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.

Downloaded from ccemjournal.com

* *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

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

Albuminuria categories have to be assigned as follows:

Table 4: Albuminuria Categories*

Category	AER (mg/24 hours)	ACR (approximate equivalent) (mg/mmol)	ACR (approximate equivalent) (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**

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

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

Downloaded from ccemjournal.com

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]

Downloaded from ccemjournal.com

- 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]

Downloaded from ccemjournal.com

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

Downloaded from ccemjournal.com

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

Downloaded from ccemjournal.com

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.

BIBLIOGRAPHY

1. Kidney International Supplements (2012) 2, 19–36; doi:10.1038/kisup.2011.32
2. Harel Z, Chan CT. Predicting and preventing acute kidney injury after cardiac surgery. CurrOpinNephrolHypertens2008; 17: 624–628.
3. Reddy VG. Prevention of postoperative acute renal failure. J Postgrad Med 2002; 48: 64–70.
4. Venkataraman R. Can we prevent acute kidney injury? Crit Care Med 2008; 36: S166–171
5. Prowle JR, Bellomo R. Continuous renal replacement therapy: recent advances and future research. Nat Rev Nephrol2010; 6: 521–529.
6. Bouchard J, Soroko SB, Chertow GM, et al. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. Kidney Int2009; 76: 422–427.
7. Payen D, de Pont AC, Sakr Y, et al. A positive fluid balance is associated with a worse outcome in patients with acute renal failure. Crit Care 2008; 12: R74.
8. Karlsson S, Varpula M, Ruokonen E, et al. Incidence, treatment, and outcome of severe sepsis in ICU-treated adults in Finland: the Finnsepsis study. Intensive Care Med 2007; 33: 435–443.
9. Van Cromphaut SJ. Hyperglycaemia as part of the stress response: the underlying mechanisms. Best Pract Res Clin Anaesthesiol 2009; 23: 375–386.
10. Kosiborod M, Inzucchi SE, Goyal A, et al. Relationship between spontaneous and iatrogenic hypoglycemia and mortality in patients hospitalized with acute myocardial infarction. JAMA 2009; 301: 1556–1564.
11. Kosiborod M, Rathore SS, Inzucchi SE, et al. Admission glucose and mortality in elderly patients hospitalized with acute myocardial infarction: implications for patients with and without recognized diabetes. Circulation 2005; 111: 3078–3086.
12. Fiaccadori E, Maggiore U, Rotelli C, et al. Effects of different energy intakes on nitrogen balance in patients with acute renal failure: a pilot study. Nephrol Dial Transplant 2005; 20: 1976–1980.

Downloaded from ccemjournal.com

13. Bellomo R, Tan HK, Bhonagiri S, et al. High protein intake during continuous hemodiafiltration: impact on amino acids and nitrogen balance. Int J Artif Organs 2002; 25: 261–268.
14. Druml W. Metabolic aspects of continuous renal replacement therapies. Kidney Int Suppl 1999: S56–61.
15. Chima CS, Meyer L, Hummell AC, et al. Protein catabolic rate in patients with acute renal failure on continuous arteriovenous hemofiltration and total parenteral nutrition. J Am Soc Nephrol 1993; 3: 1516–1521.
16. Leblanc M, Garred LJ, Cardinal J, et al. Catabolism in critical illness: estimation from urea nitrogen appearance and creatinine production during continuous renal replacement therapy. Am J Kidney Dis 1998; 32: 444–453.
17. Marshall MR, Golper TA, Shaver MJ, et al. Urea kinetics during sustained low-efficiency dialysis in critically ill patients requiring renal replacement therapy. Am J Kidney Dis 2002; 39: 556–570.
18. Lassnigg A, Donner E, Grubhofer G, et al. Lack of renoprotective effects of dopamine and furosemide during cardiac surgery. J Am Soc Nephrol 2000; 11: 97–104.
19. Lombardi R, Ferreiro A, Servetto C. Renal function after cardiac surgery: adverse effect of furosemide. Ren Fail 2003; 25: 775–786
20. Kellum JA, M Decker J. Use of dopamine in acute renal failure: a metaanalysis. Crit Care Med 2001; 29: 1526–1531.
21. Marik PE. Low-dose dopamine: a systematic review. Intensive Care Med 2002; 28: 877–883.
22. Friedrich JO, Adhikari N, Herridge MS, et al. Meta-analysis: low-dose dopamine increases urine output but does not prevent renal dysfunction or death. Ann Intern Med 2005; 142: 510–524.
23. Tumlin JA, Finkel KW, Murray PT, et al. Fenoldopam mesylate in early acute tubular necrosis: a randomized, double-blind, placebo-controlled clinical trial. Am J Kidney Dis 2005; 46: 26–34.
24. Brienza N, Malcangi V, Dalfino L, et al. A comparison between fenoldopam and low-dose dopamine in early renal dysfunction of critically ill patients. Crit Care Med 2006; 34: 707–714.
25. Ratcliffe PJ, Richardson AJ, Kirby JE, et al. Effect of intravenous infusion of atriopeptin 3 on immediate renal allograft function. Kidney Int 1991; 39: 164–168.
26. Sands JM, Neylan JF, Olson RA, et al. Atrial natriuretic factor does not improve the outcome of cadaveric renal transplantation. J Am Soc Nephrol 1991; 1: 1081–1086.

