AKI

Acute kidney injury

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Learning Objectives

* Introduction
* Definitions
* Diagnosis
* etiology
* classification
* Epidemiology
* clinical outcome
* Approach and management of AKI
* Risk factors
* preventive strategies

KIDNEY DISEASE
KDIGO
IMPROVING GLOBAL OUTCOMES

Introduction:

Acute kidney injury (AKI) has now replaced the term acute renal failure

Definition:

a sudden

sustained

and

usually reversible

decrease in the glomerular filtration rate (GFR)

occurring over a

period of hours to days

Epidemiology

AKI is common in hospitalised patients, especially in those who are critically ill

KDIGO:prevalence of ~2100 people per million population

≈ 5-10% in hospitalized patients

≈ 70% in critically ill patients

5-6% ICU patients require RRT

poor outcome:

higher risk of complications

longer stay in hospital

increased risk of needing long-term dialysis

high mortality

$$GFR_{Cockcroft} = \frac{(140 - age) \times mass\,(kg)\,[\times 0.85 \text{ if female}]}{72 \times \text{serum creatinine}\,(mg/dl)}$$

$$GFR_{Cockcroft} = \frac{(140 - age) \times mass\,(kg)\,[\times 1.23 \text{ if male}]\,[\times 1.04 \text{ if female}]}{\text{serum creatinine}\,(\mu mol/l)}$$

Healthy adults have a GFR of about 140mL/min/1.73 m2

* The normal serum creatinine range is

0.6–1.1 mg/dL in women

0.7–1.3 mg/dL in men

* Normal creatinine clearance is

88–128 mL/min in women

97–137 mL/min in men

$$\frac{\text{حجم ادرار 24 ساعته (ml/min) × کراتینین ادرار 24 ساعته (mg/dl)}}{\text{کراتینین سرم (mg/dl)}}$$

acute kidney injury is a syndrome characterized by:

- Sudden decline in GFR

(GFR over 90mls/min/1.73m^2 is normal)

- Retention of nitrogenous wastes
- Disturbance in extracellular fluid volume
- Disturbance in electrolyte
- Disturbance in acid base
- Disturbance in homeostasis

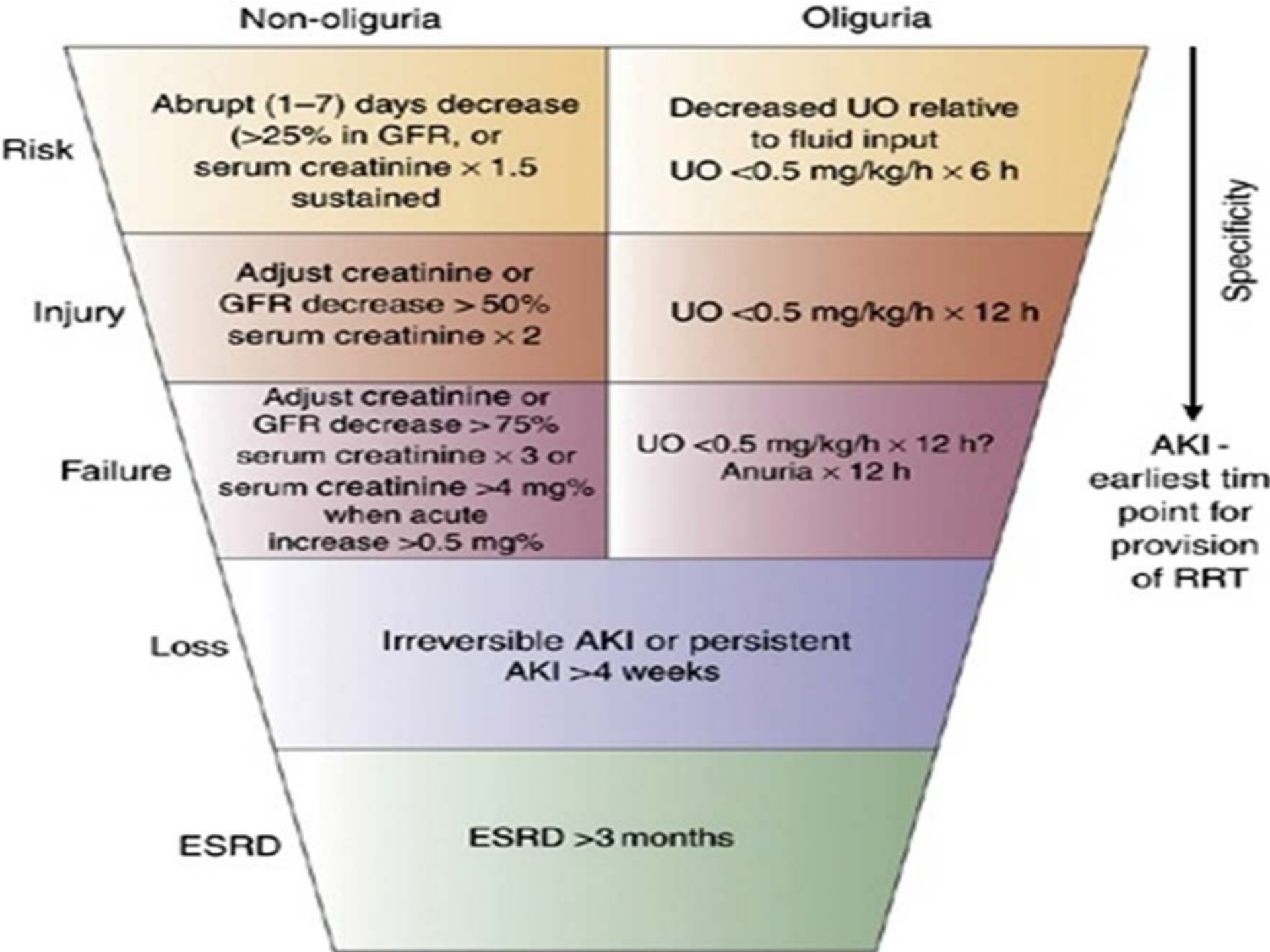
kDIGO proposed a consensus graded definition:
called the RIFLE criteria

The RIFLE criteria consists of three grade
(Risk, Injury, and Failure)

- Based upon either the magnitude of elevation in serum creatinine or urine output
- Two outcome measures
  (Loss and End-stage renal disease)

Rifle Criteria for stratifying AKI

- **R** isk
- **I** njury
- **F** ailure
- **L** oss of function
- **E** nd-Stage Renal disease


