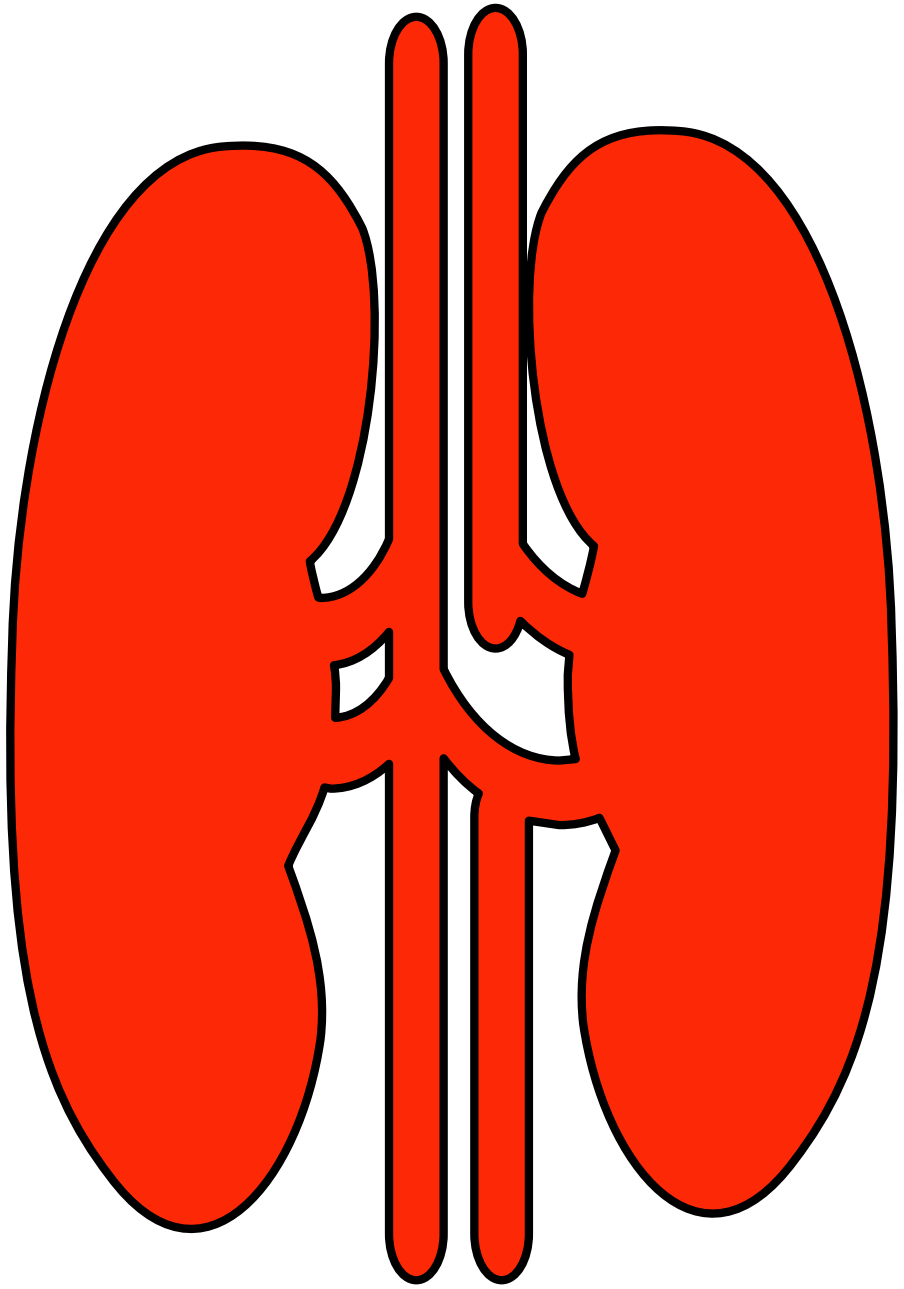
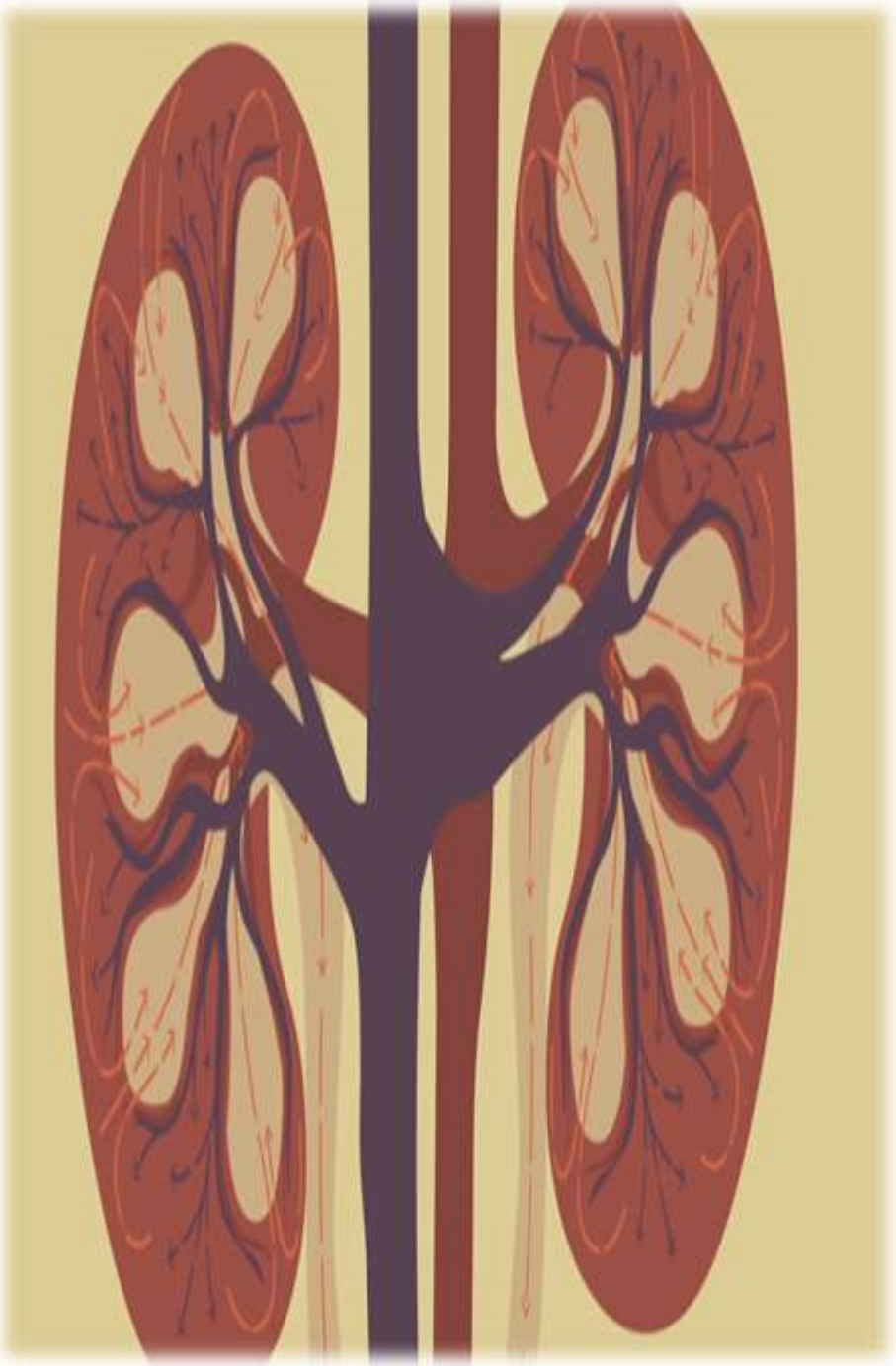


