Acute Kidney Injury

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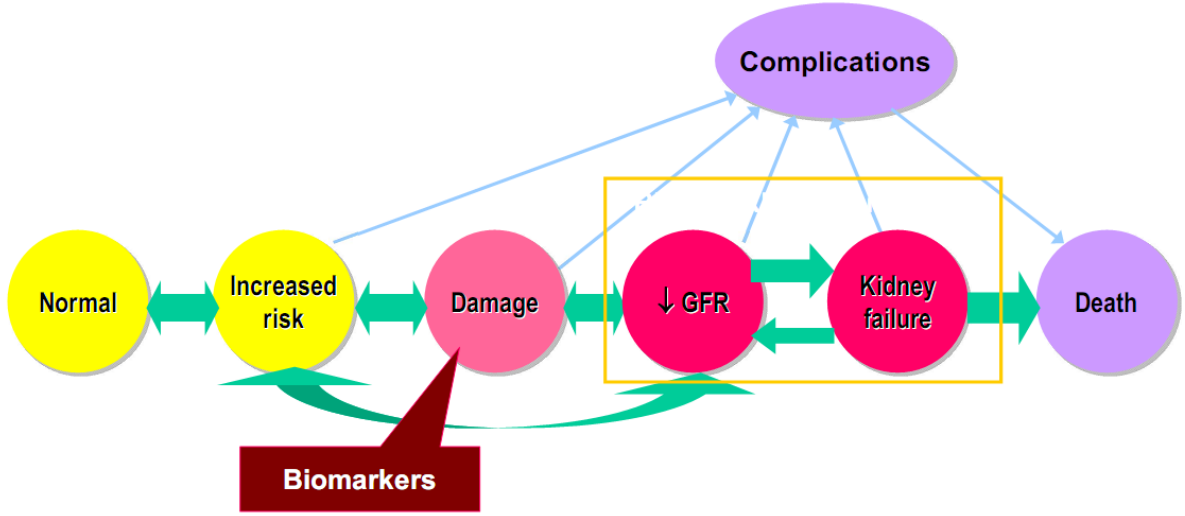
Acute Kidney Injury, AKI

- AKI is a common problem, especially in the critical care setting
- It is a complex disorder for which there was no accepted definition
- acute renal failure (ARF) fails to adequately describe the dynamic process
- Reported incidence and mortality varies widely
  - Incidence ranges 1-31%
  - Mortality ranges 28-82%

    poor prognosis

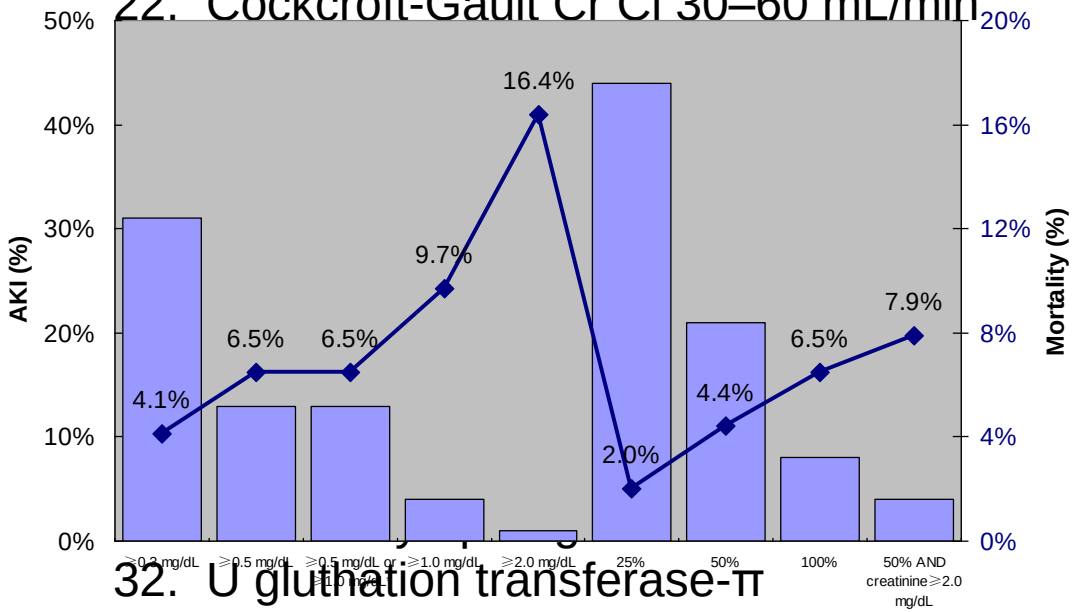
    increased cost, LOS……



DEFINITION AND CLASSIFICATION

Definitions of AKI and incidence

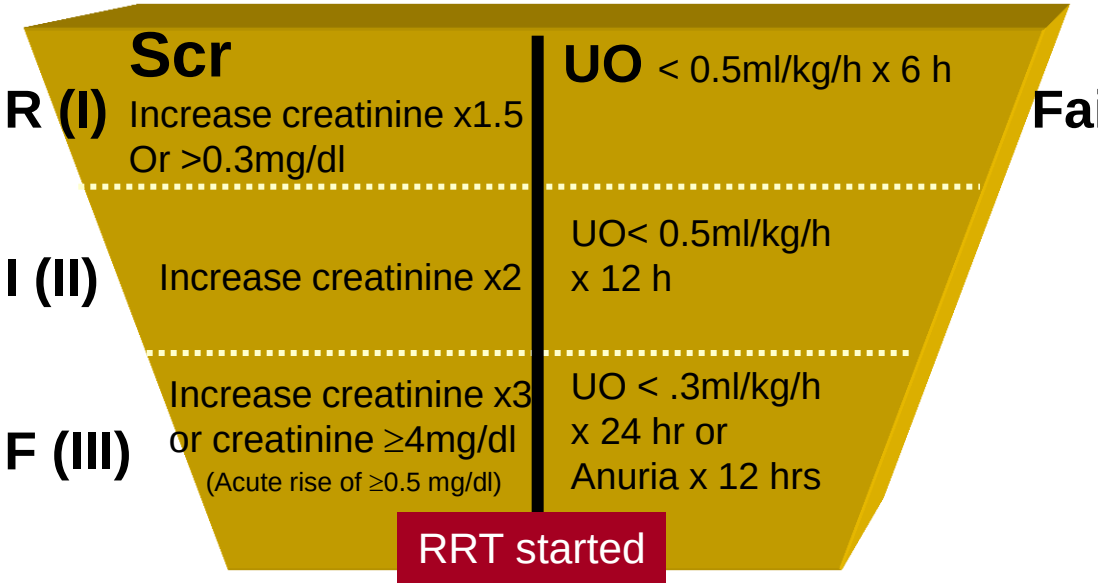
1. Creat Δ 0.1 mg/dL
2. Creat increase >0.5 mg/dL
3. Creat>= 0.5 mg/dL
4. Creat >= 1.7 mg/dL
5. Creat >= 1.5 mg/dL
6. Creat >= 2 mg/dL
7. Creat>= 2.1 mg/dL and x 2
8. Creat >= 177μmol/L Δ>62μmol/L
9. Creat > 200μmol/L (2.36 mg/dL)
10. Creat> 3.2 mg/dL or x 2
11. Creat>5 mg/dL or K > 5.5
12. RIFLE
13. Creat increase >= 25%
14. Creat increase >= 50%
15. Creat increase >= 100%
16. ΔCr72h >0μmol/L
17. ΔCr72h >25μmol/L
18. ΔCr72h >44μmol/L
19. ΔCr72h >50μmol/L
20. ΔCr72h >100μmol/L
21. Cockcroft-Gault Cr Cl < 30 mL/min
22. Cockcroft-Gault Cr Cl 30–60 mL/min



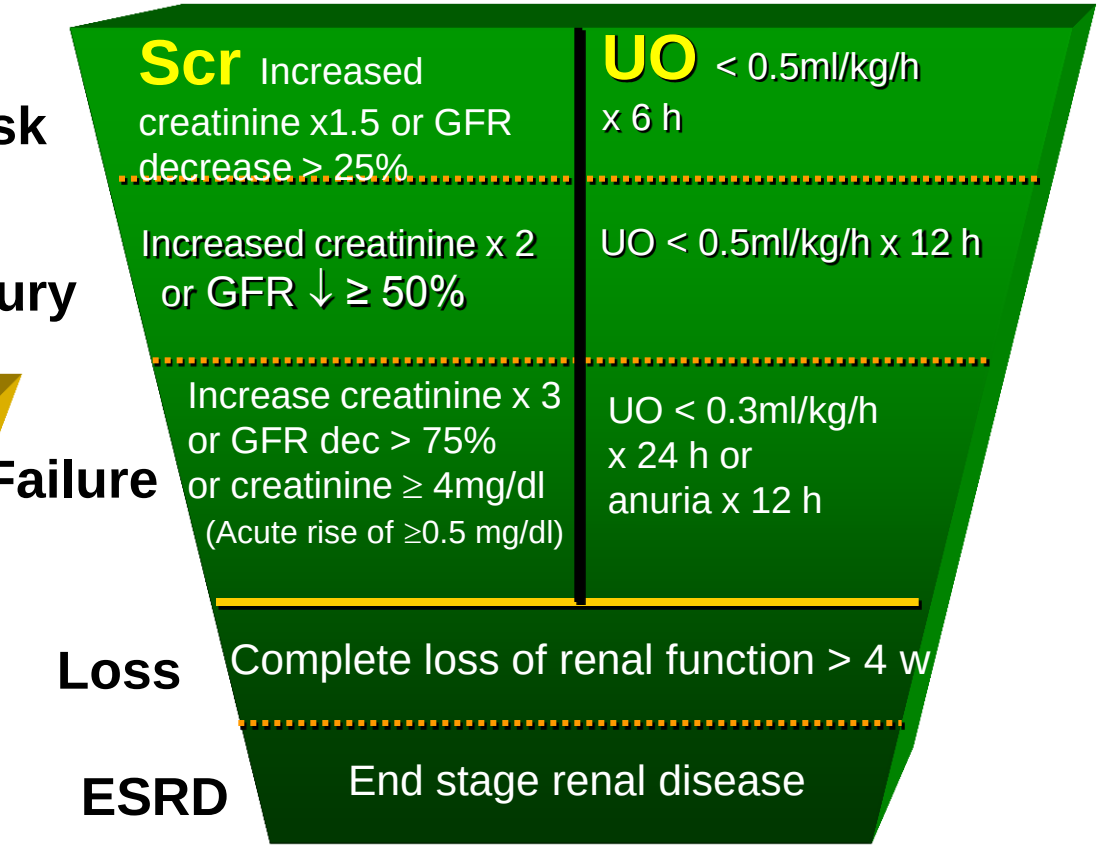
32. U gluthation transferase-π
33. U gluthation transferase- α
34. NGAL
35. RRT
36. …

Current criteria for AKI diagnosis

- Serum Creatinine
- Urine output



AKIN criteria



RIFLE criteria

KDIGO CLINICAL PRACTICE GUIDELINE FOR ACUTE KIDNEY INJURY

AKI is defined as any of the following (*Not Graded*):

- **Increase in SCr by ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours; or**
- **Increase in SCr to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or**
- **Urine volume <0.5 ml/kg/h for 6 hours.**