Non-oliguria
Oliguria
Risk
Abrupt (1–7) days decrease (>25% in GFR, or serum creatinine × 1.5 sustained
Decreased UO relative to fluid input UO <0.5 mg/kg/h × 6 h
Injury
Adjust creatinine or GFR decrease > 50% serum creatinine × 2
UO <0.5 mg/kg/h × 12 h
Failure
Adjust creatinine or GFR decrease > 75% serum creatinine × 3 or serum creatinine >4 mg% when acute increase >0.5 mg%
UO <0.5 mg/kg/h × 12 h? Anuria × 12 h
Loss
Irreversible AKI or persistent AKI >4 weeks
ESRD
ESRD >3 months
Specificity
AKI - earliest tim point for provision of RRT

Clip slide

Causes

Pre-Renal

- Cardiovascular

Renal

- Glomerulus
- Tubules
- Interstitium

Obstructive

- Ureters
- Bladder
- Urethra

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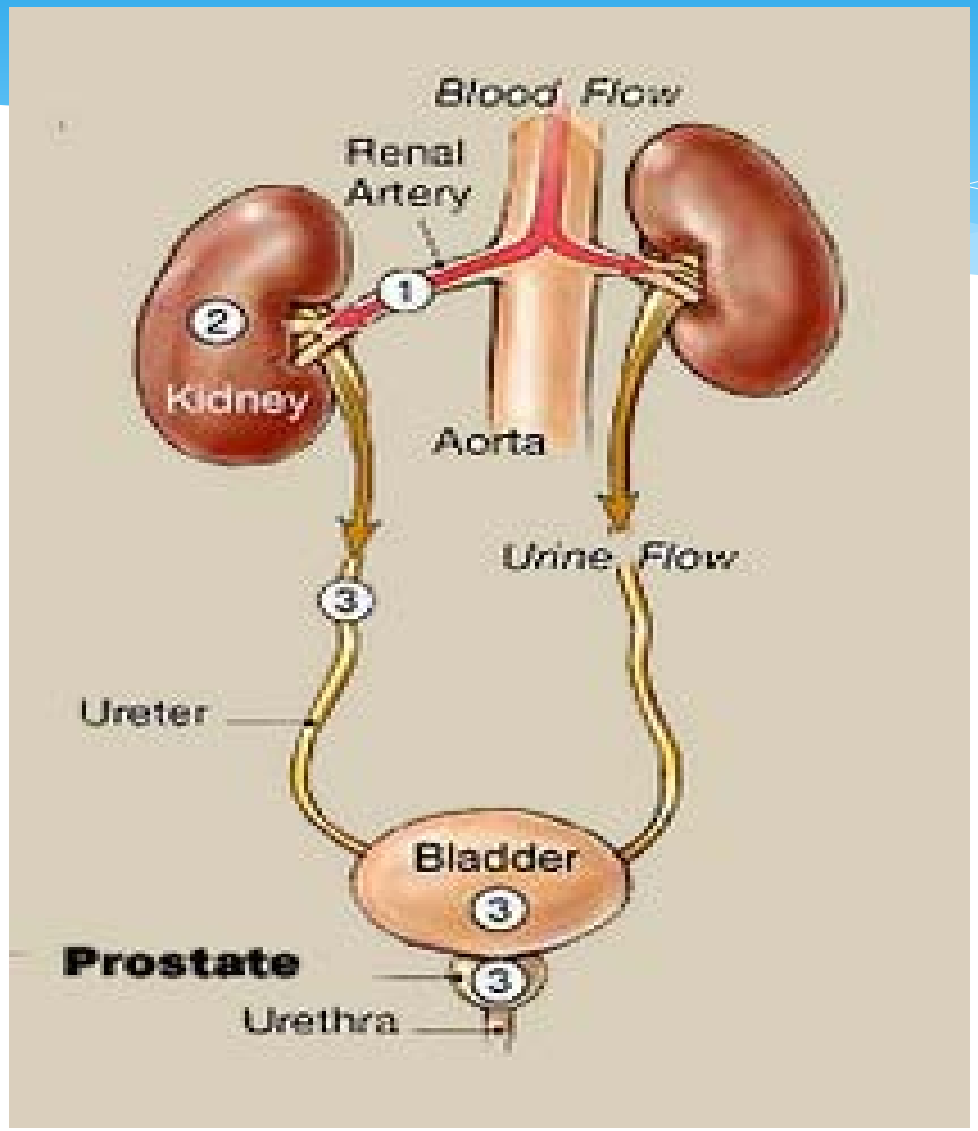
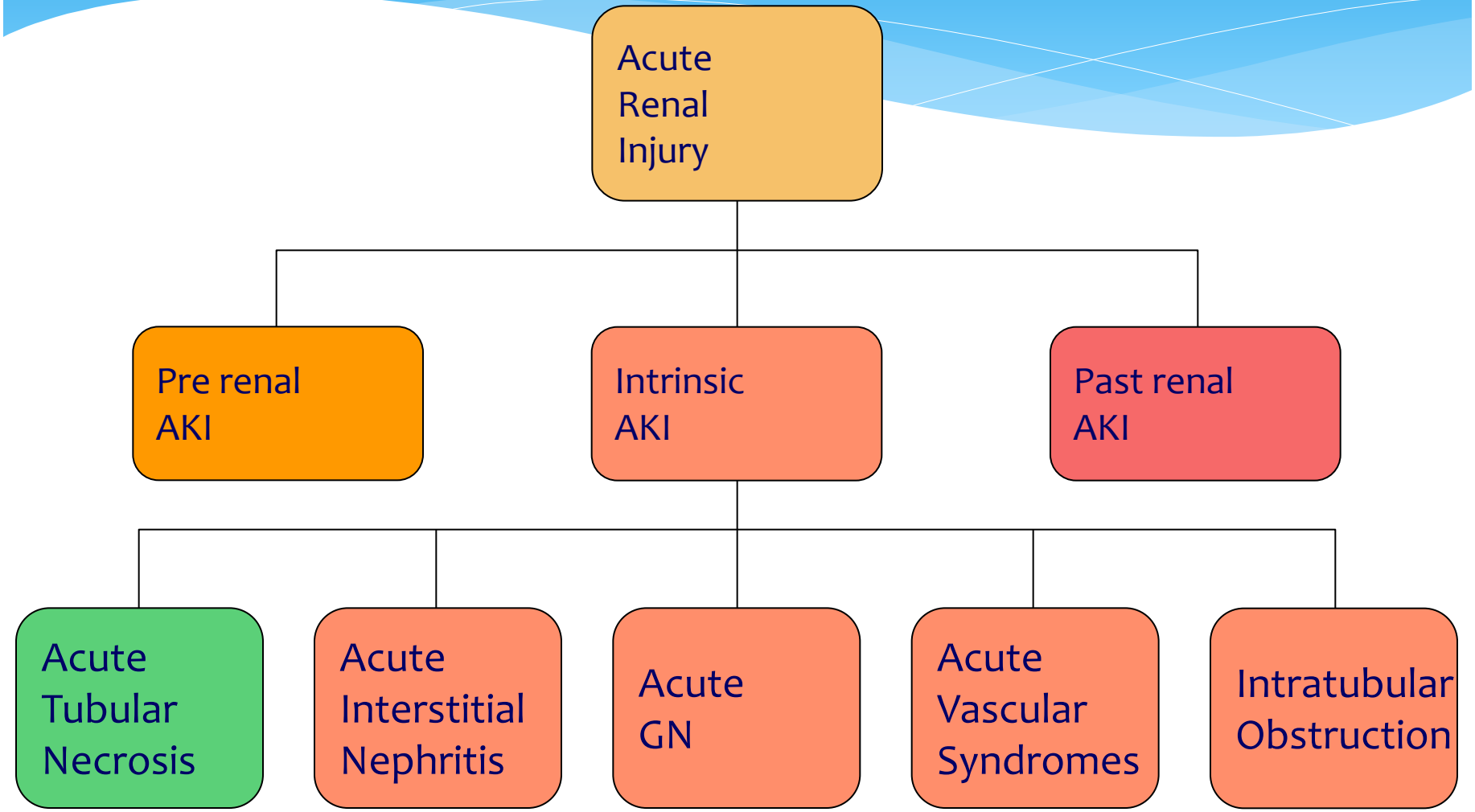
Etiology