Downloaded from ccemjournal.com

27. Kurnik BR, Allgren RL, Genter FC, et al. Prospective study of atrial natriuretic peptide for the prevention of radiocontrast-induced nephropathy. Am J Kidney Dis 1998; 31: 674–680.
28. Zahar JR, Rioux C, Girou E, et al. Inappropriate prescribing of aminoglycosides: risk factors and impact of an antibiotic control team. J Antimicrob Chemother 2006; 58: 651–656.
29. Bliziotis IA, Michalopoulos A, Kasiakou SK, et al. Ciprofloxacin vs an aminoglycoside in combination with a beta-lactam for the treatment of febrile neutropenia: a meta-analysis of randomized controlled trials. Mayo Clin Proc 2005; 80: 1146–1156.
30. Falagas ME, Matthaiou DK, Bliziotis IA. The role of aminoglycosides in combination with a beta-lactam for the treatment of bacterial endocarditis: a meta-analysis of comparative trials. J Antimicrob Chemother 2006; 57: 639–647.
31. Falagas ME, Matthaiou DK, Karveli EA, et al. Meta-analysis: randomized controlled trials of clindamycin/aminoglycoside vs. beta-lactam monotherapy for the treatment of intra-abdominal infections. Aliment Pharmacol Ther 2007; 25: 537–556.
32. Glasmacher A, von Lilienfeld-Toal M, Schulte S, et al. An evidence-based evaluation of important aspects of empirical antibiotic therapy in febrile neutropenic patients. Clin Microbiol Infect 2005; 11 (Suppl 5): 17–23.
33. Paul M, Benuri-Silbiger I, Soares-Weiser K, et al. Beta lactam monotherapy versus beta lactam-aminoglycoside combination therapy for sepsis in immunocompetent patients: systematic review and meta-analysis of randomised trials. BMJ 2004; 328: 668.
34. Paul M, Silbiger I, Grozinsky S, et al. Beta lactam antibiotic monotherapy versus beta lactam-aminoglycoside antibiotic combination therapy for sepsis. Cochrane Database Syst Rev 2006: CD003344.
35. Hoffmann U, Fischereder M, Kruger B, et al. The value of N-acetylcysteine in the prevention of radiocontrast agent-induced nephropathy seems questionable. J Am Soc Nephrol 2004; 15: 407–410.
36. Izzedine H, Guerin V, Launay-Vacher V, et al. Effect of N-acetylcysteine on serum creatinine level. Nephrol Dial Transplant 2001; 16: 1514–1151.
37. Ho KM, Morgan DJ. Meta-analysis of N-acetylcysteine to prevent acute renal failure after major surgery. Am J Kidney Dis 2009; 53: 33–40.
38. Karlowicz MG, Adelman RD. Nonoliguric and oliguric acute renal failure in asphyxiated term neonates. Pediatr Nephrol 1995; 9: 718–722.
39. Gouyon JB, Guignard JP. Theophylline prevents the hypoxemia-induced renal

