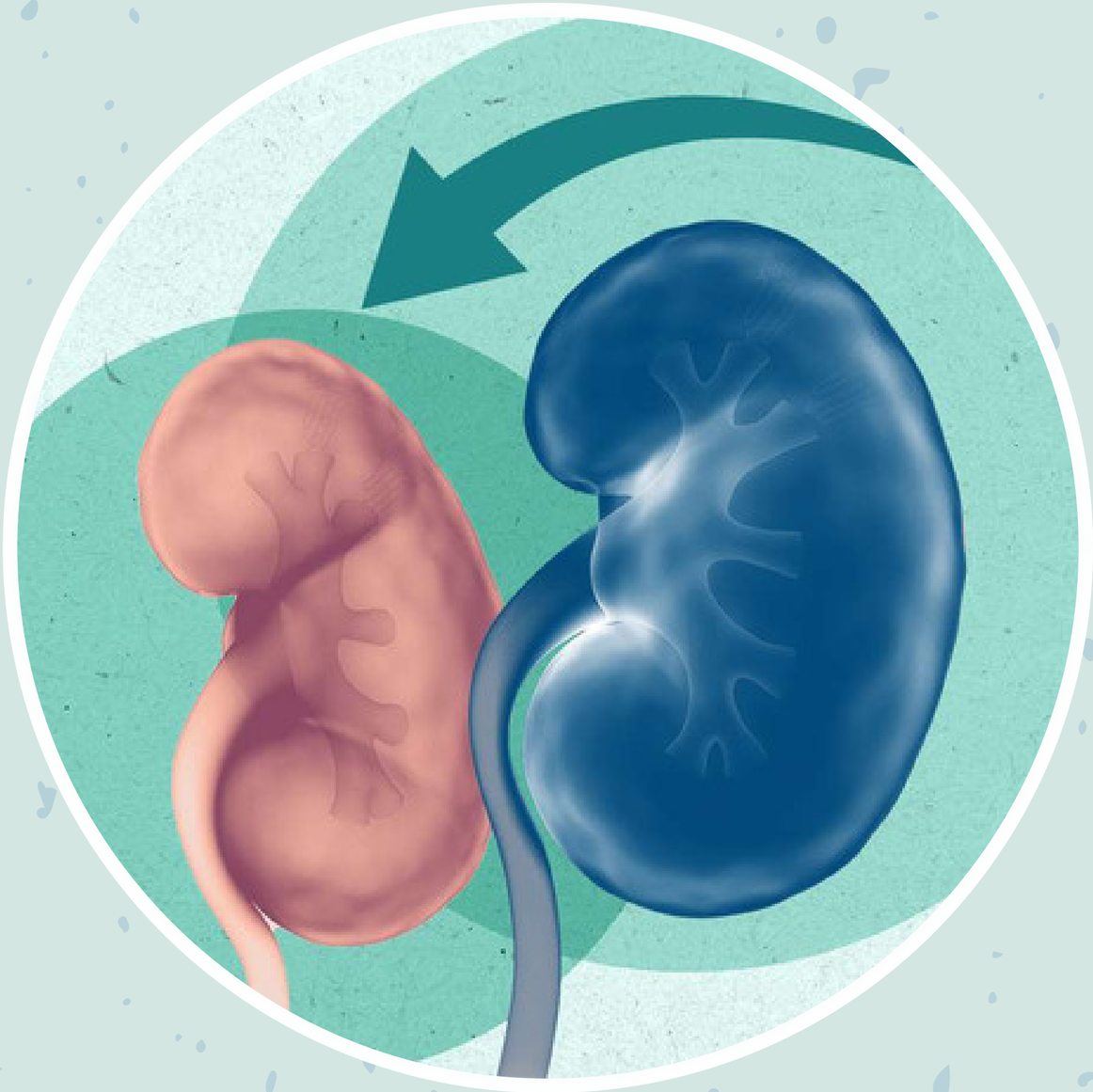
# ACUTE KIDNEY INJURY



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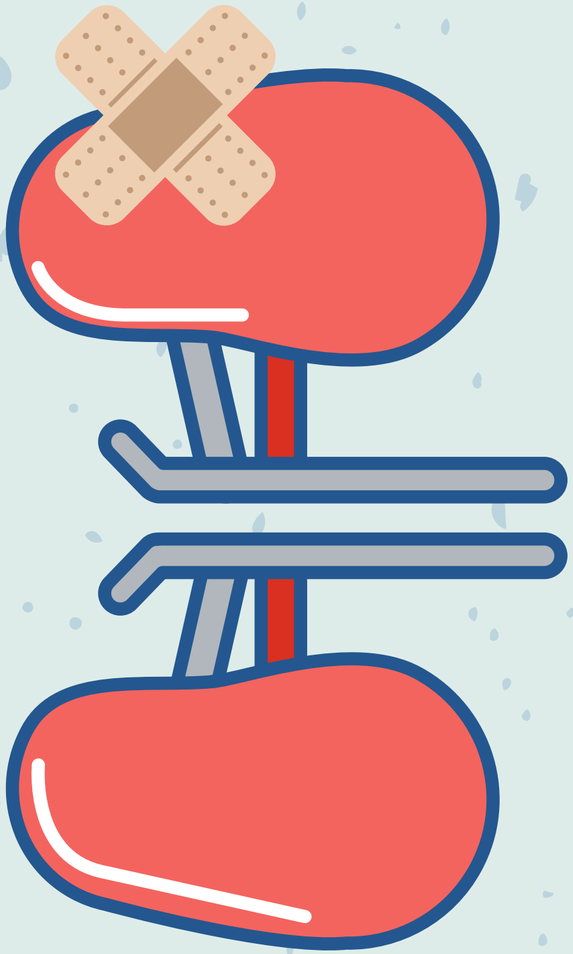

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# DEFINITION

AKI is defined as an acute decrease in kidney function or GFR over hours, days, or even weeks and is associated with an accumulation of waste products and volume.

According to KDIGO, AKI is the presence of any of the following:

1. Increase in serum creatinine by 0.3 mg/dL or more within 48 hours.
