ORIGINAL RESEARCH PAPER

Volume-8 | Issue-2 | February-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL PROFILE OF ACUTE KIDNEY INJURY IN HOSPITALIZED PATIENTS

Medicine

Dr. Sunil J. Panjwani — Associate Professor, Department of General Medicine, Sir Takhtasinhji Hospital and Govt. Medical College, Bhavnagar.

Dr. Hirava Munshi * — Assistant Professor, Department of General Medicine, Sir Takhtasinhji Hospital and Govt. Medical College, Bhavnagar.*Corresponding Author

Dr. Chirag B. Patel — 3rd Year Resident Doctor Department of General Medicine, Sir Takhtasinhji Hospital and Govt. Medical College, Bhavnagar.

ABSTRACT

AKI complicates approximately 5-7% of hospital admission & 30% of admission to I.C.U patients. The risk of AKI is contributed by the acute insult and background morbidity. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction, & parenchymal kidney disease. The study was conducted on 112 patients with elevated serum creatinine as per KDIGO guidelines and symptoms suggestive of acute kidney injury in the medical wards at Sir Takhatsinhji General Hospital, Bhavnagar. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease, nephrotoxic medications also contribute to insult.

SUMMARY:

- **Clinical spectrum of AKI :**

Most common age group was 31 to 40 yrs in this study. The incidence of AKI was more in males than females (1.7:1 ratio). Most common clinical features were oliguria (84.82%), vomiting (82.14%) and diarrhea (50.00%). Co-morbidities found in the decreasing order were DM, HTN and IHD. Oliguric AKI and non-oliguric AKI were seen in 84.82 % and 15.18% respectively.

- **Etiology of AKI :**

Acute diarrheal disease (50.00%) was the most common cause of AKI, followed by sepsis (21.43%), malaria (10.71%), liver cirrhosis (7.14%) and snakebite (3.57%). Acute diarrheal disease still remains the most common cause of acute kidney injury. Sepsis is another leading cause of AKI. According to AKIN criteria, patients classified into AKIN 1, AKIN 2 and AKIN 3 stages were 33.04%, 25.89% and 41.07% respectively. Almost half of patients presented with AKI on admission and other half developed AKI after admission. 49.11% of patients were detected to have AKI on admission while 50.89% developed AKI after admission. Requirement of dialysis support in our study was 30.36% and were treated by haemodialysis. Haemodialysis become the preferred mode of renal replacement therapy.

- **Outcome :**

Complete recovery was seen in 81.25% cases and partial recovery was seen in 8.04% cases. In-hospital mortality rate was 10.71%. Paraquat poisoning and aluminum phosphide poisoning induced AKI had the highest in-hospital mortality (100%).

KEYWORDS

INTRODUCTION

Acute kidney injury (AKI) depicts the abrupt decline in renal function mostly occurs over the course (hours to days) and ends in retention of metabolic waste products and dysregulation of fluid, electrolytes & acid base homeostasis. The risk of AKI is contributed by the acute insult and background morbidity. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction & parenchymal kidney disease. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease, nephrotoxic medications also contribute to insult.

The etiological spectrum of AKI is notably different between developing and developed nations. It is related to environmental, social and economic circumstances. Sepsis, surgery and trauma are the most common causes of AKI in developed countries. In developing countries, acute diarrhaeal diseases and tropical diseases still prevail.3 The aetiology, course and outcome of AKI differ in various parts of India.4,5

The RIFLE classification defines three grades of severity and two clinical outcomes of acute kidney injury. The three grades of severity (Risk, Injury and Failure) based on changes to serum creatinine and urine output. The two clinical outcomes are Loss and End-stage. In 2007, a modified version of the RIFLE criteria was published by the AKI Network (AKIN), an international collaboration of nephrologists and intensivists, known as the AKIN criteria.[9]

The risk of AKI is contributed by the acute insult and background morbidity. Acute insult may be in the forms of sepsis and hypoperfusion, toxicity, obstruction & parenchymal kidney disease. Background morbidities in the form of elderly, CKD, cardiac failure, liver failure, diabetes mellitus, vascular disease and nephrotoxic medication also contribute to insult.

AIM AND OBJECTIVES

AIM

1. To study of Clinical profile of acute kidney injury in hospitalized patients.

OBJECTIVES

1. To find out the clinical and etiological profile of AKI in medical wards.
2. To apply the AKIN criteria in AKI patients admitted to the medical wards and to confirm its significance.
3. To study the distribution of acute kidney injury in patients with regards to age, sex, drugs, co-morbid conditions & hospitalization.