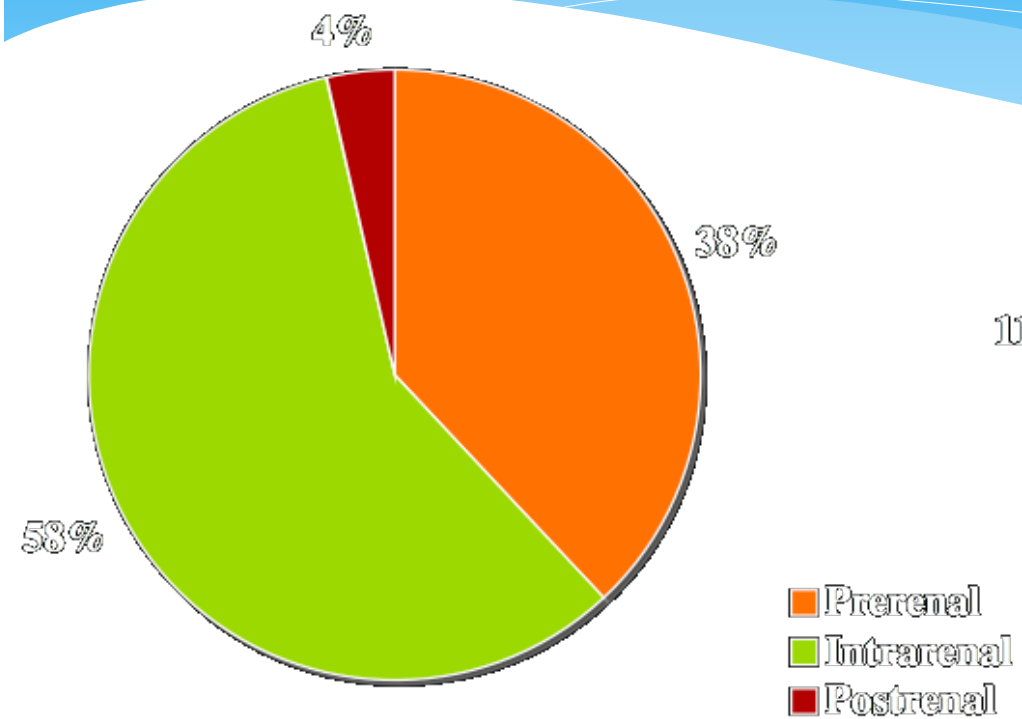


http://www.medicalassessmentonline.net/terms.php?R=3

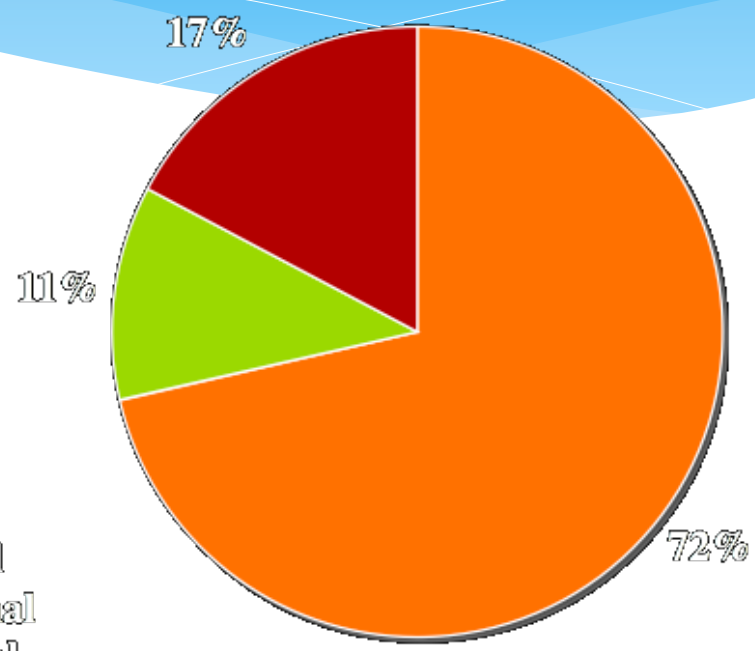
Classification of the Etiologies of AKI



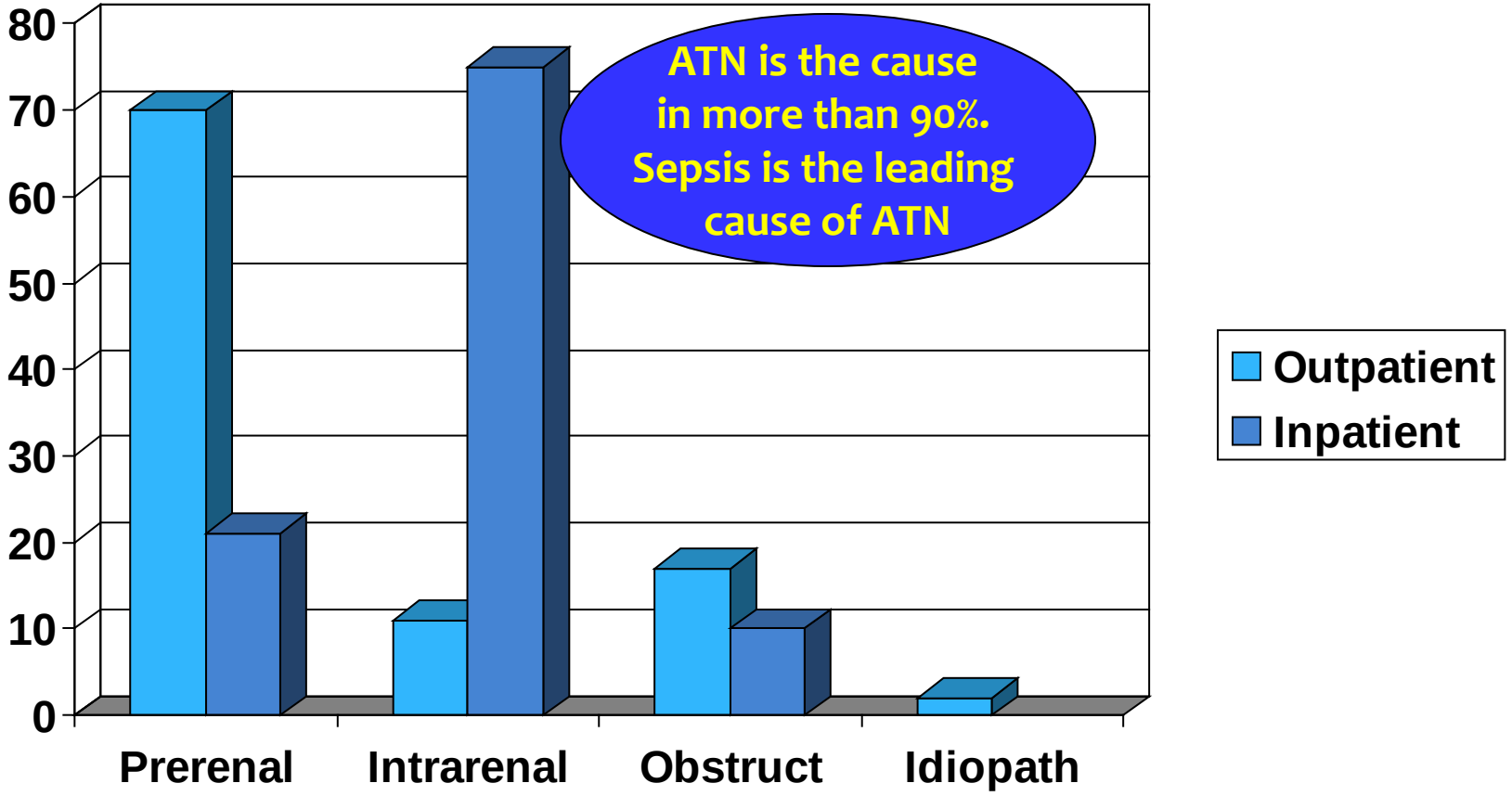
Inpatient/Hospital acquired



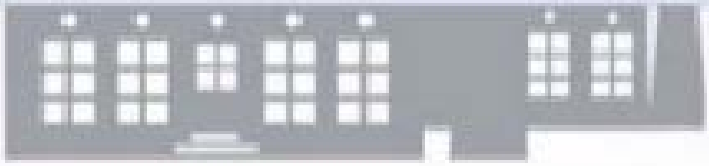
Outpatient acquired



Etiology of AKI



Causes



Prerenal

Hypoperfusion

- Hypovolemia
- Burns
- Cardiogenic shock
- Thromboembolism

Nephrotoxic Drugs

- ACEIs and ARBs
- Diuretics
- Beta Blockers
- Chemotherapy

Clip slide

B. Intrinsic Renal AKI:

Renal

- Glomerulonephritis
- ATN
- AIN
- Tumor Lysis Syndrome

Nephrotoxic Drugs

- NSAIDs and Aspirin
- Antibiotics
  - Aminoglycosides
  - Sulfonamides
  - Vancomycin
  - Quinolones
  - Rifampin
  - Tetracyclines
- Chemotherapy
- Heavy metals
- Antihyperlipidemics
  - Statins and Gemfibrozil
- Antifungal and antiviral

Acute Tubular Necrosis (ATN)

- Sepsis (48%)
- Ischemia (32%)
  - prolonged prerenal azotemia
  - Hypotension
  - hypovolemic shock
  - cardiopulmonary arrest
  - cardiopulmonary bypass
- Direct toxic Injury (20%)
  - Exogenous
    - Radiocontrast
    - Aminoglycosides
    - Vancomycin
    - Amphotericin B
    - Cisplatin
    - Acyclovir
    - Calcineurin inhibitors