2. Increase in serum creatinine to 1.5 times or more baseline within the prior seven days.
3. Urine volume less than 0.5 mL/kg/h for at least 6 hours.

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# PREVALENCE WORLDWIDE AND IN EGYPT:

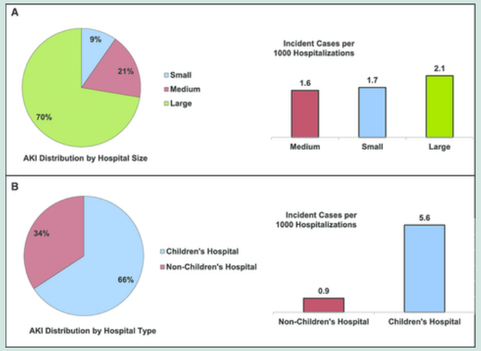
## 1. WORLDWIDE:

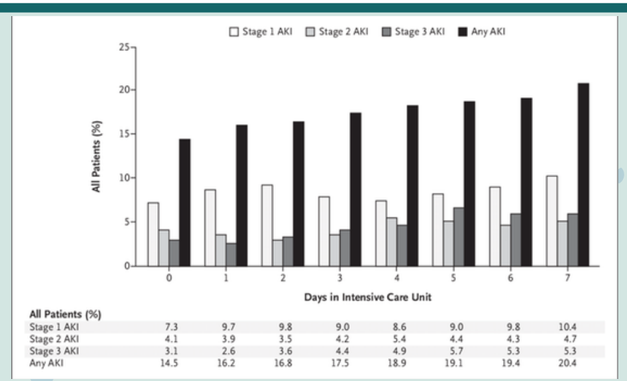
Using the KDIGO definition, 1 in 5 adults and 1 in 3 children worldwide experience AKI during a hospital episode of care.