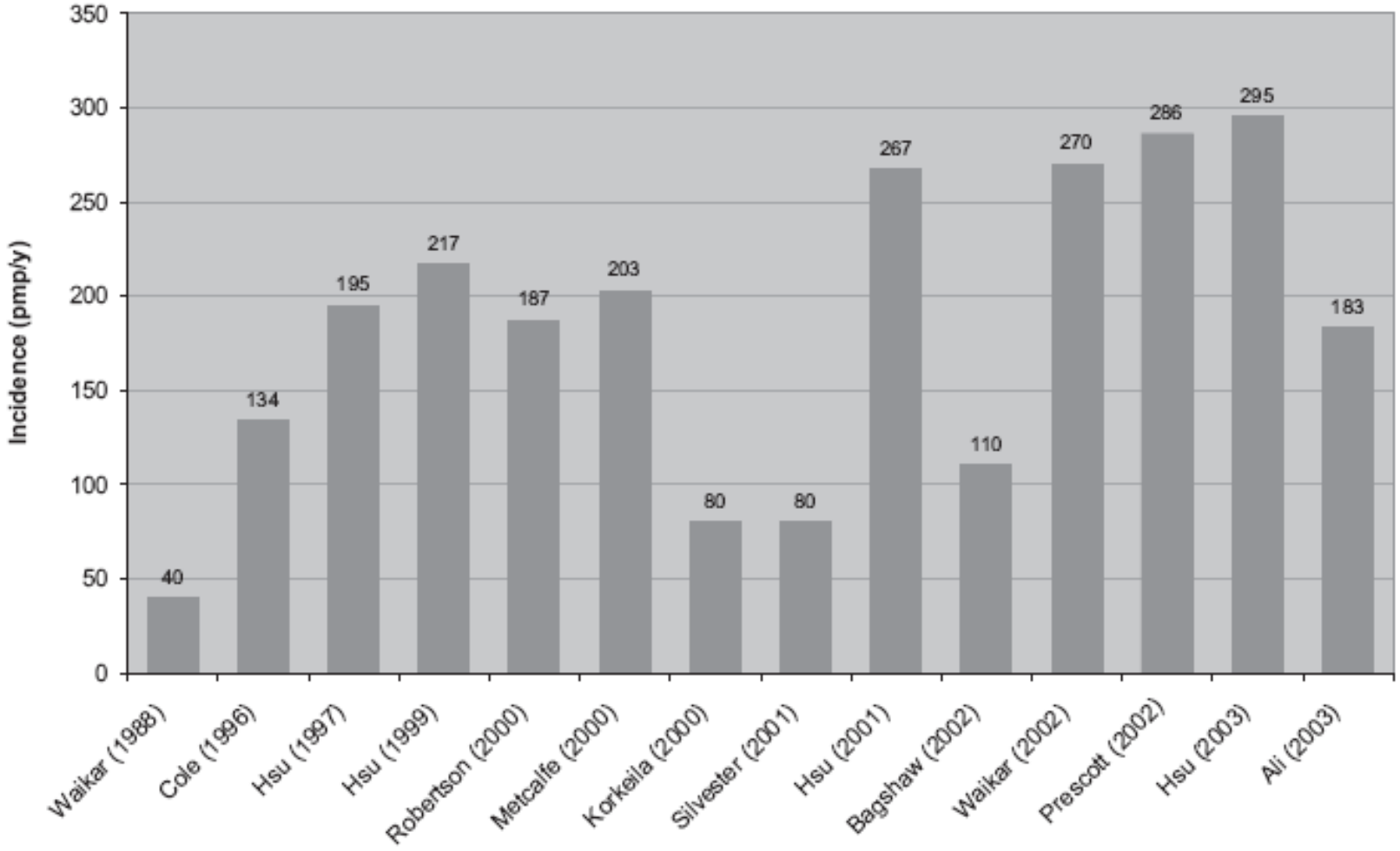
AKI is staged for severity according to the following criteria (Table 1). (*Not Graded*):

Table 1. 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

INCIDENCE

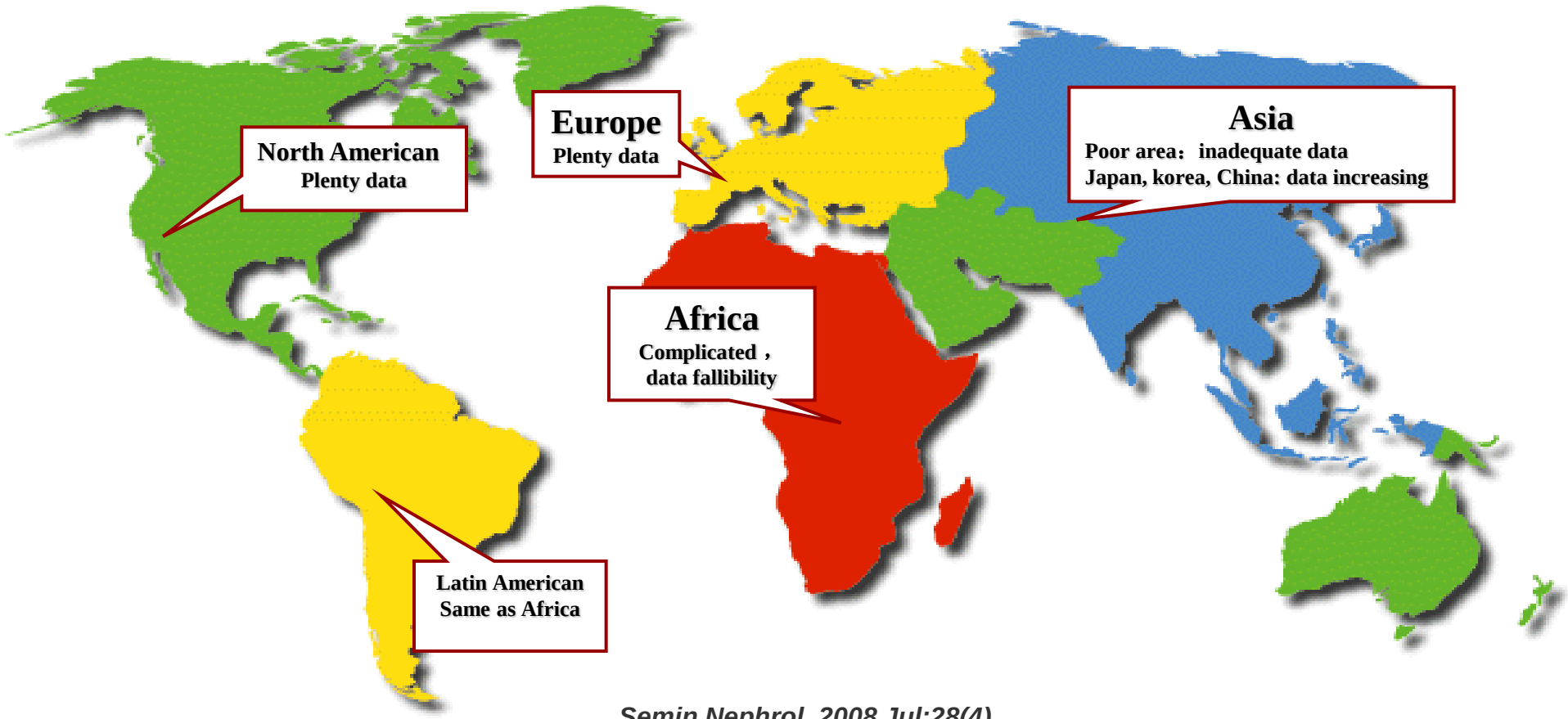
Incidence of AKI in general population



Crit Care Med, 2008,36(4 Suppl):S146-51

AKI defined as RRT requirement

Continent specific AKI



Semin Nephrol. 2008 Jul;28(4)
Nephron Clin Pract. 2008,109(4)
Korean J Intern Med. 2010, 25(2)

RIFLE studies

Patient populations

- Critically ill
  - General ICU
    - ID ICU
  - ICU treated w RRT
  - Cardiothoracic surgery
  - Elective abdominal aortic surgery
  - Stroke
  - Burns
  - BMT
  - Liver transplant
  - Cirrhosis
  - Pediatric ICU
- Outside the ICU
  - Hospital admissions
  - Population-based

Criteria used

- ΔCr only
- ΔGFR only
- ΔCr/GFR + UO

When AKI was staged

- At ICU admit
- RIFLE initial
- RIFLE max
- At renal consult
- At start of RRT

Mortality endpoints

- ICU
- Hospital
- 28d, 30d, 60d, 6 mo, 1 yr

AKI in hospitalized patients

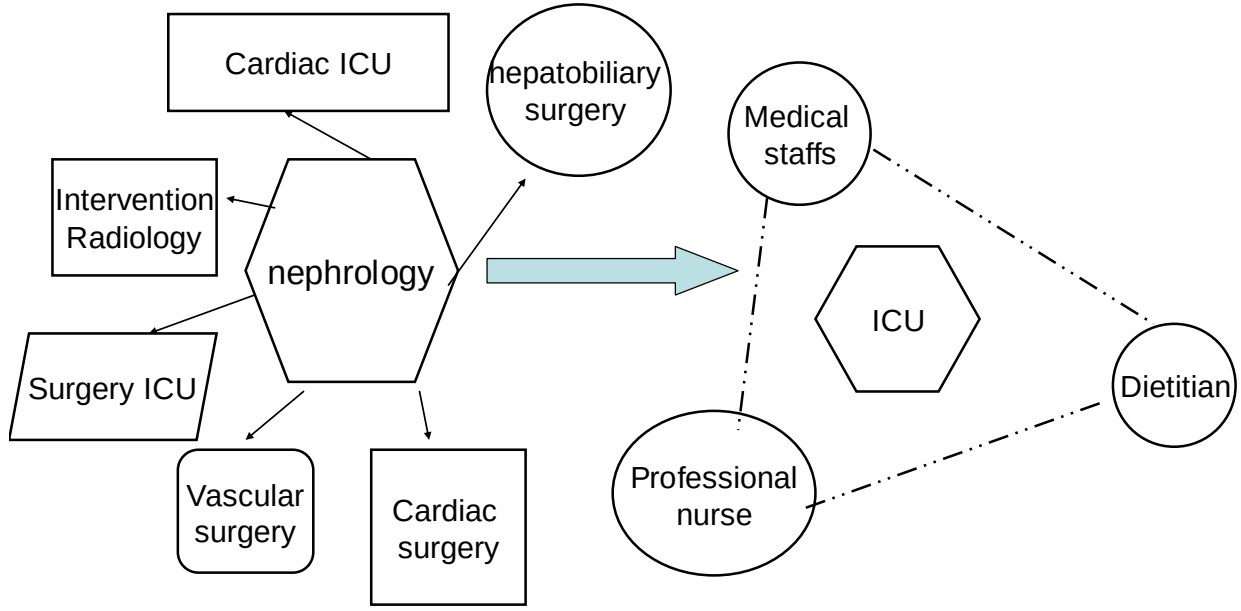
—— from published literatures

First Author (Reference No.)	Cohort	Patients, n	AKI, %	Risk, %	Injury, %	Failure, %
Cruz (14)	ICU	2,164	10.8	2.1	3.8	4.9
Heringlake[b] (51)	CS-ICU	29,623	16	9	5	2
Uchino[b] (44)	Hospital	20,126	18	9.1	5.2	3.7
Kuitunen (43)	CS-ICU	813	19.1	10.8	3.4	4.9
Lopes (48)	BMT	140	33.5	13.5	10	14.3
Lopes[b] (46)	Burn	126	35.7	14.3	8.7	12.7
Ostermann[b] (52)	ICU	41,972	35.8	17.2	11	7.6
O'Riordan[b] (53)	Liver Tx	359	35.9	NA	10.9	25.1
Lopes[b] (47)	Sepsis	182	37.4	6.0	11.5	19.8
Lopes[b] (45)	HIV-ICU	97	47.4	12.4	9.6	25.8
Ahlstrom (54)	ICU	685	52.0	25.5	15.2	11.2
Guitard[b] (55)	Liver Tx	94	63.8	NA	41.5	22.3
Hoste (11)	ICU	5,383	67	12.4	26.5	28.1
Lin (56)	ICU-ECMO	46	78.2	15.2	39.1	23.9
Abosaif (57)	ICU-AKI	183	86.9	32.8	30.6	23.5
Bell[b] (15)	ICU-RRT	207	90.8	8.2	24.2	58.5
Maccariello (16)	ICU-RRT	214	100	25.0	27.0	48.0