    - HIV med (tenofovir)
  - Endogenous (pigment nephropathy)
    - Rhabdomyolysis
    - Hemolysis

C. Post renal AKI (OBSTRUCTION)

Postrenal

- BPH
- Stones
- Malignancy
- Obstructed catheter

- Signs and Symptoms
- Vomiting and/or diarrhea, which may lead to dehydration
- Nausea
- Weight loss
- Nocturnal urination
- pale urine
- Less frequent urination, or in smaller amounts than usual, with dark colored urine
- Hematuria
- Pressure, or difficulty urinating
- Bone damage
- broken bones
- Muscle cramps (caused by low levels of calcium which can cause hypocalcaemia)
- Abnormal heart rhythms
- Muscle paralysis
- Swelling of the feet, face and/or hands
- Shortness of breath due to extra fluid on the lungs

- Pain in the back
- Feeling tired and/or weak
- Memory problems
- Difficulty concentrating
- Dizziness
- Low blood pressure
- Anorexia
- Pruritus
- Seizures (if blood urea nitrogen level is very high)
- Decrease osmolality
- Pericarditis.
- Pericardial effusion.
- Pleural effusion
- Decrease calcium
- Defect in platelet function

* Diagnostic tests
  - History & Physical examination
  - BUN, creatinine, sodium, potassium, pH , Hgb and Hct
  - Urine studies
  - US of kidneys
  - KUB
  - ABD and renal CT/MRI
  - Retrograde pylogram