DISEASES AFFECTING TUBULES AND INTERSTITIUM

- tubulointerstitial diseases

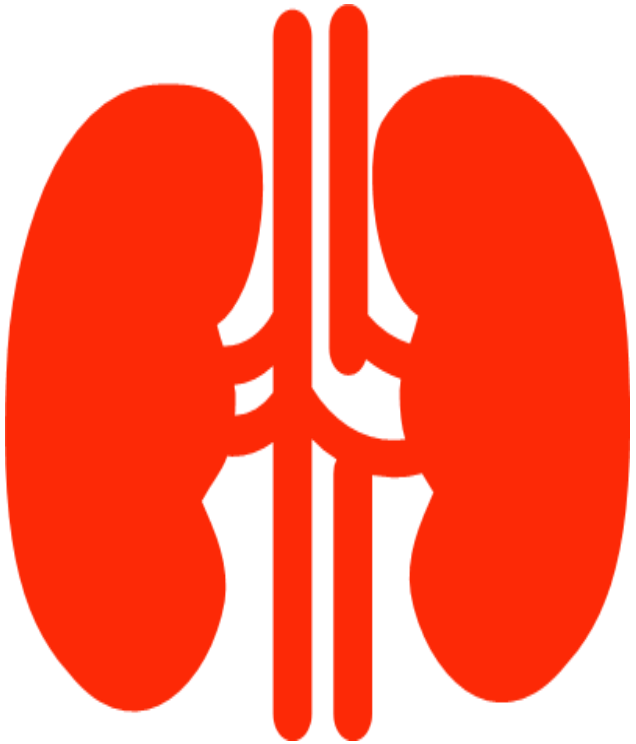
Introduction

by : Aya alsarayreh



Tubulointerstitial disorders are diseases primarily affect the renal tubules and interstitial components of the renal parenchyma with sparing of the glomerulus.

