence of conditions that can be modified with RRT, including trends of laboratory tests, rather than single BUN and creatinine thresholds alone when making the decision to start RRT. RRT should be discontinued when it is no longer required, either because intrinsic kidney function has recovered to the point that it is adequate to meet patient needs, or because RRT is no longer consistent with the goals of care. Important to note here is that, diuretics should not be used to enhance kidney function recovery, or to reduce the duration or frequency of RRT. [61]

Anticoagulation: In a patient with AKI requiring RRT, decision to use anticoagulation for RRT should be based on the assessment of the risks and benefits from anticoagulation. Anticoagulation should be used during RRT in AKI if a patient does not have an increased bleeding risk or impaired coagulation and is not already receiving systemic anticoagulation.[62,63] For such patients following is suggested:

- For anticoagulation in intermittent RRT: unfractionated or low-molecular weight

heparin.

- For anticoagulation in CRRT: regional citrate anticoagulation.
- For anticoagulation during CRRT in patients who have contraindications for citrate: either unfractionated or low-molecular-weight heparin.[64,65]

RRT in patients with AKI should be initiated via an uncuffed nontunneled dialysis catheter, rather than a tunneled catheter.[62-63]When choosing a vein for insertion of a dialysis catheter in patients with AKI, these preferences should be considered:

- First choice: right jugular vein;
- Second choice: femoral vein;
- Third choice: left jugular vein;
- Last choice: subclavian vein with preference for the dominant side.[66,67]

Medication management and patient safety in CK:

We should take GFR into account while deciding about the drug dosing in CKD patients. Potentially nephrotoxic and renally excreted drugs should be discontinued in patients with a GFR

Dos ans Don’ts:

- Medical advice should be sought before using over-the-counter medicines or nutritional protein supplements.
- Do not use herbal remedies in people with CKD.[68,69]
- Use of metformin should be reviewed in patients with GFR 30–44 ml/min/1.73 m2 (GFR category G3b) and it should be discontinued in people with GFR
- All people taking potentially nephrotoxic agents such as lithium and calcineurin inhibitors should have their GFR, electrolytes and drug levels regularly monitored.[70-77]
- People with CKD should not be denied therapies for other conditions such as cancer but there should be appropriate dose adjustment of cytotoxic drugs according to knowledge of GFR.[78]
- Imaging studies: The risk of acute impairment in kidney function due to contrast agent use should be assessed and balanced against the diagnostic value and the benefits involved. Following information is noteworthy:
    - Avoidance of high osmolar agents.
    - Use of lowest possible radiocontrast dose.
    - Withdrawal of potentially nephrotoxic agents before and after the procedure.
    - Adequate hydration with saline before, during, and after the procedure.
    - Measurement of GFR 48–96 hours after the procedure.[79-82]
    - Gadolinium-based contrast media should not be used in people with GFR 30 ml/min/1.73 m2 (GFR categories G4-G5) who require gadolinium containing contrast media should be offered a macrocyclic chelate preparation.[83-85]

Referral to specialists: referral to a specialist for kidney care for people with CKD should be advised in the following circumstances: [86-89]

- AKI or abrupt sustained fall in GFR.
- GFR
- A consistent finding of significant albuminuria (ACR >300 mg/g or AER >300 mg/24 hours.
- Progression of CKD.
- Urinary red cell casts, RBC >20 per high power field, sustained and not readily

explained.

- CKD and hypertension refractory to treatment with 4 or more antihypertensive agents.
- Persistent abnormalities of serum potassium.
- Recurrent or extensive nephrolithiasis.
- Hereditary kidney disease.

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