Medical treatment

- Management of Uremic Complications
- D/C or change cause
- Adjust medication dosages
- May need dialysis to jump start renal function

Complications

- Uremia
- Hypervolemia and Hypovolemia
- Hyponatremia
- Hyperkalemia
- Hyperphosphatemia
- Hypocalcemia
- Bleeding
- Infections
- Cardiac Complications
- Malnutrition

Management of Uremic Complications

In patients with advanced AKI, renal support should be started before the onset of life-threatening uremic complications

many patients develop AKI in the community but may not present to the Emergency Department until they develop uremic symptoms:

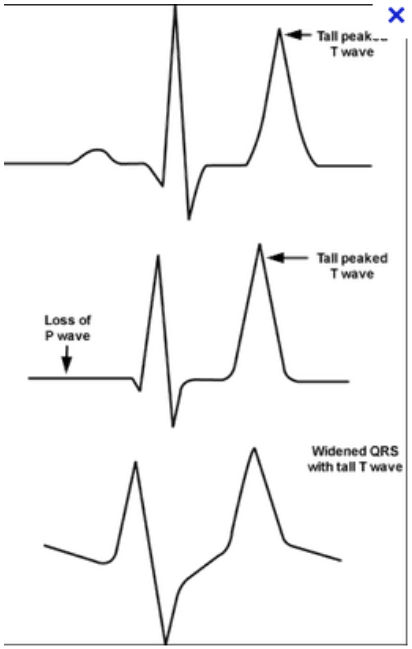
Patients with AKI and progressive:
hyperkalemia (mmol/L)
metabolic acidosis
progressive pulmonary edema
uremic pericarditis

need to be referred to the renal or critical care team urgently
Whilst waiting for RRT, medical treatment may be considered

Hyperkalaemia

- Potassium range is 3.5 – 5.5mmol/L
- Rise in serum K+ >5mmol/l
- Signs/symptoms: muscle weakness

- ECG changes:
  - Flattened P waves
  - Broad QRS complex
  - Slurring of ST segment
  - Tall tented T waves



http://www.aafp.org/afp/2006/0115/p283.html

Medical Treatment of Hyperkalaemia

Severe hyperkalemia (serum potassium >6.5 mmol/litre) ⇨ cardiac arrhythmias and should be treated immediately

Initial management :calcium gluconate or calcium chloride

beta-agonists(albuterol) and intravenous insulin and glucose were all effective at reducing potassium levels, in particular when administered in combination

use of intravenous $NaHCO_3$ to treat hyperkalemia was considered to be equivocal
Potassium-absorbing resins have no role in the emergency management

There is also no clear evidence that means to increase urine output and potassium excretion (e.g., by fluid resuscitation or by loop diuretics) are sufficient to correct hyperkalemia.
If medical therapy fails to correct hyperkalemia ⇨ RRT

Fluid Therapy

* The most commonly used fluids are crystalloids and colloids
* Although timely fluid resuscitation is important to prevent AKI in conditions associated with volume depletion
* there is increasing evidence that excessive fluid administration can be harmful and lead to dysfunction of other organs and adverse outcomes
* avoid both hypo- and hyper volumia
* patients need to be assessed regularly

Diuretics

* no role in preventing or treating established AKI.
* did not improve renal function
* did not change mortality
* no role in speeding up recovery of renal function
* in patients with progressive fluid accumulation, diuretics can minimize fluid overload and may make patient management easier, especially if RRT is not immediately available.

Medical Management of Metabolic Acidosis

* *Adverse* effects of acute metabolic acidosis include:
* decreased cardiac output
* hypotension
* altered oxygen delivery
* arrhythmias
* impairment of the immune response
* administration of $NaHCO_3$ should be considered to maintain blood pH at ~7.2
* If $NaHCO_3$ is administered, it should be given as an isotonic preparation (to prevent hyper osmolality) and as a slow infusion

Medical Management of Fluid Overload/Pulmonary edema

* supplementary oxygen
* noninvasive ventilation
* intubation and ventilation
* intravenous infusion of nitrate and diuretics(diuretics may delay the start of RRT)

* fluid removal by RRT

Management of Uremic Pericarditis

* The prevalence of uremic pericarditis in patients with acute kidney disease is 6–10%.
* Affected patients are often asymptomatic but may be complaining of chest pain
* They often have a small to moderate pericardial effusion
* The only therapy is RRT and pericardia centesis
* the presence of a pericardial rub on clinical examination and classic ECG changes consistent with pericarditis are indications for urgent RRT and an urgent echocardiogram

Nursing interventions

- Monitor I/O, including all body fluids
- Monitor lab results
- Watch hyperkalemia symptoms: malaise, anorexia, paresthesia, or muscle weakness, EKG changes
- watch for hyperglycemia or hypoglycemia if receiving TPN or insulin infusions
- Maintain nutrition
- Mouth care
- Daily weights
- Assess for signs of heart failure
- GCS
- Skin integrity problems

Nursing Management

In

Acute Kidney Injury

9/6/2017

Nursing diagnoses

- **excess fluid volume** related to decrease urine output and retention of sodium and water
- **Imbalanced nutrition ,less than body requirements** related to altered metabolic state and dietary restrictions , anorexia, nausea ,vomiting,…
- **Risk for infection** related to invasive lines ,uremic toxins ,altered immune responses secondary to kidney failure
- **Activity intolerance** related to fatigue and retention of waste products , anemia
- **Anxiety** related to disease processes , therapeutic interventions , uncertainly of prognosis
- **Knowledge deficit about self care and control of disease process**