MATERIAL & METHOD

Source of Data

The study was conducted on patients coming with elevated serum creatinine as per KDIGO guidelines and symptoms suggestive of acute kidney injury in the medical wards at Sir Takhtasinhji General Hospital, Bhavnagar.

Sample Size: 112 cases
Sample procedure: Observational cross-sectional study
Duration: 2017-2018

Inclusion criteria:

- Patients above the age of 18 years given informed consent for the study.
- Already diagnosed cases of medical disorders irrespective of treatment status and showing biochemical evidence of acute kidney injury using renal biomarkers as specified in the study.

Exclusion criteria:

- Patient not giving consent

- Age < 18yrs
- Pregnancy
- Chronic kidney disease
- Previously diagnosed kidney disease

TERMINOLOGY AND DEFINITIONS:

- AKI was defined and classified by the AKIN criteria.

.

Classification/Staging system for acute kidney injury

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of more than or equal to 0.3 mg/dl or increase to more than or equal to 150% to 200%(1.5- to 2-fold) from baseline	Less than 0.5 ml/kg per hour for more than6 hours
2	Increase in serum creatinine to more than 200% to 300% (>2- to 3-fold)from baseline	Less than 0.5 ml/kg per hour for more than12 hours
3	Increase in serum creatinine to more than 300% (>3-fold) from baseline (or serum creatinine of more than or equal to 4.0 mg/dl with an acute increase of at least 0.5 mg/dl, individuals who receive RRT)	Less than 0.3 ml/kg per hour for 24 hours or anuria for 12 hours

Regarding the outcome, complete recovery (CR) was defined as the serum creatinine <1.6mg/dl at the time of discharge. Partial recovery (PR) was defined as persistent dialysis-independent renal failure. The third outcome was in hospital mortality (IHM).

METHOD:

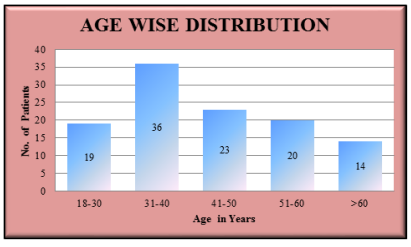
Detailed history, clinical examination and laboratory investigations were carried out in all patients. Following important data were recorded: date when AKI was detected, date of consultation, type and frequency of dialysis support instituted. Also, exposure to nephrotoxic drugs prior to or during hospital stay, co-morbid conditions and base line serum creatinine were noted if available. All patients were subjected to urine analysis, haemogram, blood biochemistry (which included urea, creatinine, electrolytes, calcium). USG was done to rule out CKD and obstructive causes.

OBSERVATION AND RESULT

- In this study, 112 cases of AKI were studied in the medical wards of Sir Takhtasinhji General Hospital, Bhavnagar during the study period. The following observations were noted:

TABLE 1: AGE WISE DISTRIBUTION

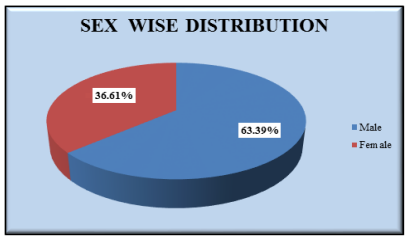
Age (years)	No. of patients	(%) of patients
18-30	19	16.96%
31-40	36	32.14%
41-50	23	20.54%
51-60	20	17.86%
>60	14	12.50%
Total	112	100%
Mean Age	43.47 + 12.73	



Almost 50% of our patients were in the 3rd and 4th decades. As we have not included pre-existing CKD in our study, age group >50 yrs have low incidence. The age ranges between 43.47 + 12.73 years. Median age was 42 years.

TABLE 2: SEX WISE DISTRIBUTION

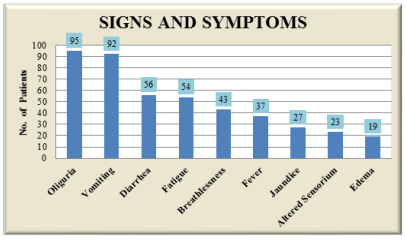
Sex	No. of patients	(%) of patients
Male	71	63.39%
Female	41	36.61%
Total	112	100%



Out of 112 patients, 71 (63.39 %) were males while 41 (36.61%) were females. The sex ratio is 1.7:1.