Downloaded from ccemjournal.com

hemodynamic changes in rabbits. Kidney Int 1988; 33: 1078–1083.
40. Bakr AF. Prophylactic theophylline to prevent renal dysfunction in newborns exposed to perinatal asphyxia–a study in a developing country. Pediatr Nephrol 2005; 20: 1249–1252.
41. Bhat MA, Shah ZA, Makhdoomi MS, et al. Theophylline for renal function in term neonates with perinatal asphyxia: a randomized, placebo controlled trial. J Pediatr 2006; 149: 180–184.
42. Jenik AG, Ceriani Cernadas JM, Gorenstein A, et al. A randomized, doubleblind, placebo-controlled trial of the effects of prophylactic theophylline on renal function in term neonates with perinatal asphyxia. Pediatrics 2000; 105: E45.
43. Kleinberg M. What is the current and future status of conventional amphotericin B? Int J Antimicrob Agents 2006; 27 (Suppl 1): 12–16.
44. Saliba F, Dupont B. Renal impairment and amphotericin B formulations in patients with invasive fungal infections. Med Mycol 2008; 46: 97–112.
45. Ullmann AJ, Sanz MA, Tramarin A, et al. Prospective study of amphotericin B formulations in immunocompromised patients in 4 European countries. Clin Infect Dis 2006; 43: e29–38.
46. Yoo BK, Jalil Miah MA, Lee ES, et al. Reduced renal toxicity of nanoparticular amphotericin B micelles prepared with partially benzylatedpoly-L-aspartic acid. Biol Pharm Bull 2006; 29: 1700–1705.
47. 47.Lameire N, Adam A, Becker CR, et al. Baseline renal function screening. Am J Cardiol 2006; 98: 21K–26K
48. Cigarroa RG, Lange RA, Williams RH, et al. Dosing of contrast material to prevent contrast nephropathy in patients with renal disease. Am J Med 1989; 86: 649–652.
49. Goldfarb S, Spinler S, Berns JS, et al. Low-osmolality contrast media and the risk of contrast-associated nephrotoxicity. Invest Radiol 1993;28 (Suppl 5): S7–10; discussion S11–12.
50. Barrett BJ, Carlisle EJ. Metaanalysis of the relative nephrotoxicity of high- and low-osmolality iodinated contrast media. Radiology 1993; 188: 71–178.
51. Weisbord SD, Mor MK, Resnick AL, et al. Prevention, incidence, and outcomes of contrast-induced acute kidney injury. Arch Intern Med 2008; 168: 1325–1332.
52. McCullough PA. Contrast-induced acute kidney injury. J Am Coll Cardiol 2008; 51: 1419–1428.
53. Klein-Schwartz W, Doyon S. Intravenous acetylcysteine for the treatment of acetaminophen overdose. Expert Opin Pharmacother 2011; 12: 119–130.
54. Kelly AM, Dwamena B, Cronin P, et al. Meta-analysis: effectiveness of drugs for

Downloaded from ccemjournal.com

preventing contrast-induced nephropathy. Ann Intern Med 2008; 148: 284–294.
55. Bagshaw SM, Ghali WA. Theophylline for prevention of contrast-induced nephropathy: a systematic review and meta-analysis. Arch Intern Med 2005; 165: 1087–1093
56. Stone GW, McCullough PA, Tumlin JA, et al. Fenoldopam mesylate for the prevention of contrast-induced nephropathy: a randomized controlled trial. JAMA 2003; 290: 2284–2291.
57. Allaqaband S, Tumuluri R, Malik AM, et al. Prospective randomized study of N-acetylcysteine, fenoldopam, and saline for prevention of radiocontrastinduced nephropathy. Catheter Cardiovasc Interv 2002; 57: 279–283.
58. Deray G. Dialysis and iodinated contrast media. Kidney Int Suppl 2006: S25–29.
59. Cruz DN, Perazella MA, Ronco C. The role of extracorporeal blood purification therapies in the prevention of radiocontrast-induced nephropathy. Int J Artif Organs 2008; 31: 515–524.
60. Vogt B, Ferrari P, Schonholzer C, et al. Prophylactic hemodialysis after radiocontrast media in patients with renal insufficiency is potentially harmful. Am J Med 2001; 111: 692–698.
61. Wu VC, Ko WJ, Chang HW, et al. Risk factors of early redialysis after weaning from postoperative acute renal replacement therapy. Intensive Care Med 2008; 34: 101–108.
62. Bellomo R, Cass A, Cole L, et al. Intensity of continuous renal-replacement therapy in critically ill patients. N Engl J Med 2009; 361: 1627–1638.
63. Palevsky PM, Zhang JH, O'Connor TZ, et al. Intensity of renal support in critically ill patients with acute kidney injury. N Engl J Med 2008; 359: 7–20.
64. 64.Lim W, Cook DJ, Crowther MA. Safety and efficacy of low molecular weight heparins for hemodialysis in patients with end-stage renal failure: a meta-analysis of randomized trials. J Am Soc Nephrol 2004; 15: 3192–3206.
65. European Best Practice Guidelines for Haemodialysis (Part 1). V. Chronic intermittent haemodialysis and prevention of clotting in the extracorporal system. Nephrol Dial Transplant 2002; 17 (Suppl 7): 63–71.
66. National Kidney Foundation. KDOQI clinical practice guidelines and clinical practice recommendations for 2006 updates: vascular access. Am J Kidney Dis 2006; 48: S176–S307.
67. O'Grady NP, Alexander M, Dellinger EP, et al. Guidelines for the prevention of intravascular catheter-related infections. Infect Control Hosp Epidemiol 2002; 23: 759–769.
68. Gokmen MR, Lord GM. Aristolochic acid nephropathy. BMJ 2012; 344:e4000.
69. Su T, Zhang L, Li X et al. Regular use of nephrotoxic medications is an