Excess Fluid volumes

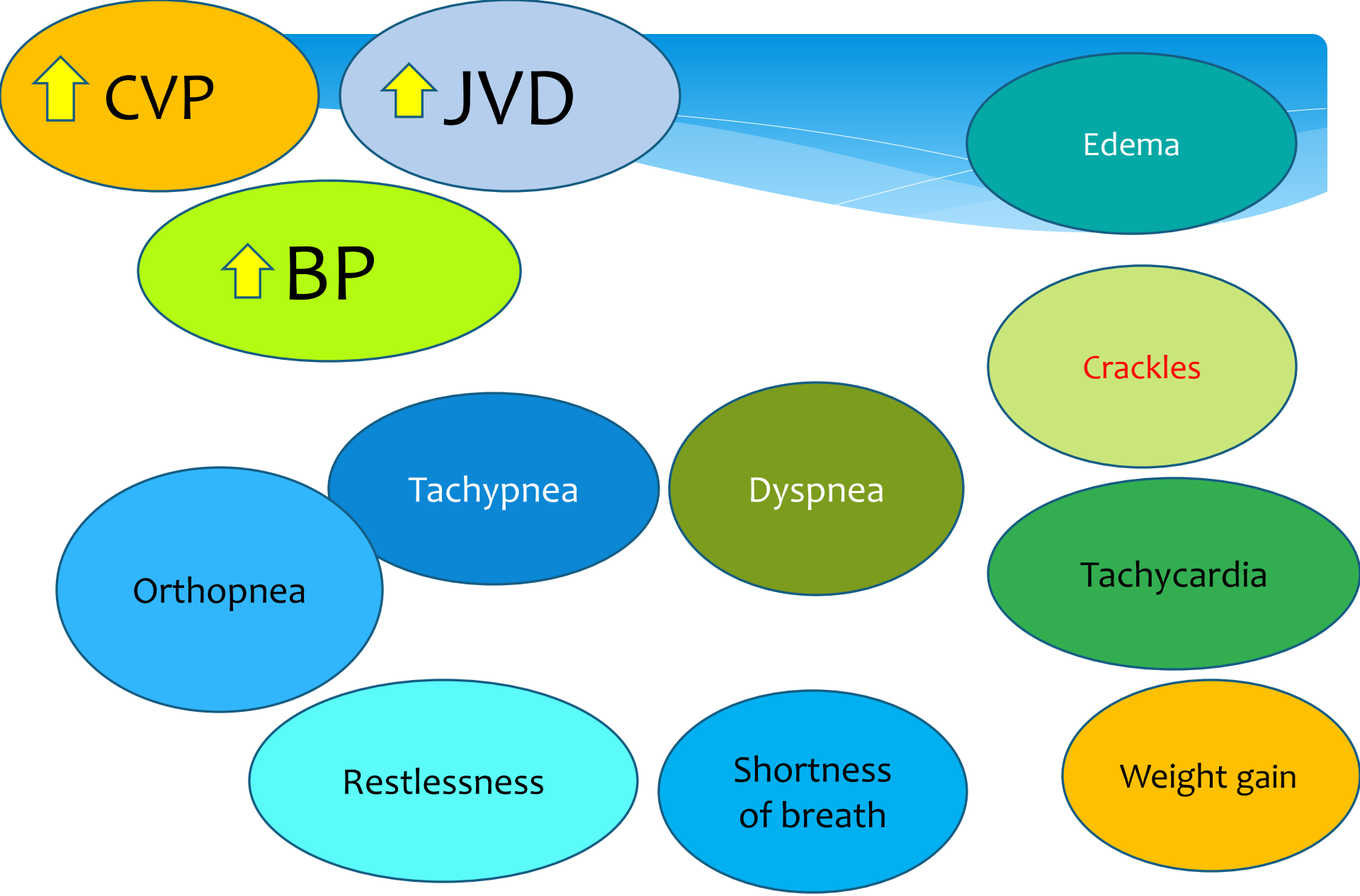
Water retention

Decrease urinary output

Decrease perfusion in kidney

Decrease blood flow to kidneys

Renal injury


CVP
JVD
Edema
BP
Crackles
Tachypnea
Dyspnea
Orthopnea
Tachycardia
Restlessness
Shortness of breath
Weight gain

Intervention

1-Weigh daily at same time of day, on same scale with same clothing

2- Record accurate intake and output (I&O)

3- Assess skin, face , dependent areas for edema

4-Assess for signs of circulatory overload(Monitor heart rate ,BP,CVP)

5-Monitor respiratory rate

6-Auscultate lung sounds and heart sounds for sign of fluid overloud

7- Restrict fluids

8-Administer medicatios as prescribed in least amount of fluid possible

Expected outcome

* patient will display appropriate urinary output with specific gravity/laboratory studies near normal
* stable weight, vital signs within patient's normal range
* And absence of edema.

Risk for decreased cardiac output

* Dysrhythmias caused by electrolyte imbalance from AkI:

Hyperkalemia

Hyponatremia

Hypocalcemia

Volume overload leading to heart failure

Pericarditis or pericardial effusion

Dehydration resulting from the diuretic therapy

Intervention

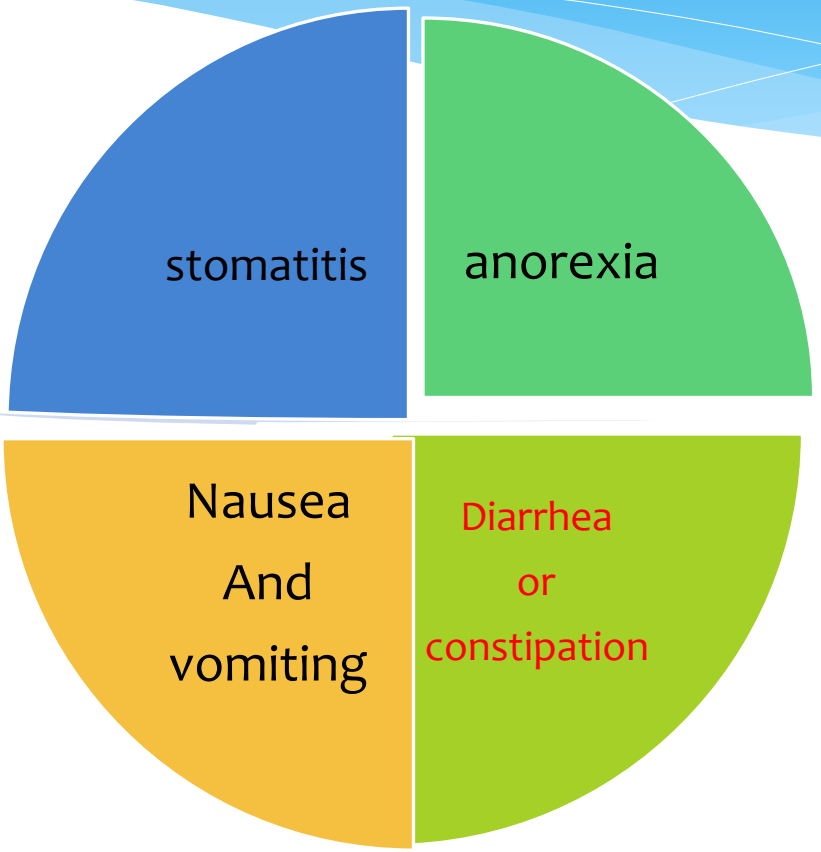
* Assess for signs of decreased CO(BP,CVP,Heart rate,peripheral pulses , JVp , Decreased urinary out put , abnormal heart sounds ,dysrhythmias , anxiety or restlessness
* Assessing for electrolyte disturbances
* Monitor cardiac rhythm
* Monitor for signs and symptoms of metabolic acidosis