TABLE 3: CLINICAL PARAMETERS OF AKI

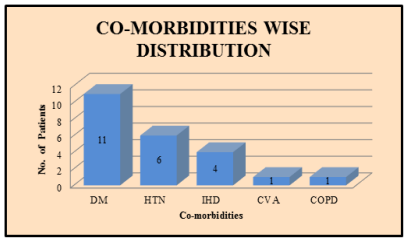
Clinical Parameters	No. of patients	(%) of patients
Oliguria	95	84.82
Vomiting	92	82.14
Diarrhea	56	50.00
Fatigue	54	48.21
Breathlessness	43	38.39
Fever	37	33.04
Jaundice	27	24.11
Altered Sensorium	23	20.54
Edema	19	16.96



The clinical features observed in our study were oliguria (84.82%), vomiting (82.14%), diarrhea (50.00%), fatigue (48.21%), breathlessness (38.39%), fever (33.04%), altered sensorium (20.54%), jaundice (24.11%) and oedema (16.96%). Clinical features depend on the underlying condition and its severity.

TABLE 4: CO-MORBIDITIES WISE DISTRIBUTION

Co-morbidities	No. of patients	(%) of patients
DM	11	9.82%
HTN	6	5.36%
IHD	4	3.57%
CVA	1	0.89%
COPD	1	0.89%

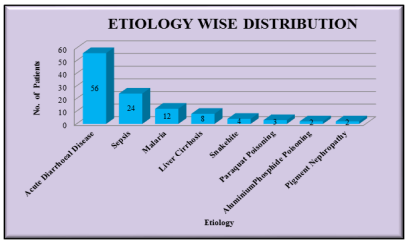


Co-morbidities were absent in 79.46% patients. Out of the 23 patients with co-morbidities, DM was present in 11 patients (9.82%), HTN was present in 6 patients (5.36%) and IHD was present in 4 patients (3.57%). CVA and COPD were present in 1 patient each (0.89%).

TABLE 5: ETIOLOGICAL PROFILE OF AKI

Etiology	No. of patients	(%) of patients
Acute Diarrhoeal Disease	56	50.00%
Sepsis	24	21.43%

Malaria	12	10.71%
Liver Cirrhosis	8	7.14%
Snakebite	4	3.57%
Paraquat Poisoning	3	2.68%
Aluminium Phosphide Poisoning	2	1.79%
Pigment Nephropathy	2	1.79%
NSAID Induced	1	0.89%



The most common cause of AKI was acute diarrhoeal disease (50%) followed by sepsis (21.43%), malaria (10.71%), liver cirrhosis (7.14%), snakebite (3.57%) and paraquat poisoning (2.68%).

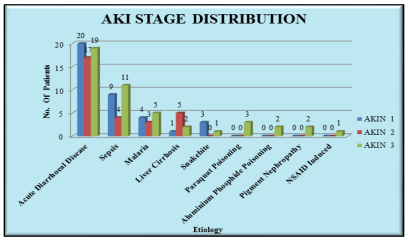
TABLE 6: BIOCHEMICAL PARAMETERS OF AKI

Biochemical parameters	Mean	SD
Hemoglobin (gm %)	9.54	1.58
TC (cumm)	10,957	5,157
PC (lakhs)	2.68	1.00
RBS (mg/dl)	114	34
Serum Creatinine(mg/dl)	4.74	2.39
Serum Urea(mg/dl)	128	68
Serum Sodium(meq/l)	135.74	4.96
Serum Potassium(meq/l)	4.98	1.42
Serum Uric acid (meq/l)	6.65	1.75
Total Protein(g/dl)	6.3	0.7
Serum Albumin(g/dl)	2.98	0.54

The most common changes of AKI were seen in serum urea and serum creatinine by using biochemical parameters.