## 2. IN EGYPT

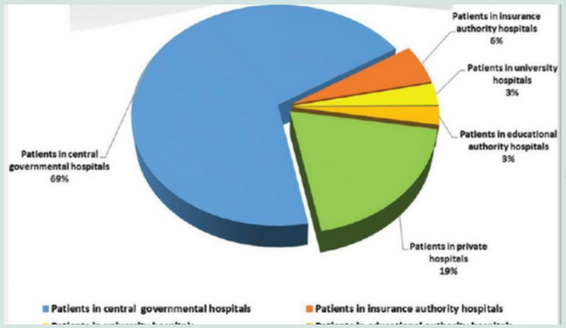
Epidemiology of acute kidney injury (AKI) in developing countries is under-studied. the risk and prognosis of AKI in patients admitted to intensive care units (ICUs) in Egypt has been assessed. ICUs in Alexandria Teaching Hospitals over six months was observed. KDIGO criteria for AKI had been used. participants until the earliest of ICU discharge, death, day 30 from entry or study end had been observed.

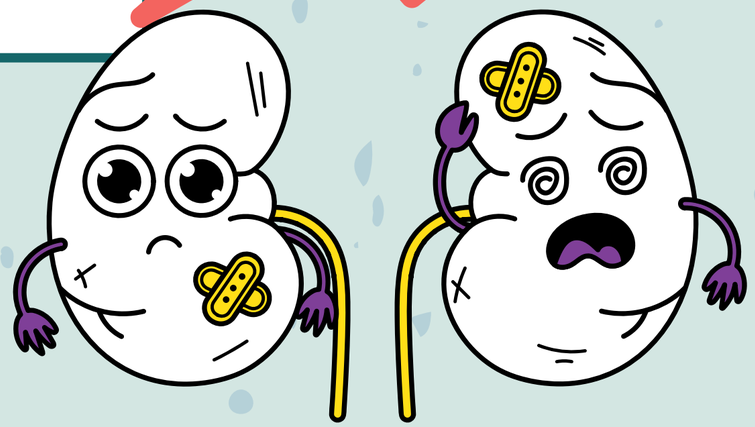
Of the 532 participants (median age 45 (Interquartile range [IQR]: 30–62) years, 41.7% male, 23.7% diabetics), 39.6% had AKI at ICU admission and 37.4% developed AKI after 24 hours of ICU admission. The risk of AKI is high in critically ill people and predicts poor outcomes. Further studies are needed to estimate the burden of AKI among patients before ICU admission.




| All Patients (%) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Stage 1 AKI | 7.3 | 9.7 | 9.8 | 9.0 | 8.6 | 9.0 | 9.8 | 10.4 |
| Stage 2 AKI | 4.1 | 3.9 | 3.5 | 4.2 | 5.4 | 4.4 | 4.3 | 4.7 |
| Stage 3 AKI | 3.1 | 2.6 | 3.6 | 4.4 | 4.9 | 5.7 | 5.3 | 5.3 |
| Any AKI | 14.5 | 16.2 | 16.8 | 17.5 | 18.9 | 19.1 | 19.4 | 20.4 |



# CLASSIFICATION

## 1. PRERENAL AKI

a. Initially, the kidney is undamaged.
b. Characterized by hypoperfusion to the kidney:
➢ Systemic hypoperfusion: Hemorrhage, volume depletion.
➢ Isolated kidney hypoperfusion: Renal artery stenosis.

## 2. FUNCTIONAL AKI

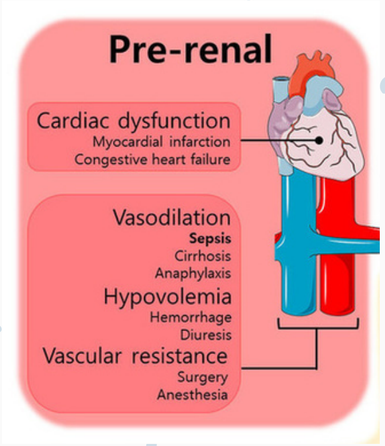
a. Kidney is undamaged; often classified as prerenal azotemia.
b. Caused by reduced glomerular hydrostatic pressure; often without hypotension