Expected outcome

* Patient maintains adequate cardiac output

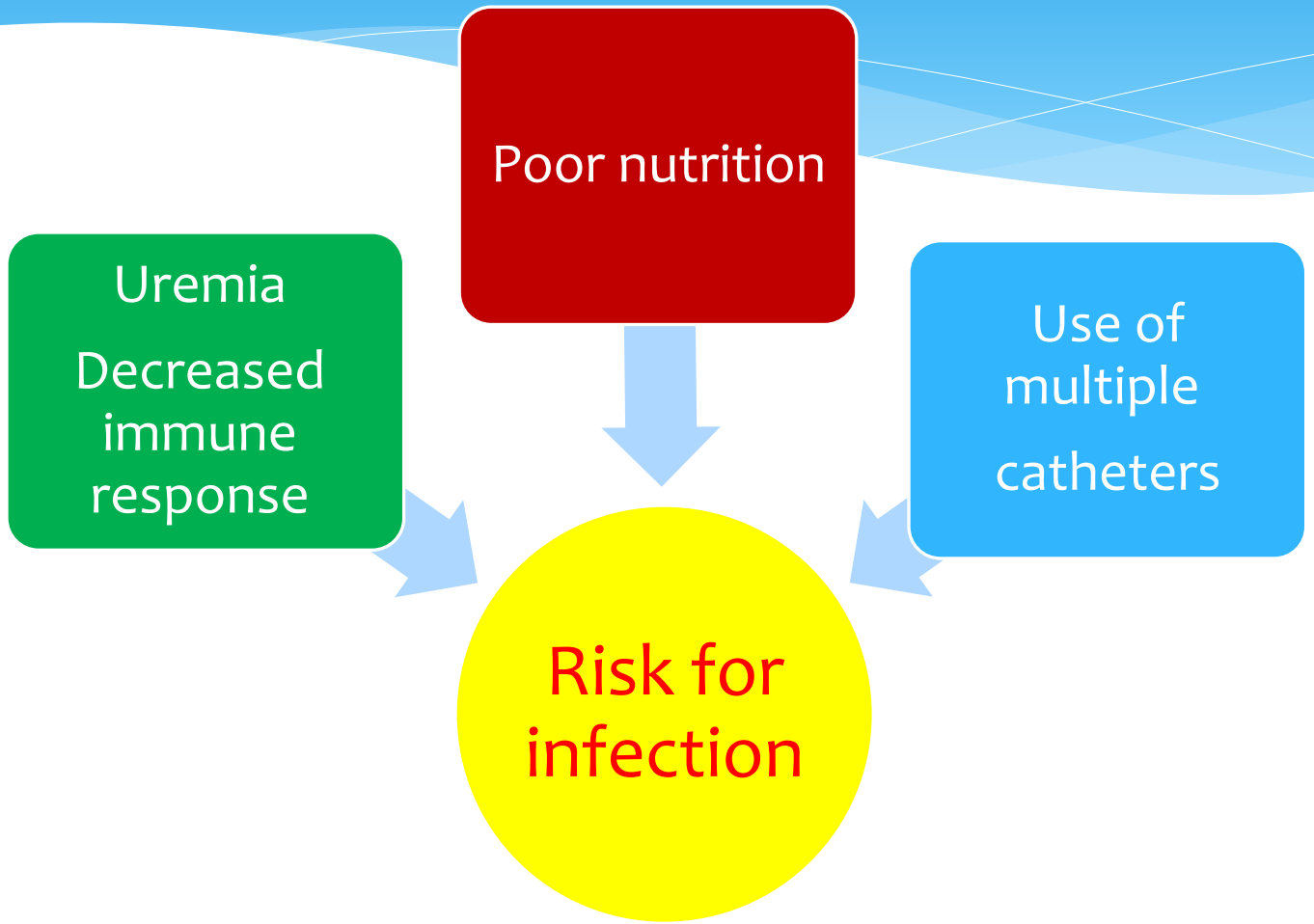
(stable cardiac rhythm , BP,….)