AKI was diagnozed with RIFLE criteria

Hoste, et al. *Crit Care 2006;* ***Cruz, et al.*** *Clin JASN, 2007;* ***Maccariello, et al.*** *Intensive Care Med 2007;* ***Bell, et al.*** *Nephrol Dial Transplant, 2005;* ***Lopes, et al.*** *Crit Care, 2007;* ***Heringlake, et al.*** *Minerva Anestesiol 2006;* ***Kuitunen, et al.*** *Ann Thorac Surg 2006;* ***Uchino, et al.*** *Crit Care Med 2006;* ***Osterman, et al.*** *Crit Care Med* 2007; **O'Riordan , et al.** *Crit Care Med 2007;* ***Ahlstrom , et al.*** *AJKD, 2006;* ***Guitard, et al.*** *Clin Nephrol, 2006; Lin, et al. Nephrol Dial Transplant, 2006;* ***Lopes, et al.*** *Nephrol Dial Transplant, 2007;* ***Abosaif, et al.*** *AJKD 2005*

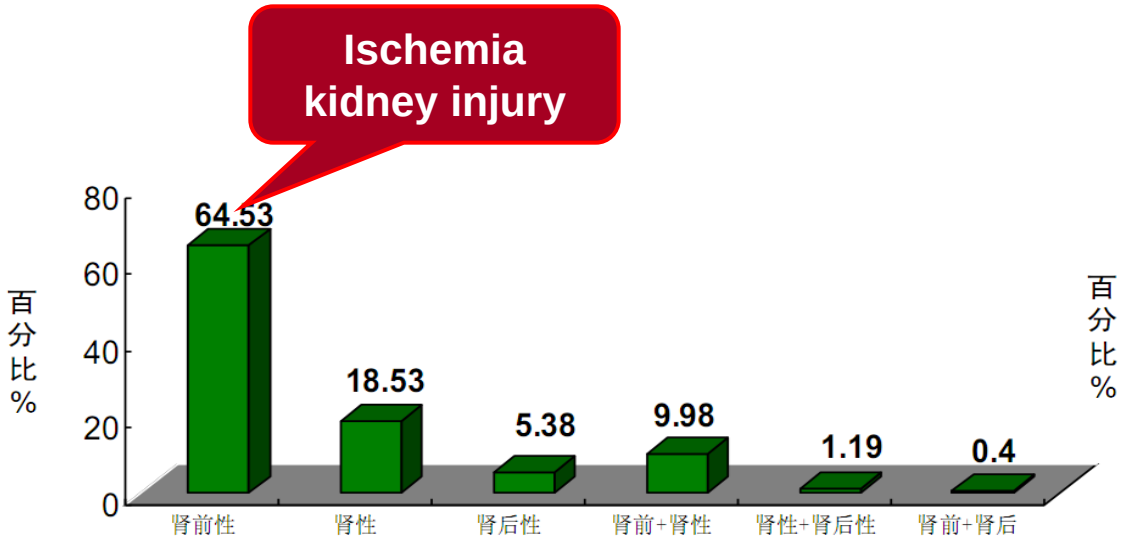
Epidemiologic studies on AKI

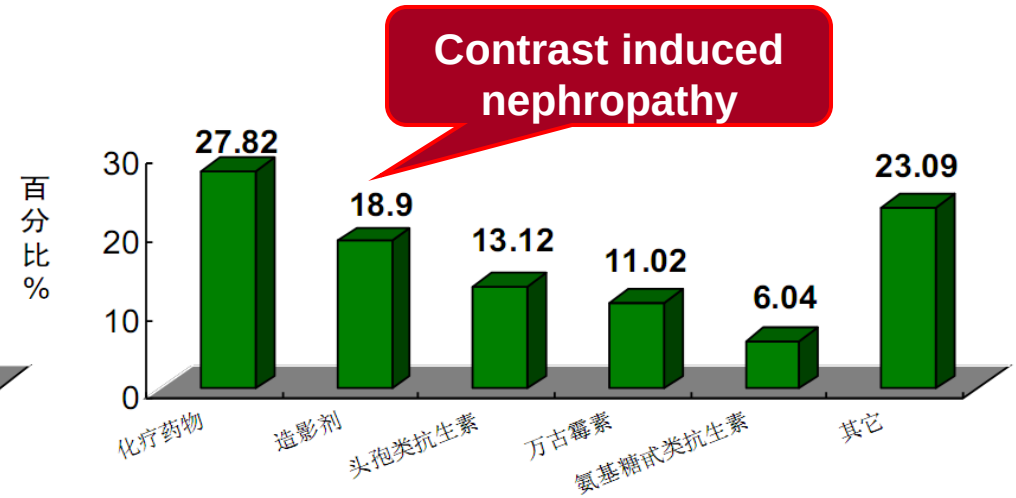


AKI registration systems → **AKI cooperative network**

2004-2008， 17,6155 admissions

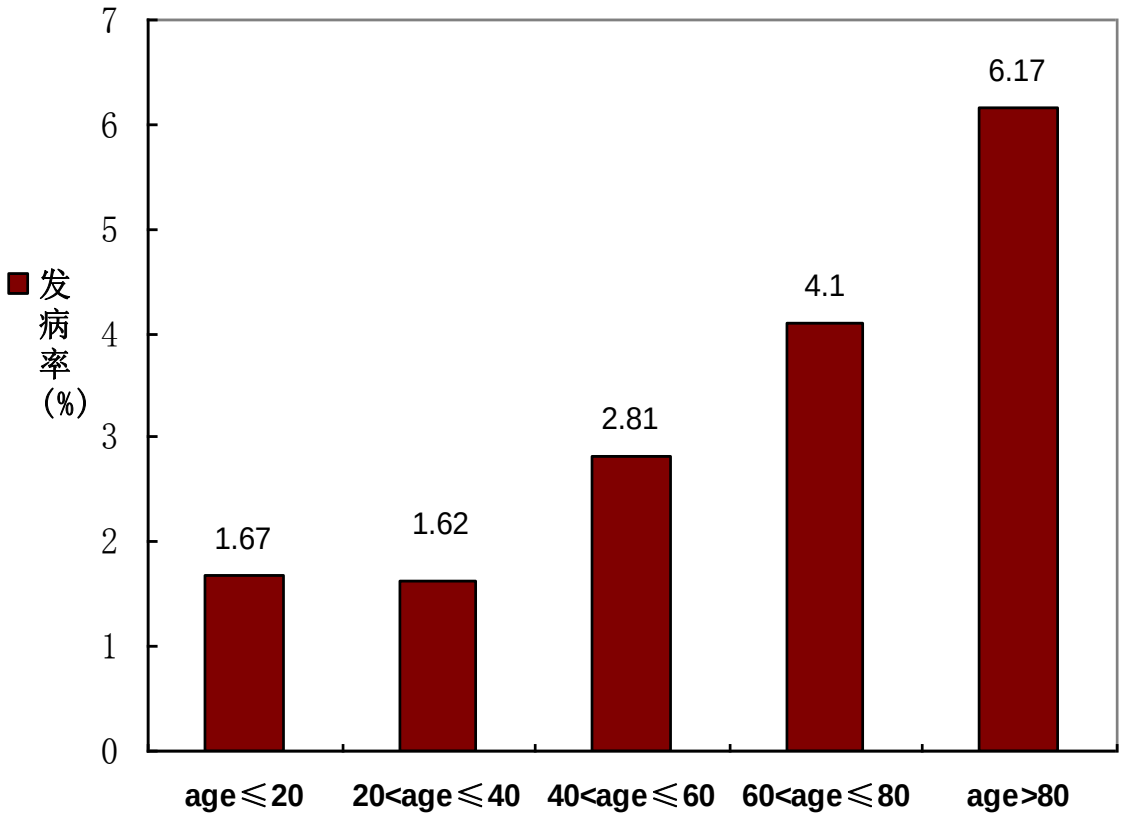
Incidence of AKI 3.19%

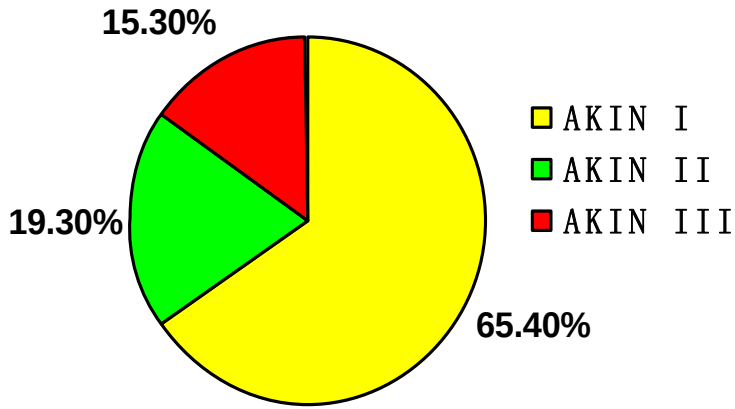




Yi Fang, Xiaoqiang Ding, et al. Blood Purification, 2010

AKI in hospitalized population





AKI stages during intensive care unit stay (n = 937).

Data were collected on a consecutive series of hospitalized patients between September 2004 and June 2008

Fang Y, Ding XQ, et al. Chinese Journal of nephrology, 2007
Fang Y, Ding XQ, et al. Blood Purification, 2010

LOS and costs associated with AKIN staging

AKIN stage I mortality 7.0%
non-AKI mortality 2.3%

Criterion	Mortality rate	[illegible] (y)	[illegible]* (RMB)	LOS*
AKIN stage I	7.0%	65.26±15.07	21791.82 (11369.66~41761.1)	17.5(11.5~27.0)
AKIN stage II	49.5%	60.66±16.71	32510.54 (17967.86~72251.08)	22.0(14.0~32.5)
AKIN stage III	66.7%	58.74±16.32	34243.59 (15936.56~85413.71)	23.0(12.5~36.6)

*Non-normal distribution, described as median (with interquartile range in parentheses); data were log transformed before ANOVA test.