TABLE 7: AKI STAGE AND ITS DISTRIBUTION

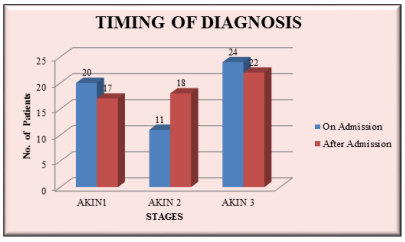
Etiology	AKIN 1	AKIN 2	AKIN 3	Total
Acute Diarrhoeal Disease	20 (35.71%)	17 (30.36%)	19 (33.93%)	56 (50%)
Sepsis	9 (37.5%)	4 (16.66%)	11 (45.83%)	24 (21.4%)
Malaria	4 (33.33%)	3 (25%)	5 (41.67%)	12 (10.7%)
Liver Cirrhosis	1 (12.5%)	5 (62.5%)	2 (25%)	8 (7.1%)
Snakebite	3 (75%)	0	1 (25%)	4 (3.6%)
Paraquat Poisoning	0	0	3 (100%)	3 (2.7%)
Aluminium PhosphidePoisoning	0	0	2 (100%)	2 (1.8%)
Pigment Nephropathy	0	0	2 (100%)	2 (1.8%)
NSAID Induced	0	0	1 (100%)	1 (0.9%)
Total	37 (33.04%)	29 (25.89%)	46 (41.07%)	112 (100%)



Out of the 112 Patients, 37 (33.04%), 29 (25.89%) and 46 (41.07%) were in the AKIN 1, AKIN 2 and AKIN 3 stages respectively.

TABLE 8: TIMING OF DIAGNOSIS

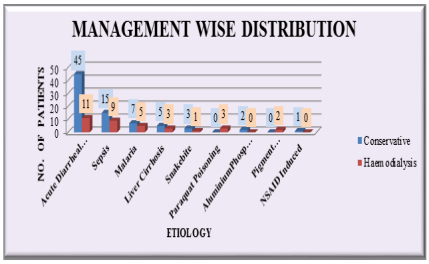
Timing of Diagnosis	AKIN 1	AKIN 2	AKIN 3
On Admission	20(36.36%)	11(20.00%)	24(43.64%)
After Admission	17(29.82%)	18(31.58%)	22(38.60%)
Total	37(33.04%)	29(25.89%)	46(41.07%)



Out of 112 Patients 55 (49.11%) were detected to have AKI at the time of admission [AKIN: STAGE 1, 20 (36.36%); STAGE 2, 11 (20.00%) and STAGE 3, 24 (43.64%)] and57 (50.89%) developed AKI after admission [AKIN: STAGE 1, 17 (29.82%); STAGE 2, 18 (31.58%) and STAGE 3, 22 (38.60%)].

TABLE 9: MANAGEMENT WISE DISTRIBUTION

Etiology	Conservative (No. of patients)	Haemodialysis (No. of patients)	Conservative (% of patients)	Haemodialysis (% of patients)
Acute Diarrheal Disease	45	11	80.36	19.64
Sepsis	15	9	62.50	37.50
Malaria	7	5	58.33	41.67
Liver Cirrhosis	5	3	62.50	37.50
Snakebite	3	1	75	25
Paraquat Poisoning	0	3	0	100
Aluminium Phosphide Poisoning	2	0	100	0
Pigment Nephropathy	0	2	0	100
NSAID Induced	1	0	100	0
Total	78	34	69.64	30.36

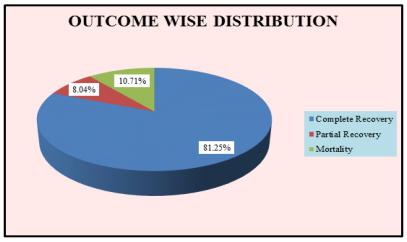


Dialysis was done as per the indications. In all, 34 patients (30.36%) required dialysis support and were treated by haemodialysis. The

remaining 78 patients (69.64%) were treated conservatively. The commonest indication for dialysis was symptomatic uraemia.

TABLE 10: OUTCOME WISE DISTRIBUTION

Outcome	No of Patients	(%) of patients
Complete Recovery	91	81.25%
Partial Recovery	9	8.04%
Mortality	12	10.71%
Total	112	100%



Regarding the outcome of AKI, 91 patients (81.25%) had complete recovery (CR) and 9 patients (8.04%) had partial recovery (PR). The overall in hospital mortality (IHM) rate was 10.71%.

DISCUSSION

AGE :

	Present Study	Bhadade et al	Maulita et al
Mean Age(in years)	43.47	42.64	44.5

Almost 50% of our patients were in the 3rd and 4th decades. The mean age of the population was 43.47 ± 12.73 years. Median age was 42 years. Only 12.5% patients were more than 60 yrs of age which have the least incidence. This is in concordance with almost all previous studies.

SEX :

	Present Study	Bhadade et al	Prakash Shende et al	Maulita et al
Male	63.39%	66.7%	56%	73%
Female	36.61%	33.3%	44%	27%

In our study, out of 112 patients, 71 (63.39 %) were males while 41 (36.61%) were females. Results are comparable with previous studies.