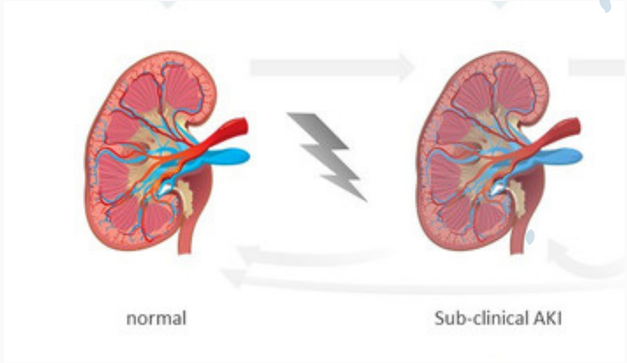
## 3. INTRINSIC AKI

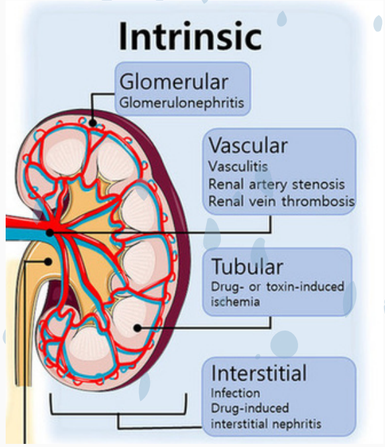
Kidney is damaged and damage can be linked to the structure involved: small blood vessels, glomeruli and renal tubules

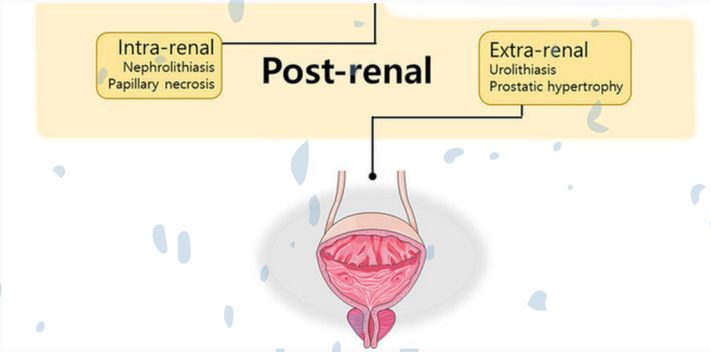
## 4. POSTRENAL AKI

Kidney is initially undamaged. Bladder outlet obstruction is the most common cause of postrenal AKI.



# WHAT ARE THE RISK FACTORS?

Preexisting CKD.

Volume depletion: Vomiting, diarrhea, poor fluid intake, fever, diuretic use, intravascular or effective volume depletion (e.g., CHF, liver disease with ascites)

Obstruction of the urinary tract

Use of nephrotoxic agents or medications
✧ Intravenous radiographic contrast.
✧ Aminoglycosides and amphotericin.
✧ Nonsteroidal anti-inflammatory drugs (NSAIDs) and cyclooxygenase-2 (COX-2) inhibitors.
✧ Cyclosporine and tacrolimus