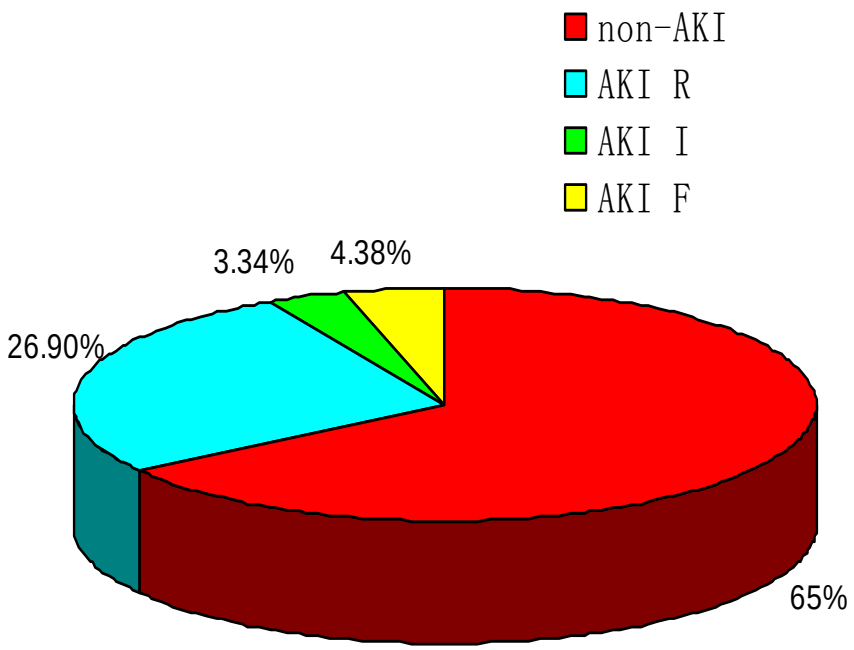
Yi Fang, Xiaoqiang Ding, et al. Blood Purification, 2010

Prognostically significant variables for AKI mortality

Parameters	β-coefficient	OR 95% CI	*p*值
age（every 10 years）	0.15	1.10(1.02~1.35)	0.007
AKIN classification	0.37	1.45(1.17-2.660	0.004
septic shock	0.64	2.87(1.07-4.90)	0.003
OSF no	0.43	1.78(1.33~2.51)	0.009
ICU admission	0.33	2.23(1.20-3.98)	0.026
Need for RRT	0.28	2.47(1.95-3.39)	0.008

Yi Fang, Xiaoqiang Ding, et al. Blood Purification, 2010

AKI after cardiac surgery



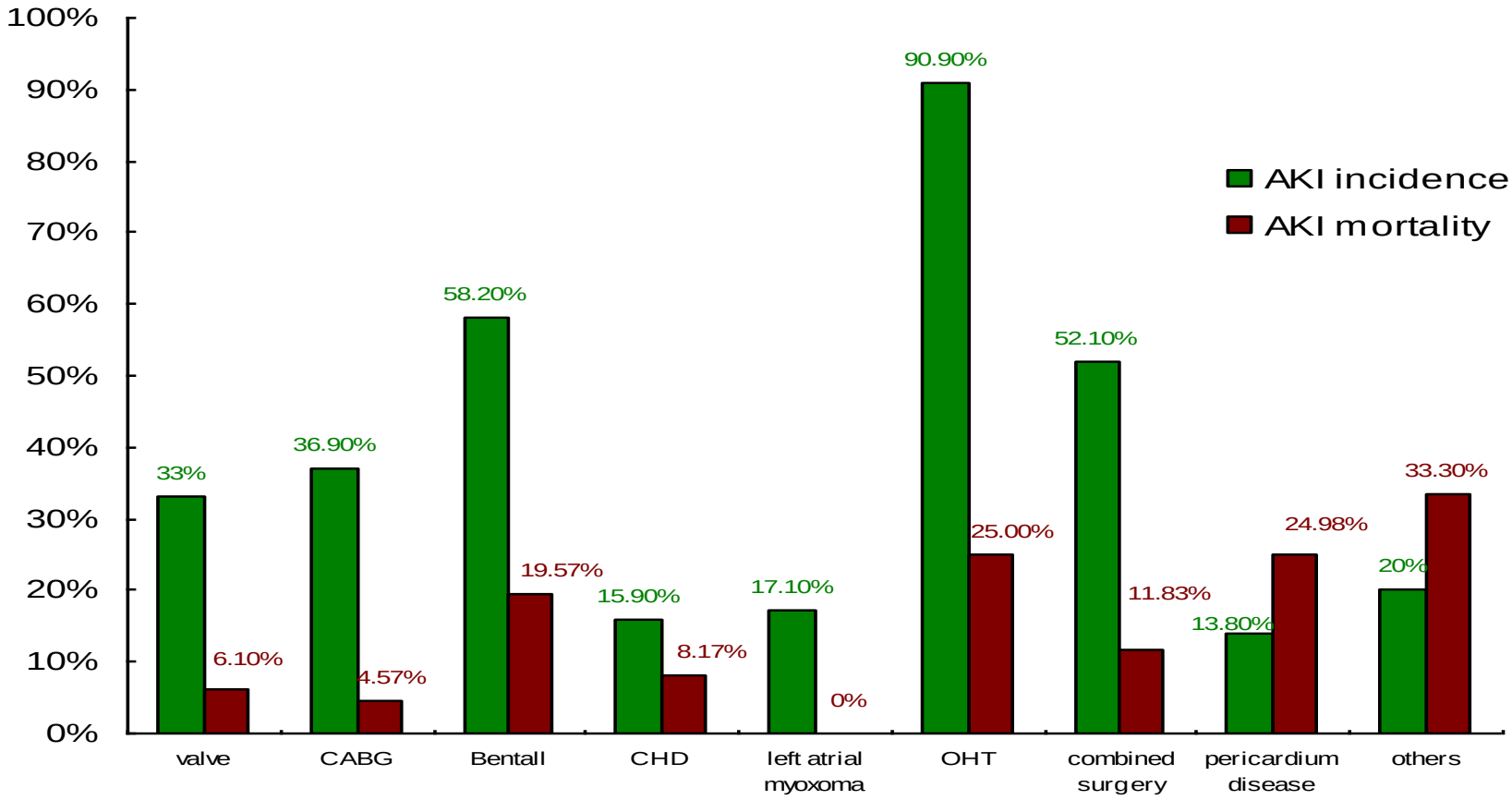
Data were collected on a consecutive series of hospitalized patients between September 2004 and June 2008

AKI was staged with RIFLE criteria

Mortality of AKI: 35.0%

Heng YY, Ding XQ, et al. Chinese Journal of nephrology, 2011

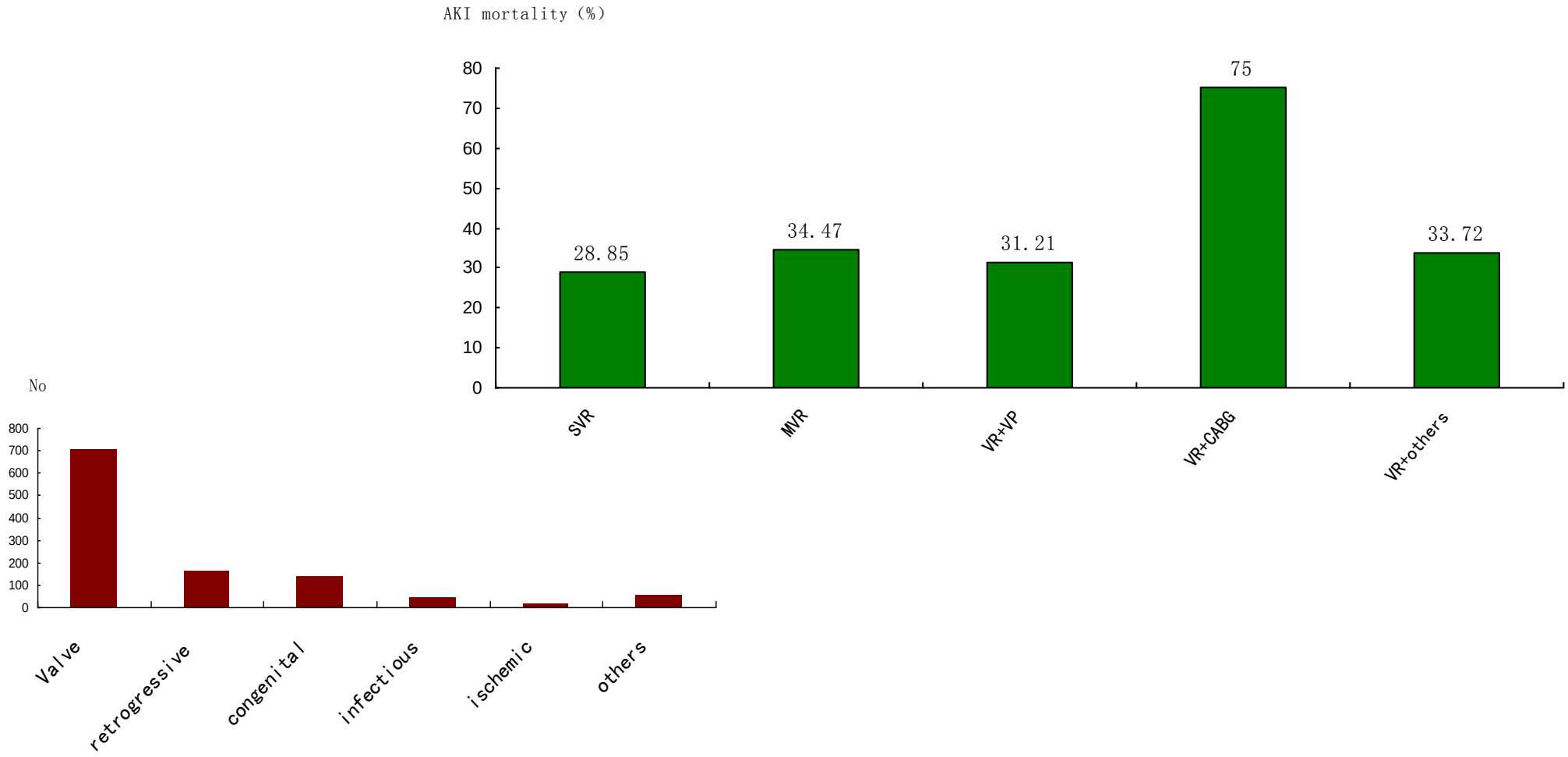
AKI after cardiac surgery



AKI was staged with RIFLE criteria

Heng YY, Ding XQ, et al. Chinese Journal of nephrology, 2011

AKI after cardiac valve surgery



Heng YY, Ding XQ, et al. Chinese Journal of nephrology, 2011

ETIOLOGY

Classification and Major Disease Categories Causing AKI

Historically, AKI was classified as

- Nonoliguric (urine output > 400mL/day)
- Oliguric (urine output < 400mL/day)
- Anuric (urine output < 100mL/day)