CLINICAL FEATURES :

	Present Study	Prakash Shende et al	Maulita et al
Oliguria	84.82%	66%	63%
Vomiting	82.14%	80%	81%
Diarrhea	50%	22%	18%
Fatigue	48.21%	62%	-
Breathlessness	38.39%	72%	13%
Altered sensorium	20.54%	-	8%
Fever	33.04%	56%	61%
Jaundice	24.11%	22%	5%
Edema	19%	-	37%

The common clinical features were oliguria (84.82%), vomiting (82.14%), diarrhea (50.00%), and fatigue (48.21%). Edema was the least common symptom present in 16.96%. This means that most of our patients presented early in the course of illness. This also reflects the adequacy of referral services.

Based on urine volume, patients were classified into oliguric and non-oliguric groups. In our study, oliguric AKI predominates (84.82%). This is in concordance with a previous study Prakash Shende et al and Maulita et al.

CO-MORBIDITIES :

	Present Study	Maulita et al	Prakash Shende et al
DM	9.82%	41.82%	28.3%
HTN	5.36%	24.09%	34.7%
IHD	3.57%	16.36%	30.4%

Co-morbidities were present in 20.54% of patients. Diabetes was the commonest (9.82%), followed by hypertension (HTN) (5.36%) and ischemic heart disease (IHD) (3.57%).

In present study co-morbidities were observed less compare to other studies.

ETIOLOGY :

	Present Study	P.VijayAnanth et al	Kumar et al	Maulita et al
Acute diarrheal disease	50%	44%	20.60%	11%
Sepsis	21.43%	10%	32.40%	48%
Malaria	10.71%	8%	0.9%	9%
Liver Cirrhosis	7.14%	-	-	7%
Snake bite	3.57%	5%	4%	2%
Paraquat poisoning	2.68%	-	-	-
Aluminium poisoning	1.79%	-	-	-
Pigment nephropathy	1.79%	-	-	-
Drug induced	0.89%	2%	-	6%

In our study, AKI due to acute diarrheal disease was most common in the 3rd decade (30.3%). Males (64.29%) were commonly affected by acute diarrheal disease than females. Because they are the bread winners of the family, they are used to consuming outside food. Acute diarrheal disease was the leading cause of AKI in our study accounting for 50% of cases, which is in concordance with study of P. Vijay Ananth et al.

TIMING OF DIAGNOSIS:

50.89% patients developed AKI after admission. This may be due to multiple risk factors. 49.11% of patients had AKI on admission. Measures should be aimed at prevention of AKI with early initiation of treatment and referral from the community and primary healthcare to tertiary hospital level.

DIALYSIS:

	Present Study	Prakash Shende et al	Ananth PV et al	Maulita et al
Conservative	69.64%	76%	57.7%	83.0%
Haemodialysis	30.36%	24%	38.5%	17.0%
Peritoneal Dialysis	-	-	3.84%	

OUTCOME:

	Present Study	Yousuf Khan et bal	Prakash Shinde et al	Maulita et al
Recovery	89.09%	90%	92%	93%
Mortality	10.71%	10%	8%	7%

CONCLUSION

- Timely diagnosis, early referral and prompt treatment of acute kidney injury confer a favourable prognosis to the patient. The progression of oliguric AKI with higher AKIN stage leads to increased morbidity & mortality. Proper management of acute diarrheal disease at periphery level can reduce the patients developing AKI. All malaria patients with decreased urine output should be screened early for AKI and early referral to higher centre may decrease the burden of AKI in malaria.

REFERENCES

1. Alan F Almeida, Jatin P Kothari, Acute kidney injury; API Text book of medicine; Y P Munjal: 9th edition; 2012:Jaypee; PP 1291.
2. SushrutS.Waikar, Joseph V.Bonventre-Acute Kidney Injury In Dan L.Lango, Dennis L Kasper., Joseph Loscalzo, J.Larry Jameson et.al eds. Harrison's principles of internal medicine, 18th ed. New york,Mcgrawhill,2012 :PP2294.