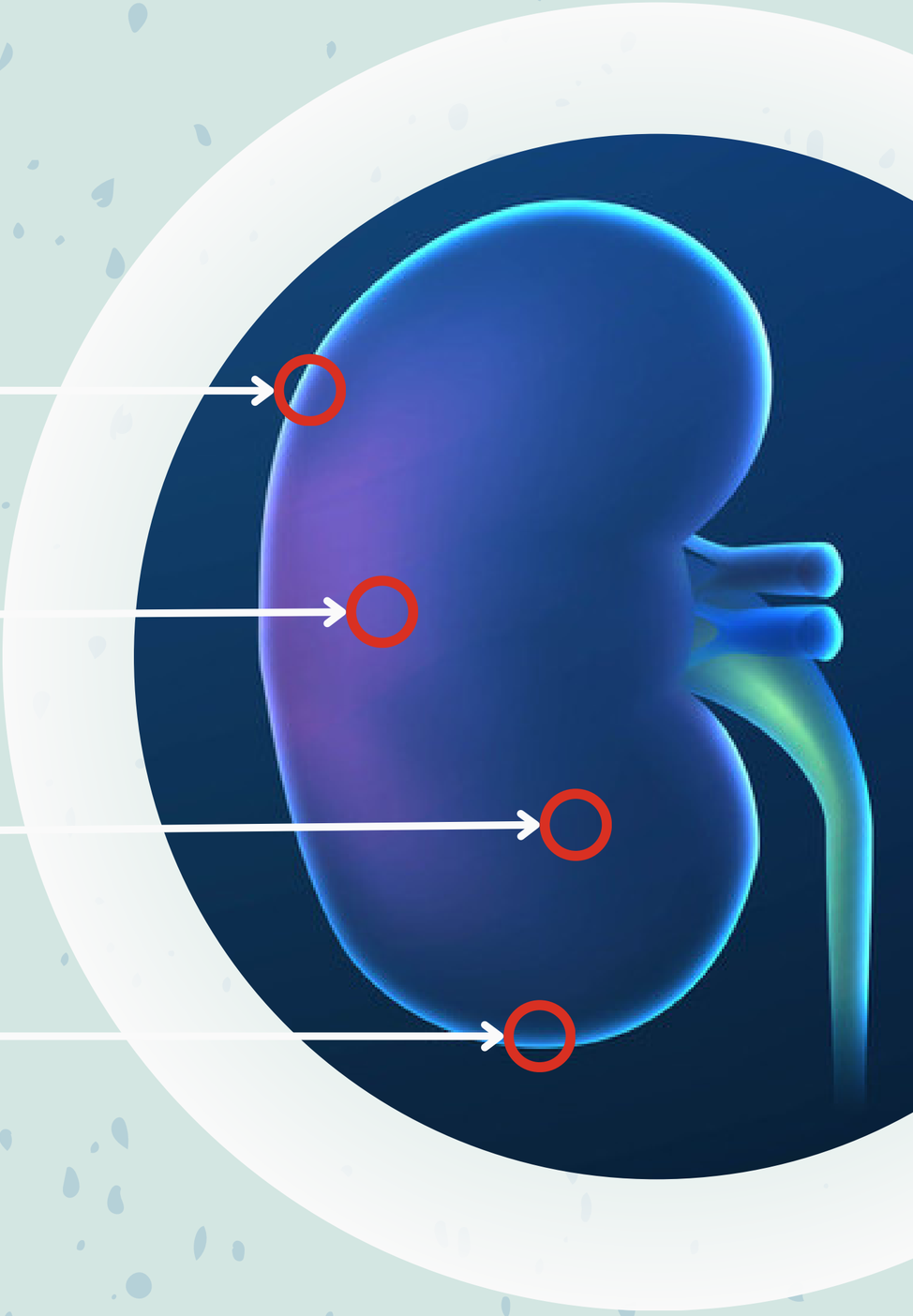
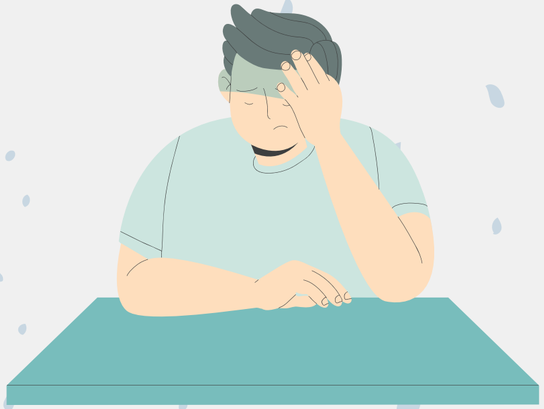


**Table 6 | Causes of AKI: exposures and susceptibilities for non-specific AKI**

| Exposures | Susceptibilities |
|---|---|
| Sepsis | Dehydration or volume depletion |
| Critical illness | Advanced age |
| Circulatory shock | Female gender |
| Burns | Black race |
| Trauma | CKD |
| Cardiac surgery (especially with CPB) | Chronic diseases (heart, lung, liver) |
| Major noncardiac surgery | Diabetes mellitus |
| Nephrotoxic drugs | Cancer |
| Radiocontrast agents | Anemia |
| Poisonous plants and animals | |

CKD, chronic kidney disease; CPB, cardiopulmonary bypass.