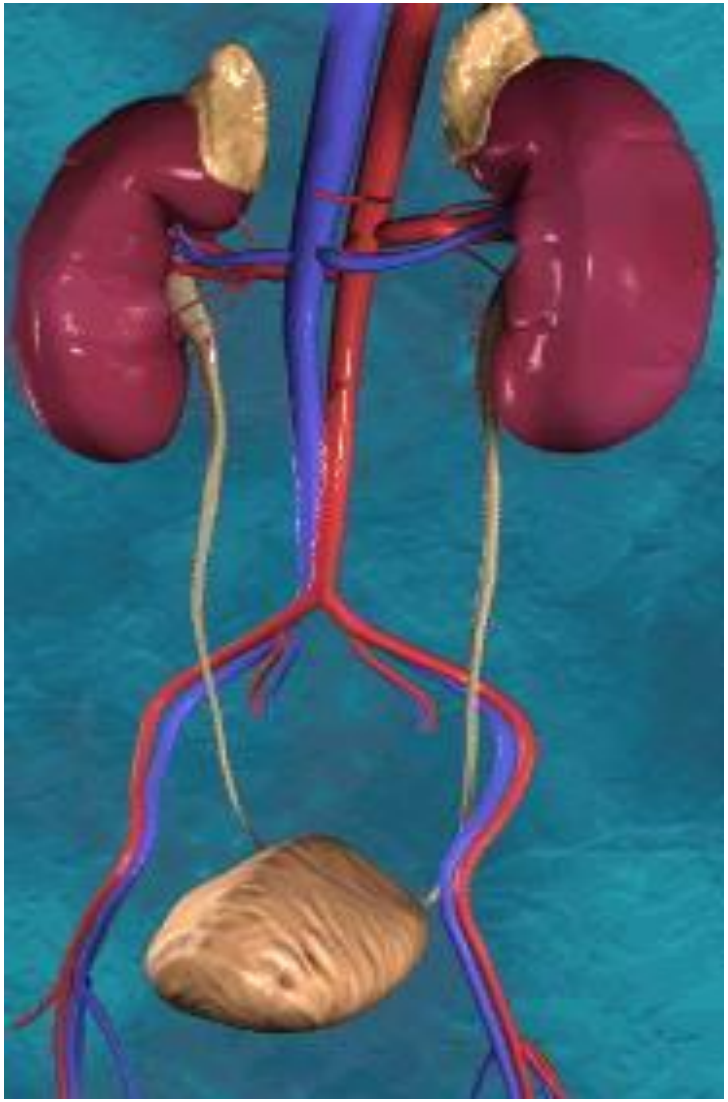
Classification and Major Disease Categories Causing AKI

Disease Category	Percentage of Patients with AKI
Prerenal azotemia caused by acute renal hypoperfusion	**55-60**
Intrinsic renal azotemia caused by acute diseases of renal parenchyma	**35-40**
• Diseases involving large renal vessels	
• Diseases of small renal vessels and glomeruli	
• Acute injury to renal tubules mediated by ischemia or toxins	
• Acute diseases of the tubulointerstitium	
Postrenal azotemia caused by acute obstruction of urinary collecting system	**<5**

main causes & clinical features of AKI

- Prerenal AKI
- Intrinsic Renal AKI
- Postrenal AKI

Outcome： GFR↓



Major Causes of Prerenal Azotemia

Intravascular volume depletion

Hemorrhage: traumatic, surgical, gastrointestinal, postpartum

Gastrointestinal losses: vomiting, nasogastric suction, diarrhea

Renal losses: drug-induced or osmotic diuresis, diabetes insipidus, adrenal insufficiency

Skin and mucous membrane losses: burns, hyperthermia, and other causes of increased insensible losses

“Third-space” losses: pancreatitis, crush syndrome, hypoalbuminemia

Decreased cardiac output

Diseases of myocardium, valves, pericardium, or conducting system

Pulmonary hypertension, pulmonary embolism, positive-pressure mechanical ventilation

Systemic vasodilatation

Drugs: antihypertensives, afterload reduction, anesthetics, drug overdoses

Sepsis, liver failure, anaphylaxis

Major Causes of Prerenal Azotemia

Renal vasoconstriction

Norepinephrine, ergotamine, liver disease, sepsis, hypercalcemia

Pharmacologic agents that acutely impair autoregulation and glomerular filtration rate in specific settings

Angiotensin-converting enzyme inhibitors in renal artery stenosis or severe renal hypoperfusion

Inhibition of prostaglandin synthesis by nonsteroidal anti-inflammatory drugs during renal hypoperfusion

Major Causes of Intrinsic renal Azotemia

Ischemic ATN and toxic ATN account for about 80% to 90% of intrinsic AKI

Diseases characterized by prominent inju often with ATN

Ischemia caused by renal hypoperfusion

Exogenous toxins (e.g., antibiotics, anticancer agents, radiocontrast agents, poisons)

Endogenous toxins (e.g., myoglobin, hemoglobin, myeloma light chains, uric acid, tumor lysis)

Acute diseases of the tubulointerstitium

Allergic interstitial nephritis (e.g., antibiotics, nonsteroidal anti-inflammatory drugs)

Infectious (viral, bacterial, fungal)

Acute cellular allograft rejection

Infiltration (e.g., lymphoma, leukemia, sarcoid)

Major Causes of Intrinsic renal Azotemia

Diseases involving large renal vessels

Renal arteries: thrombosis, atheroembolism, thromboembolism, dissection, vasculitis (e.g., Takayasu)

Renal veins: thrombosis, compression

Diseases of glomeruli and the renal microvasculature

Inflammatory: acute or rapidly progressive glomerulonephritis, vasculitis, allograft rejection, radiation

Vasospastic: malignant hypertension, toxemia of pregnancy, scleroderma, hypercalcemia, drugs, radiocontrast agents

Hematologic: hemolytic-uremic syndrome or thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, hyperviscosity syndromes

Major Causes of Postrenal Azotemia

Ureteric obstruction

Intraluminal: stones, blood clot, sloughed renal papillae, uric acid or sulfonamide crystals, fungus balls

Intramural: postoperative edema after ureteric surgery, BK virus-induced ureteric fibrosis in renal allograft

Extraureteric: iatrogenic (ligation during pelvic surgery)

Periureteric: hemorrhage, tumor, or fibrosis

Bladder neck obstruction

Intraluminal: stones, blood clots, sloughed papillae

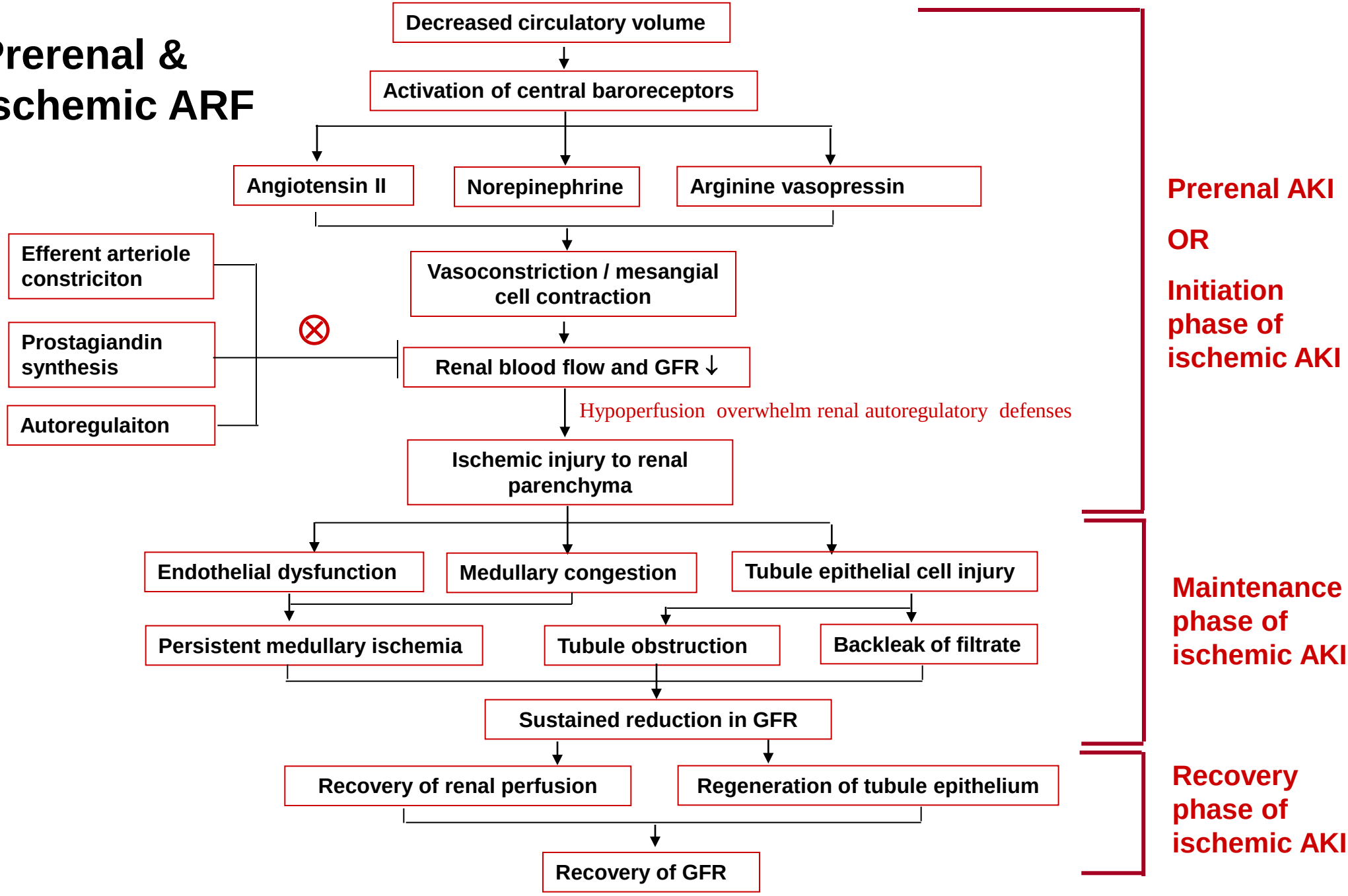
Intramural: bladder carcinoma, bladder infection with mural edema, neurogenic, drugs (e.g., tricyclic antidepressants, ganglion blockers)