3. Utas C, Yalcindag C, Taskapan H, Guven M, OymakO,Yucesoy M. Acute renal failure in central Anatolia. Nephrol Dial Transplant.2000;15:152–155.
4. Muthusethupathi MA, Shivakumar SJ. Acute renal failure in South India. J Assoc of Physicians India. 1987;36(7):504–507
5. Prakash J, Tripathi K, Malhothra V, Kumar O, Srivatsava PK. Acute renal failure in Eastern India. Nephrol Dial Transplant. 1995;10:2009–2012.
6. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P, and the Acute Dialysis Quality Initiative workgroup. Acute renal failure— definition, outcome measures, animal models, fluid therapy and information technology needs: the second international consensus conference of the Acute Dialysis Quality Initiative Group. Crit Care 2004; 8: R204–R12.
7. Kellum JA, Ronco C, Mehta R, Bellomo R: Consensus development in acute renal failure: The Acute Dialysis Quality Initiative. CurrOpinCrit Care 2005, 11:527-532.
8. Leblanc M, Kellum JA, Gibney RT, Lieberthal W, Tumlin J, Mehta R: Risk factors for acute renal failure: inherent and modifiable risks. CurrOpinCrit Care 2005, 11:533-536.
9. Mehta RL, Kellum JA, Shah SV et al. Acute kidney injury network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007; 11: R31
10. Hou SH, Broc ME. Hospit al acquired renal indifficiency. A prospective study. American journal of medicine 1983 ; 74 : 243 - 248.
11. Lamiere N, Vanbiesen W, VanholderR. Acute renal failure. Lancet. 2005;365:417–430.
12. Brady HR, Singer GG: Acute renal failure. Lancet 1995; 346:1533-1540
13. Klahr S, Miller SB: Acute oliguria. N Engl J Med 1998; 338:671-675.
14. Barry M. Brenner, Floyd C. Rector. Brenner and Rector's The Kidney. 8th ed. Saunders Elsevier, 2008.
15. Mehta RL, McDonald B, Gabbai FB, et al: A randomized clinical trial of continuous versus intermittent dialysis for acute renal failure. Kidney Int 2001; 60:1154-1163.
16. Kellum JA, Levin N, Bouman C, et al: Developing a consensus classification system for acute renal failure. CurrOpinCritCare 2002; 8:509-514.
17. Thakar CV, Christianson A, Freyberg R, et al. Incidence and outcomes of acute kidney injury in intensive care units: a Veterans Administration study. Crit Care Med 2009; 37: 2552–2558.
18. Bagshaw SM, George C, Bellomo R, and the ANZICS Database Management Committee. Early acute kidney injury and sepsis: a multicentre evaluation. Crit Care 2008; 12: R47
19. Liangos O, Wald R, O'Bell JW, Price L, Pereira BJ, Jaber BL. Epidemiology and outcomes of acute renal failure in hospitalized patients: a national survey. Clin J Am SocNephrol 2006; 1: 43–51.
20. Longo D, Fauci A, Kasper D, Hauser S, Jameson J, Loscalzo J. Harrison's Principles of Internal Medicine. 19th ed. New York, NY: McGraw-Hill Professional;2017.
21. Nash K, Hafeez A, Hou S: Hospital-acquired renal insufficiency. AmJ Kidney Dis 2002; 39:930-936.
22. Metcalfe W, Simpson M, Khan IH, et al: Acute renal failure requiring renal replacement therapy: incidence and outcome. QJM 2002; 95:579-583.
23. Kon V, Yared A, Ichikawa I: Role of renal sympathetic nerves in mediating hypoperfusion of renal cortical microcirculation in experimental congestive heart failure and acute extracellular fluid volume depletion. J Clin Invest 1985;76:1913-1920.
24. Thadhani R, Pascual M, Bonventre JV: Acute renal failure. N Engl J Med 1996; 334:1448-1460.
25. Kontogiannis J, Burns KD: Role of AT1 angiotensin II receptors in renal ischemic injury. Am J Phtsiol 1998; 274:F79-F90.
26. Badr KF, Ichikawa I: Prerenal failure: a deleterious shift from renal compensation todecompensation. N Engl J Med 1988; 319:623-629.
27. Fisch BJ, Linas LL: Prerenal acute renal failure. In: Brady HR, Wilcox CS, ed. Therapy in Nephrology and Hypertension, Philadelphia: W.B. Saunders; 1998:17-20.
28. Wali RK, Henrich WL: Recent developments in toxic nephropathy. CurrOpinHypertenNephrol2002; 11:155-163.
29. Gutthann SP, Rodriguez LAG, Raiford DS, et al: Nonsteroidal anti-inflammatory drugs and the risk of hospitalization for acute renal failure. Arch Int Med 1996; 156:2433-2439.
30. Textor SC: Renal failure related to angiotensin-converting enzyme inhibitors. SeminNephrol1997; 17:67-76.