# CLINICAL PRESENTATION

- Signs and symptoms of acute kidney injury differ depending on the cause.
- Non specific symptoms: decreased urination-nausea-vomiting-diarrhea-anorexia-fatigue-pruritus).
- More specific: (orange concentrated urine-Weight loss in prerenal AKI)(Brown urine which indicate blood in urine associated with glomerulonephritis)(Weight gain - shortness of breath due to fluid retention)(Anuria alterating with polyuria-colicky abdominal pain in postrenal AKI)

**Physical examination finding:**

- Non specific : change in mental status
- More specific: Hypotension or orthostatic hypotension- rash - bladder distension-prostatic enlargement(in post renal AKI)

**In case of fluid overload:**

- Increase BP- peripheral edema-JVD-pulmonary edema-crackles.

# PROPHYLAXIS: PREVENTIVE MEASURES

1-Patients with AKI and at increased risk for AKI require careful attention to be paid to their hemodynamic status. This is first because hypotension results in decreased renal perfusion and, if severe or sustained, may result in kidney injury. Second, the injured kidney loses autoregulation of blood flow.

2- Management of blood pressure and cardiac output require careful titration of fluids and vasoactive medication. Vasopressors can further reduce blood flow to the tissues if there is insufficient circulating blood volume. Conversely, patients with AKI are also at increased risk for fluid overload and continued fluid resuscitation despite increased intravascular volume can cause harm.

3- Avoid nephrotoxic agents ( Aminoglycosides - IV contrast medium or use low-osmolar or iso-osmolar )
4-Manage underlying conditions as Diabetes and Hypertension

# NON-PHARMACOLOGIC TREATMENT

01

IV crystalloids indicated in patients with hypovolemia to support the kidney function.

02

Dietary adjustment to decrease kidney stress.

03

Lifestyle modifications such as optimal hydration and avoid nephrotoxic drugs.

04

Renal Replacement Therapy(RRT) is necessary in patients with established AKI to treat resistant pulmonary edema and volume overload(unresponsive to diuretics) ,minimize accumulation of nitrogenous waste products, and correct electrolyte and acid–base abnormalities (eg, hyperkalemia, metabolic acidosis) while renal function recovers.

# PHARMACOLOGIC TREATMENT

## GENERAL TREATMENT REGARDLESS AKI TYPE:

1

No specific treatments improve the outcome of AKI generally as the management focusing on give intravenous (IV) administration of crystalloid solution to keep the patient euvolemic or even hyper-volemic and treatment of any acute illness triggering AKI.

2

Stopping medication that may exacerbate the renal injury.

3

The most important drugs according to KDIGO in this respect are ACEI/ARB and diuretics.

# PHARMACOLOGIC TREATMENT

## SPECIFIC TREATMENT

01

### PRE-RENAL AZOTEMIA

**Treatment focus on correcting primary hemodynamics by:**

a. Normal saline, if volume depleted
b. Pressure management, if needed
c. Blood products, if needed
d. Hold or discontinue medications that affect renal hemodynamics (ACEIs, ARBs, NSAIDs).

02

### INTRINSIC AKI

**Treatment focus on:**

a. Eliminate the causative hemodynamic abnormality
b. Avoid additional insults.
c. Manage fluid and electrolytes to prevent volume depletion or overload
d. Nutrition support is important.
e. Medical therapy according to the KDIGO guidelines

03

### POSTRENAL AKI

**Treatment focus on:**

a.Relieve obstruction.
b.Consult urology or radiology.

# TEAMWORK

## LAB 1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