Extramural: prostatic hypertrophy, prostatic carcinoma

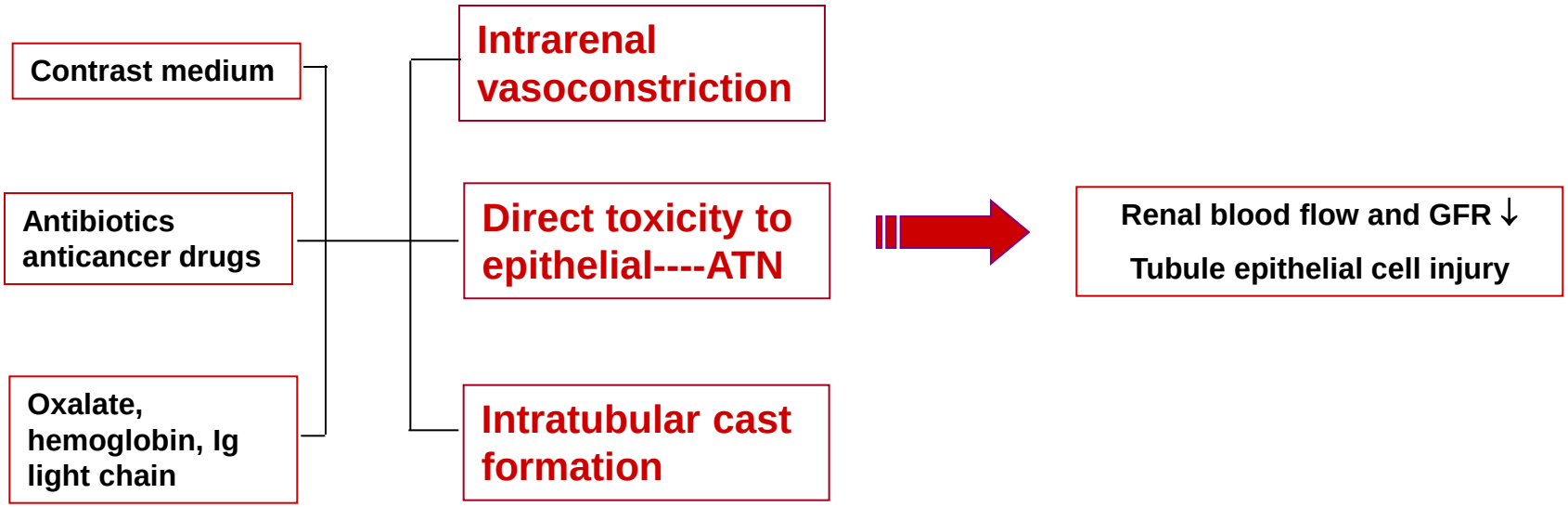
Urethral obstruction

Phimosis, congenital valves, stricture, tumor

Prerenal & Ischemic ARF



Nephrotoxic ARF

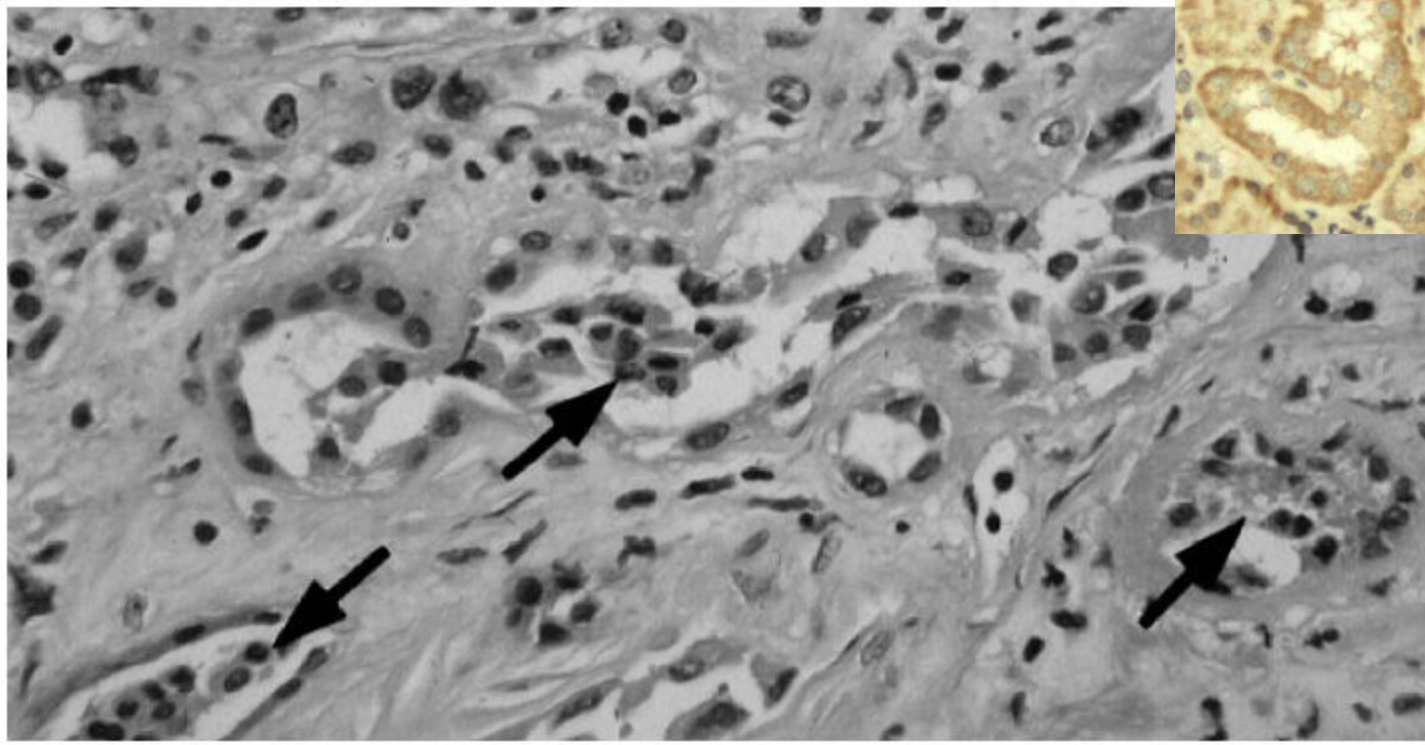


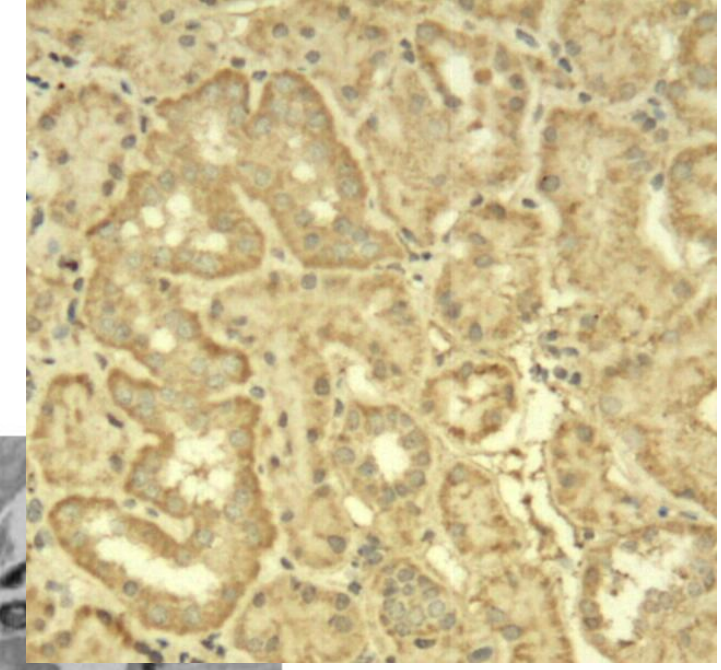
Pathology: ATN

Cell necrosis

Damage to the brush border of the proximal tubules

Accumulation of tubular casts

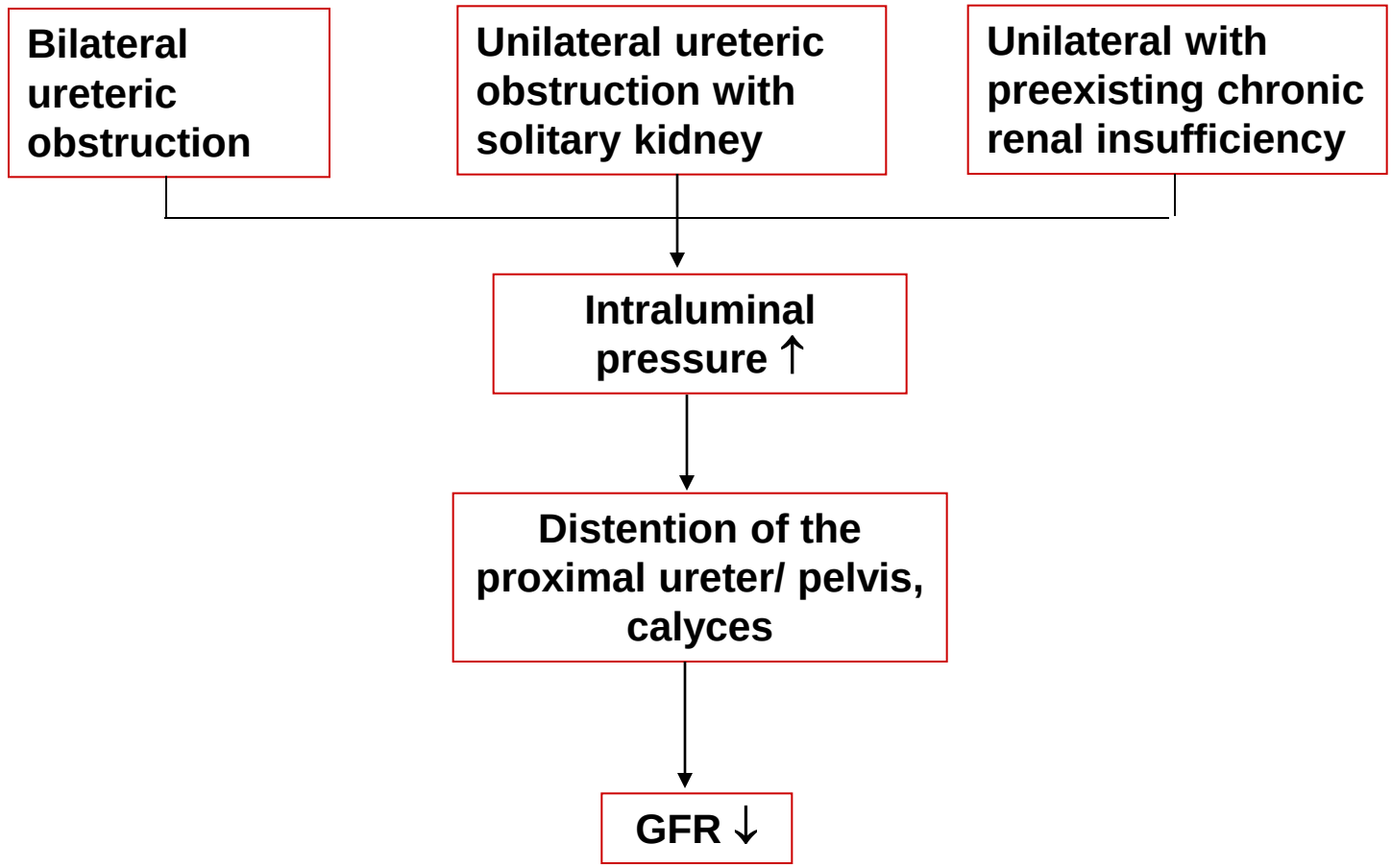




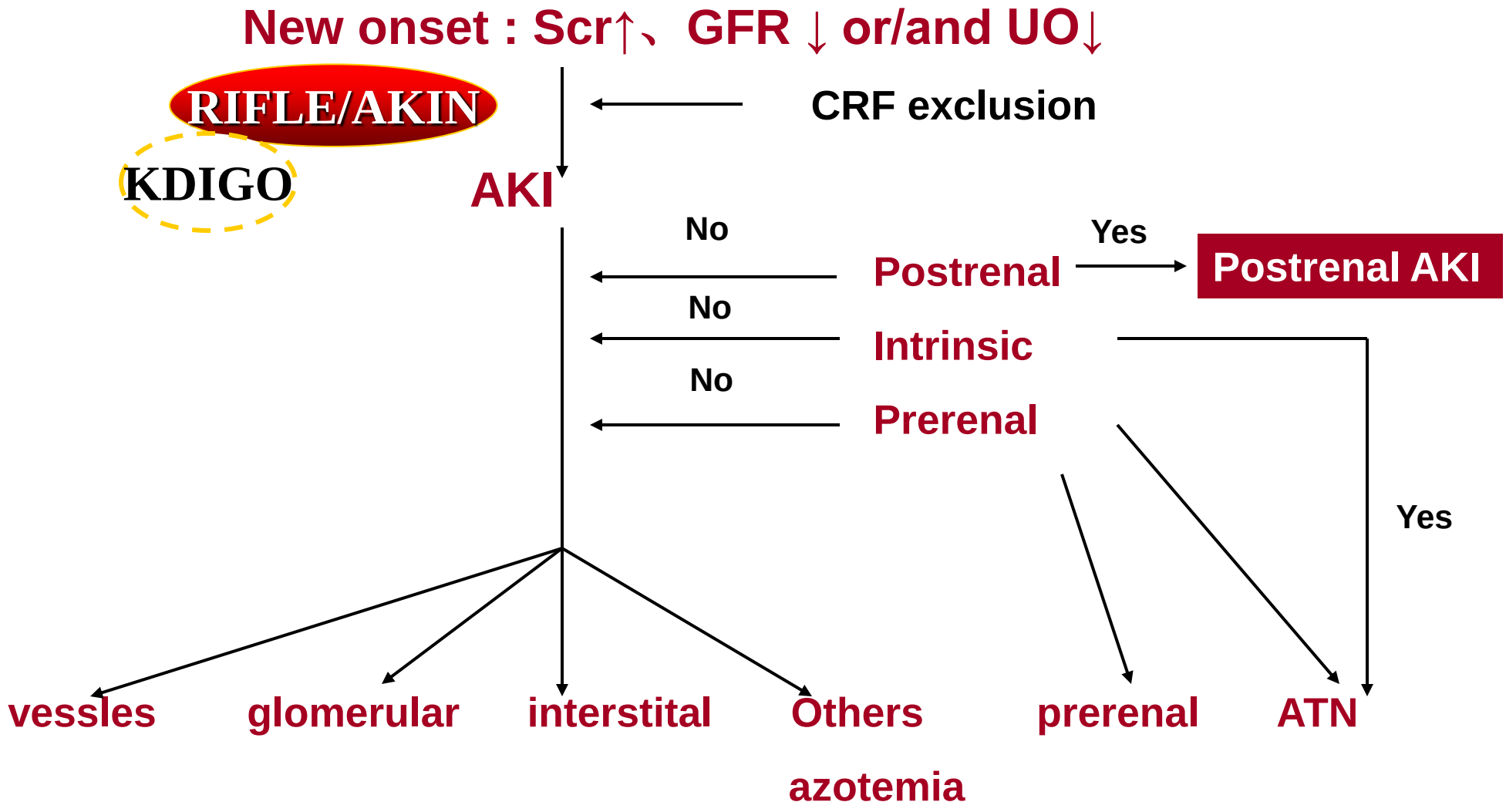
Normal (control)

FIGURE 29-2 Cellular cast formation *(arrows)* in renal tubules of a human renal biopsy with acute tubular necrosis. *(Courtesy of Dr. Yashpal Kanwar.)*

Post renal ARF



Diagnosis


New onset : Scr↑、GFR ↓ or/and UO↓
RIFLE/AKIN
KDIGO
CRF exclusion
AKI
No
Postrenal
Yes
Postrenal AKI
No
Intrinsic
No
Prerenal
Yes
vessles
glomerular
interstital
Others
azotemia
prerenal
ATN

Clinical Approach to the Diagnosis of Acute Kidney Injury

- **History, physical examination**
- **Urinalysis including specific gravity, dipstick, sulfosalicylic acid, microscopy, and staining for eosinophils**
- **Routine blood chemistry assays and hematologic tests**

  Urine chemistry, eosinophils, and/or immunoelectrophoresis

  Radiologic evaluation: plain abdominal film, renal ultrasonography, intravenous pyelography, renal angiography, magnetic resonance angiography.

  Serologic tests: antiglomerular basement membrane antibodies, antineutrophil cytoplasmic antibodies, complement, antinuclear antibodies, cryoglobulins, serum protein electrophoresis, anti-streptolysin O or anti-DNase titers

- **Flowchart of serial blood pressures, weights, BUN, serum creatinine, major clinical events, interventions, and therapies**
- **Renal biopsy**

Differential diagnosis

- **Differentiation of Prerenal Acute Kidney Injury and Ischemic Acute Tubule Necrosis**
- **Differntiation of AKI form CRI**

Medical history

Size of the kidney

Anemia

Renal osteopathy

Serum calcium, phosphate

Urine Indices Used in the Differential Diagnosis of Prerenal and Ischemic Intrinsic Renal Azotemia

Diagnostic Index	Prerenal Azotemia	Ischemic Intrinsic Azotemia
Fractional excretion of Na^+ (%)	<1	>1
$\frac{U_{Na} \times P_{Cr}}{U_{Cr} \times P_{Na}}$		
Urinary Na^+ concentration (mEq/L)	<10	>20
Urinary creatinine/plasma creatinine ratio	>40	<20
Urinary urea nitrogen/plasma urea nitrogen ratio	>8	<3
Urine specific gravity	>1.018	<1.012
Urine osmolality (mOsm/kg H_2O)	>500	<250
Plasma BUN/creatinine ratio	>20	<10–15
Renal failure index, $U_{Na}/U_{cr}/P_{cr}$	<1	>1