Imbalanced nutrition:less than body requirements



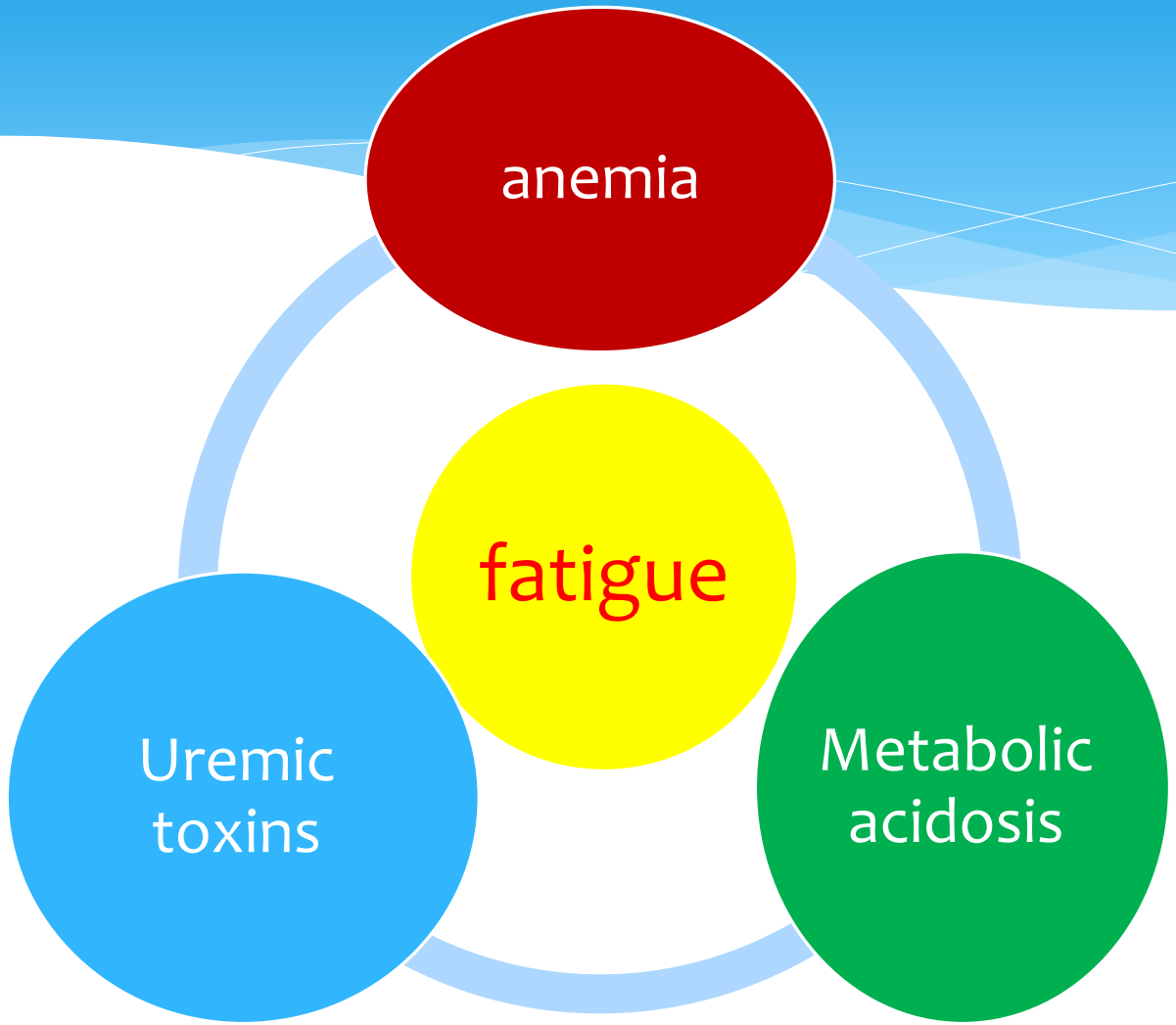
Intervention

- Administer small ,frequent feedings as tolerated
- Make meals look appetizing
- Provide frequent oral hygiene
- Offer ice chips or hard candy if not contraindicated
- Consult a dietitian
- Adjust potassium restriction as indicated
- Provide renal replacement therapy if indicated


Poor nutrition
Uremia
Decreased immune response
Use of multiple catheters
Risk for infection

Intervention

- Assess for potential sites of infection : urinary ,pulmonary, wound or iv line.
- Monitor temperature
- Monitor white blood cell count
- Note signs of localized or systemic infection
- Provide meticulous skin care
- Use aseptic technique
- Avoid use of indewelling catheters or IV lines
- Protect the patient from exposure to other infection people


anemia
fatigue
Uremic toxins
Metabolic acidosis

Intervention

* Observe and document signs of fatigue ,pallor,weekness
* Administer oxygen as prescribed

کاهش سرعت متابولیسم:

پرستار باید به کاهش سرعت متابولیسم در طی پنج مرحله بیماری توجه داشته باشد، زیرا کاهش متابولیسم منجربه کاهش سوخت وساز و در نتیجه کاهش پتاسیم آزاد شده و کاهش تجمع محصولات زاید نهایی (اوره و کراتینین) می شود.

ممکن است برای کاهش فعالیت و کاهش میزان سوخت و ساز در طی مرحله حاد ، استراحت در بستر لازم باشد.تب و عفونت، هر دو میزان متابولیسم و کاتابولیسم را افزایش می دهند و باید فورا درمان شوند.

فعالیت و استراحت :

بیمار برای کاهش فعالیت متابولیک نیاز به استراحت دارد و برای انجام فعالیت های روزانه زندگی نیازمند کمک می باشد . دوره های استراحت برای حفظ انرژی بیمار لازم است.

پیشگیری از ایجاد آسیب

:

آگاهی نسبت به زمان ، مکان و شخص مورد بررسی قرار گیرد

در زمان استراحت در بستر نرده های کنار تخت بالا نگهداشته شوند

در صورتی که بیمار قادر به راه رفتن می باشد مهارت های حرکتی بررسی شده و حرکت بیمار زیر نظر گرفته شود

Deficient knowledge

- Assess knowledge and understanding of AKI
- Encourage the patient and family to ask questions
- Explain all testes and procedures before they occur
- Explain the purpose of fluid restrictions
- Discuss the need for a reduced-protein diet
- Discuss the need for follow –up visits after discharge

inspiringshot

preventive strategies

- Recognize people at risk of AKI
- Maintain renal perfusion
- Avoid nephro toxins

At Risk of AKI

* Aged 75 or over
* Cardiac Disease
* Liver Disease
* Diabetes
* Chronic Kidney Disease
* Cancer
* Acute conditions such as Sepsis
* Patients susceptible to dehydration
* Exposure to nephrotoxins(Aminoglycosides, amphotericin, immunosuppressive agents, chemo, NSAIDs, intravenous contrast media)
* Past cardiac or vascular Surgery pts or ICU pts with multi organ failure

داروهای نفروتوکسیک

* آمینوکلیکوزیدها،جنتامایسین،توبرامایسین،پلی میکسن B،آمفوتریپسین B،وانکومایسین،آمیکاسین،سیکلوسپورین
* هر دارویی که موجب کاهش جریان خون کلیوی شود مانند مصرف طولانی مدت مسکن ها بویژه NSAID
* اندازه گیریBUNو کراتینین سرم،24 ساعت بعد از شروع دارو و حداقل 2 بار در هفته در طول درمان الزامی است

مواد حاجب

* بیماران در معرض خطر
* کراتینین سرم <2 mg/dl
* دادن مایعات کافی قبل و بعد از رادیوگرافی با ماده حاجب
* دادن استیل سیستئین روز قبل از آزمایش(یک آنتی اکسیدان که رادیکال آزاد را تصفیه می کند)

Indications for Dialysis

A – Acidosis

E – Electrolyte disturb , usually hyperkalemia

I – Intoxications (lithium, ethylene glycol, etc)

O – Overload

U – Uremia (symptoms, signs)

BUN=100 mg/dl

Pulmonary edema

* The kidneys don't usually complain.
* The kidneys can lose up to 90% of their function before you may even begin to notice
* The kidneys are clever organs but need a good blood supply to work effectively
* Consider the kidneys in your daily visits
* Ask if your patient has passed urine?
* Ask if they are well hydrated
* Consider if your patient has risk factors for AKI and whether further investigations such as monitoring of creatinine levels are required
* Consider if your patient has an acute insult that may warrant temporary cessation of medications
* Consider further review by GP

یاد بگیریم دوست بداریم تا دوستمان بدارند

THANK YOU!