Downloaded from ccemjournal.com

independent risk factor for chronic kidney disease–results from a Chinese population study. Nephrol Dial Transplant 2011; 26:1916–1923.
70. National Institute for Health and Clinical Excellence. NICE Clinical Guideline 38: Bipolar Disorder. 2006.
71. Lipska KJ, Bailey CJ, Inzucchi SE. Use of metformin in the setting of mild to-moderate renal insufficiency. Diabetes Care 2011; 34: 1431–1437.
72. Rachmani R, Slavachevski I, Levi Z et al. Metformin in patients with type 2 diabetes mellitus: reconsideration of traditional contraindications. Eur J Intern Med 2002; 13: 428.
73. Salpeter S, Greyber E, Pasternak G et al. Risk of fatal and nonfatal lactic acidosis with metformin use in type 2 diabetes mellitus. Cochrane Database Syst Rev 2010: CD002967.
74. Fellstrom BC, Jardine AG, Schmieder RE et al. Rosuvastatin and cardiovascular events in patients undergoing hemodialysis. N Engl J Med 2009; 360: 1395–1407.
75. Ginsberg HN, Elam MB, Lovato LC et al. Effects of combination lipid therapy in type 2 diabetes mellitus. N Engl J Med 2010; 362:1563–1574.
76. Keech A, Simes RJ, Barter P et al. Effects of long-term fenofibrate therapy on cardiovascular events in 9795 people with type 2 diabetes mellitus (the FIELD study): randomised controlled trial. Lancet 2005; 366: 1849–1861.
77. Wanner C, Krane V, Marz W et al. Atorvastatin in patients with type 2 diabetes mellitus undergoing hemodialysis. N Engl J Med 2005; 353:238–248.
78. Limdi NA, Beasley TM, Baird MF et al. Kidney function influences warfarin responsiveness and hemorrhagic complications. J Am Soc Nephrol 2009; 20: 912–921.
79. Goldfarb S, McCullough PA, McDermott J et al. Contrast-induced acute kidney injury: specialty-specific protocols for interventional radiology, diagnostic computed tomography radiology, and interventional cardiology. Mayo Clin Proc 2009; 84: 170–179.
80. American College Radiology Guidelines. Manual on Contrast Media Version 8. http://www.acr.org/B/media/ACR/Documents/PDF/Quality Safety/Resources/Contrast%20Manual/Contrast%20Nephrotoxicity.pdf. Accessed October 12, 2012.
81. European Society for Urological Radiology (ESUR). http://www.esur.org/ Contrast-media.51.0.html, Accessed October 9, 2012.
82. Heinrich MC, Haberle L, Muller V et al. Nephrotoxicity of iso-osmolar iodixanol compared with nonionic low-osmolar contrast media: metaanalysis of randomized controlled trials. Radiology 2009; 250: 68–86.

Downloaded from ccemjournal.com

83. Perazella MA. Current status of gadolinium toxicity in patients with kidney disease. Clin J Am Soc Nephrol 2009; 4: 461–469.
84. Agarwal R, Brunelli SM, Williams K et al. Gadolinium-based contrast agents and nephrogenic systemic fibrosis: a systematic review and metaanalysis. Nephrol Dial Transplant 2009; 24: 856–863.
85. American College Radiology Guidelines. Manual on Contrast Media Version 8. http://www.acr.org/B/media/ACR/Documents/PDF/Quality-Safety/Resources/Contrast%20Manual/Nephrogenic%20Systemic%20Fibrosis.pdf. Accessed October 12, 2012.
86. KDIGO AKI Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney inter., Suppl. 2012; 2: 1–138.
87. KDIGO GN Work Group. KDIGO clinical practice guideline for glomerulonephritis. Kidney inter., Suppl. 2012; 2: 139–274.
88. KDIGO CKD-MBD Work Group. KDIGO clinical practice guideline for the diagnosis, evaluation, prevention, and treatment of Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD). Kidney Int Suppl 2009; 76(Suppl 113): S1–130.
89. KDIGO BP Work Group. KDIGO clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney inter.,Suppl. 2012; 2: 337–414.

Author:

Dr. Shashank Gupta (Consultant Nephrologist & Renal Transplant Physician, Narayana Superspeciality Hospital, Guwahati)

Author



CCEM Journal
View all posts

Downloaded from ccemjournal.com