Urine sediment	Hyaline casts	Muddy brown granular casts

COMPLICATIONS

Complications

Metabolic	Cardiovascular	Gastrointestinal	Neurologic	Hematologic	Infectious	Others
•Hyperkalemia •Metabolic Acidosis •Hyponatremia •Hypocalcemia •Hyperphosphatemia Hypermagnesemia •Hyperuricemia	•Pulmonary edema •Arrhthymias •Pericardiitis •Pericardial effusion •Pulmonary Embolism •Hypertension •Myocardial Infarction	•Nausea •Vomiting •Malnutition •GI hemorrhage	•Neuromuscular irritability •Asterixis •Seizures •Mental status changes	•Anemia •Bleeding	•Pneumonia •Septicemia •Urinary tract infection	•Hiccups •Elevated parathroid hormone •Low total triiodothyronine and throxine Normal free throxine

Managements

Goals

- Prevent death
- Ameliorate complications
- Preserve renal function

- **Prevention**
- **Specific Therapies**
- **Supportive Measures**

Preventions

- Fluids replacements *(hypovolemia)*
- Optimization of cardiovascular function and intravascular volume *(major surgery and trauma)*
- Hydration *(contranst induced nephropathy, using isotonic saline)*
- N-acetylcysteine (oral)
- Drugs should be used with caution *(Diuretics, NSAIDs (including COX-II inhibitors), ACE inhibitors, and other vasodilators)*

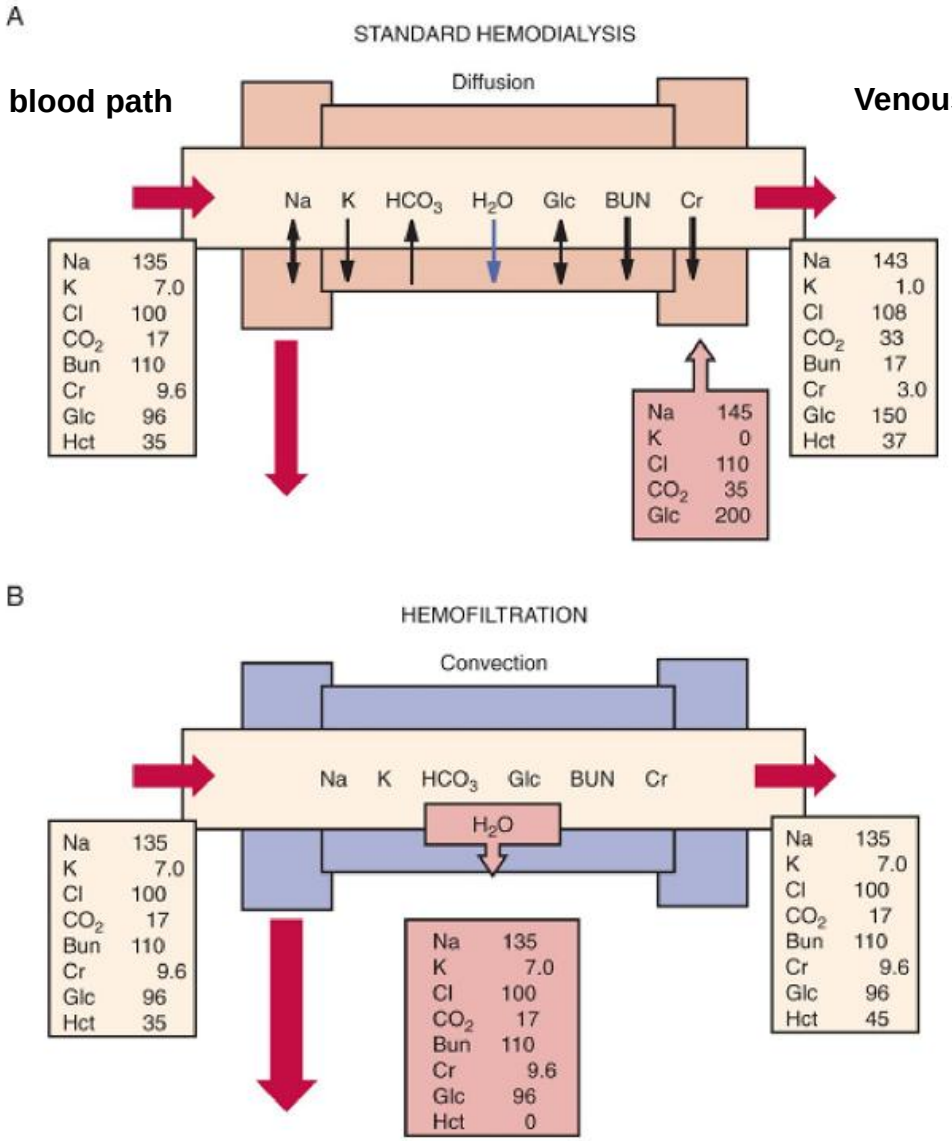
Supportive treatments

Complication	Treatment
Intravascular Volume Overload	Restriction of salt (<1 – 1.5 g/day) and water (<1 L/day)
	Consider diuretics (usually loops +/- thiazide)
	Ultrafiltration
Hyponatremia	Restriction of oral and intravenous free water
Hyperkalemia	Restriction of dietary potassium
	Discontinue K^+ supplements or K^+-sparing diuretics
	K^+-binding resin
	Loop diuretic
	Glucose (50 mls of 50%) + insulin (10 – 15 U regular) IV
	Sodium bicarbonate (50 – 100 meq IV)
	Calcium gluconate (10 mLs of 10% solution over 5 min)
	Dialysis/hemofiltation
Metabolic Acidosis	Restriction of dietary protein
	Sodium bicarbonate (if HCO_3^- <15 mEq/L)
	Dialysis/hemofiltation
Hyperphosphatemia	Restriction of dietary phosphate intake
	Phosphate binding agents (calcium carbonate, calcium acetate, sevalemer)
Hypocalcemia	Calcium carbonate (if symptomatic or sodium bicarbonate to be administered)
Hypermagnesemia	Discontinue magnesium containing antacids
Nutrition	Restriction of dietary protein (<0.8 g/kg/day up to 1.5 g/kg/day on CVVHD) 25 – 30 kcal/day
	Enteral route of nutrition preferred
Drug Dosage	Adjust all doses for GFR and renal replacement modality
Absolute Indications for RRT	Clinical evidence of uremia
	Intractable volume overload
	Hyperkalemia or severe acidosis resistant to conservative management

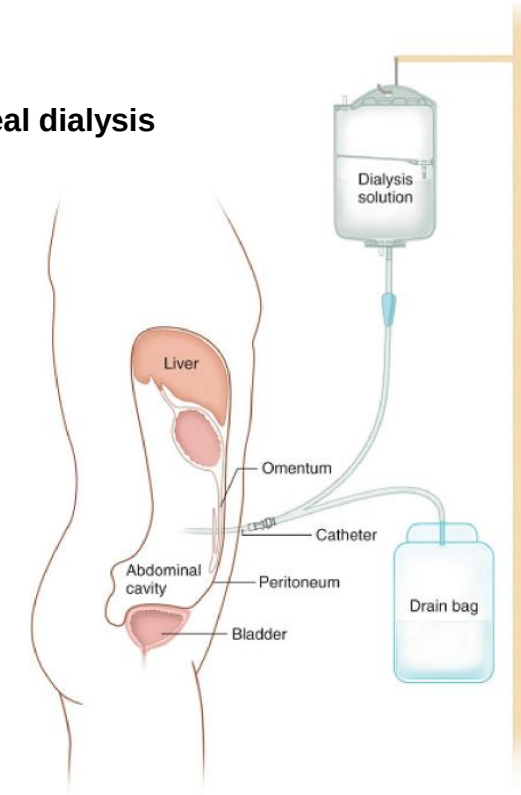
Classic Indications for RRT

- Non-obstructive oliguria (<200mL/12 hr) or anuria
- Azotemia (urea>30 mmol/L) or uremic organ involvement
- Hyperkalemia (K+ >6.5 mmol/L) or rapidly rising
- Severe acidemia (pH <7.1) from metabolic acidosis
- Progressive and/or uncontrolled dysnatremia
- Uncontrolled hyperthermia and/or hypothermia (>39.5 C)
- Clinically significant, diuretic-unresponsive organ edema
- Drug overdose with dialyzable toxin
- Coagulopathy requiring large amount of blood products

Modalities of Dialysis



Peritoneal dialysis

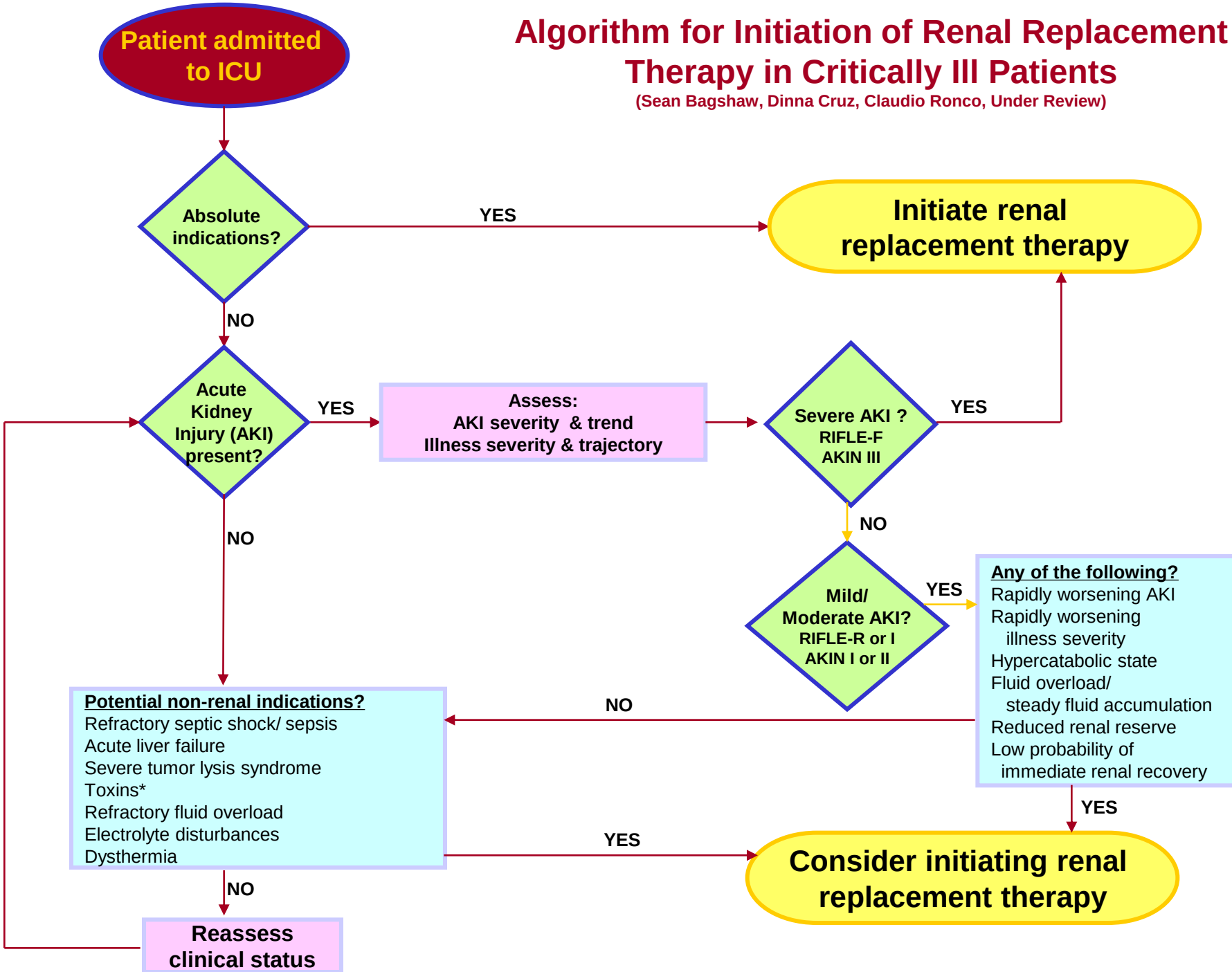


The Changing Paradiam of AKI

Renal Replacement Therapy		Renal Support Therapy
Life Threatening Indications	"Absolute"	✓Nutrition support ✓Volume removal in refractory CHF ✓Immuno-modulation in sepsis ✓Cancer chemotherapy ✓Attenuate ARDS-induced respiratory acidosis ✓Volume homeostasis in multi-organ dysfunction/failure
✓Solute control ✓Fluid balance ✓Acid-base regulation		

Algorithm for Initiation of Renal Replacement Therapy in Critically Ill Patients

(Sean Bagshaw, Dinna Cruz, Claudio Ronco, Under Review)



Case study

- A 55-year old male patient was admitted because of oliguria and anorexia for 3 days.
- A week ago, diarrhea and vomiting
- diagnosed as acute gastroenteritis and Amikacin was administered intravenously (IV) for 4 days at a daily dose of 0.4 g.
- daily urine output diminished to about 300ml in the last 3 days
- felt fatigue and anorexia

Case study

At Emergency Room

- Renal test: BUN 29.7mmol/L, Scr 1006μmol/L, UA 498μmol/L. Serum electrolytes: Na^+ 132mmol/L，K^+ 6.2mmol/L，Cl- 94mmol/L，$Ca2^+$ 2.0mmol/L，CO_2CP 12mmol/L
- EKG: sinus bradycardia with tall, peaked T-wave，HR 58 bpm.

- Past medical history was entirely negative
- No family history of renal disease
- Nothing abnormal in his last routine physical examination 6 months ago.

Case study

Physical Examination

- T 36.8℃, BP: 160/100mmHg, R:20/min，HR:58 bpm. Alert and oriented, no pallor, no jaundice, no palpable enlarged lymph nodes
- Periorbital edema
- Lungs (-) Heart (-).
- Abdominal findings: soft, nontender, no palpation of liver and spleen, shift dullness (-)
- Mild edema of the lower limbs, NS(-).

Case study

Clinical Thinking

- Oliguria for 3 days with systemic symptoms
- Renal test and serum electrolytes showed renal failure and hyperkalemia
- Acute episode
- Medication with nephrotoxic aminoglycosides

The first step for treating this patient was

- Maintain homeostasis
- Correct fluid-electrolyte disturbance
- Maintain acid–base balance

Case study

CLINICAL COURSE

- Urinalysis protein +, RBC 15~20/HP, WBC 5~7/HP.
- Complete blood count (CBC): RBC 3.56×10^{12}/L, Hb 98g/L, WBC 4.9×10^{9}/L, N68.2%, BPL 125×10^{9}/L.
- Liver function test: ALT 24 U/L, AST 34 U/L, A 40g/L, G 28g/L, TB 6.3μmol/L, SB 1.7μmol/L.
- Blood gas analysis: pH 7.31, $HCO3^{-}$ 16mmol/L, PaO_2 90mmHg, $PaCO_2$ 30mmHg, SBE^{-} 3.2mmol/L, AG 15mmol/L.

A slow IV push of 10ml of 10% calcium gluconate

IV push of 40 mg of furosemide

IV drip of 125ml of 5% $NaHCO_3$

Case study

CLINICAL COURSE

- Mild anemia : fluid retention and blood dilution .
- Blood gas analysis:decompensated metabolic acidosis
- Treatment of hyperkalemia

IV calcium gluconate :antagonizes the effects of hyperkalemia on the myocardial conduction system and on myocardial repolarization

IV push of 40 mg of furosemide: stimulate the renal excretion of potassium

IV drip of 125ml of 5% $NaHCO_3$: stabilize membrane potential, correct acidosis

Case study

CLINICAL COURSE

- Serum electrolytes were retested: Na^+ 132mmol/L, K^+ 5.8mmol/L, Cl^- 95mmol/L, $Ca2^+$ 2.1mmol/L, CO2CP 15mmol/L. On the next morning, serum potassium rose again to 6.5mmol/L
- Renal test: BUN 31.6mmol/L, Scr 1260μmol/L, UA 505μmol/L.
- Daily urine output : 100ml /day, didn't respond to a large dose of furosemide (200mg IV push) or 20% mannitol (250ml, IV drip
- chest tightness and short of breath
- PE: BP 130/100mmHg, R 22 per minute, HR 120 bpm. An apical third heart sound was heard. Pulmonary auscultation: coarse breathing sounds, bibasilar rales were present.

- Renal toxicity of aminoglycosides
- Hypovolemia

Conservative treatment was unsatisfactory

fluid retention caused acute left heart failure

Evidence of a hypercatabolic state

Case study

CLINICAL COURSE

- Emergent hemodialysis was performed immediately
- After four successive daily sessions, serum creatinine level decreased gradually and serum potassium was also kept within normal range. Daily urine output picked up, BP normalized, heart failure symptoms including peripheral edema and GI symptoms all disappeared.
- After another 2 weeks of conservative therapy, patient's renal function returned to normal.

With the help of supportive hemodialysis, renal functioncould be restored gradually with tubular regeneration and functional restructuring

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