BUT in severe and prolonged cases , the entire kidneys may become involved
with glomerular dysfunction and even progress to Renal failure



- Tubulointerstitial disorders
  - Acute & Chronic interstitial nephritis

Acute Interstitial Nephritis

A. General characteristics

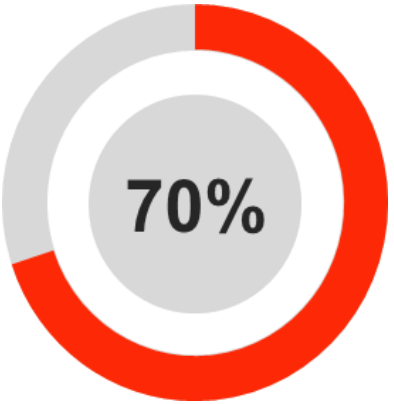
1. Acute inflammation affecting the tubulo-interstitium of the kidney. It is commonly drug-induced but can be caused by other factors, such as renal toxins, and can complicate a variety of systemic diseases and infections.

2. Accounts for 15% of cases of acute kidney injury (intrinsic renal failure)

Causes

Acute allergic reaction to medication

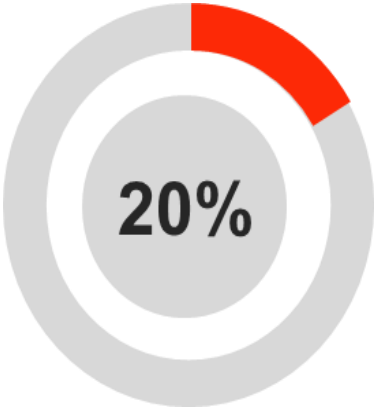
is the most common cause—for example, NSAIDs (MC), penicillin, cephalosporins, sulfa drugs, diuretics (furosemide, thiazide), anticoagulants, phenytoin, rifampin, allopurinol, proton pump inhibitors.



[1]

Autoimmune and systemic diseases

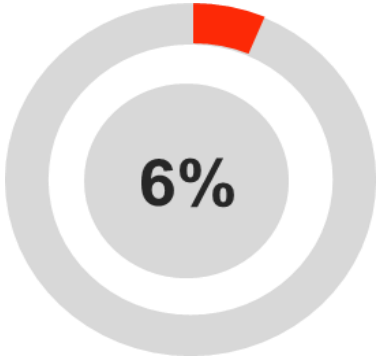
for example, SLE, Sjögren syndrome,



[1]Biopsy-Proven Acute Interstitial Nephritis, 1993-2011 - Angela K. Muriithi, MBChB, MPH

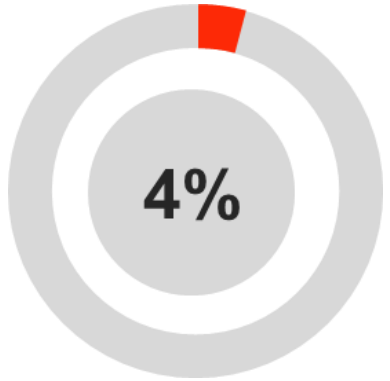
Collagen vascular diseases

for example, sarcoidosis



Infection

- Acute bacterial pyelonephritis, Leptospirosis, Tuberculosis
-Commonly occurs in children)—due to a variety of agents, including Streptococcus spp. and Legionella pneumophila
- also some viral and fungal infection



Nephrotoxins

Many agents may be toxic to the kidneys either by direct damage to the tubules, or by causing an interstitial nephritis (see earlier in topic). Examples (not an exhaustive list and idiosyncratic reactions are possible):

Analgesics: NSAIDs

Antimicrobials: Aminoglycosides sulfamethoxazole (in co-trimoxazole), penicillins, rifampicin, amphotericin, aciclovir.

Anticonvulsants: Lamotrigine, valproate, phenytoin.

Other drugs: PPIs, cimetidine, furosemide, thiazides, ACE-i/ARB, lithium, iron, calcineurin inhibitors, cisplatin.

Anaesthetic agents: Methoxyflurane, enflurane.

Radiocontrast material:

Proteins: Igs in myeloma, light chain disease, Hb in haemolysis, myoglobin in rhabdomyolysis

Crystals: Urate

Bacteria: Streptococci, *Legionella*, *Brucella*, *Mycoplasma*, *Chlamydia*, TB, *Salmonella*, *Campylobacter*, leptospirosis, syphilis.

Viruses: EBV, CMV, HIV, polyomavirus, adenovirus, measles.

Parasites: *Toxoplasma*, *Leishmania*.

Other: Ethylene glycol, radiation aristolochic acid

Pathophysiology …

- Acute drug-induced tubulointerstitial nephritis causes AKI.

- It entails infiltration by activated T cells and eosinophils, indicating a **type IV cell-mediated** immune reaction.

- The immunogen could be the drug itself, the drug bound to certain tissue components, a drug metabolite or a tissue component altered by the drug.

- Can occur immediately after the 1st does of the drug

- NSAID-induced AIN is different in that the NSAIDs are typically ingested for months before symptoms occur

Acute Interstitial Nephritis

by : waed alharahsheh

Acute Interstitial Nephritis

B. Clinical features

1. Signs and symptoms of acute renal dysfunction: acute or subacute onset of nausea, vomiting, and malaise .

2. Patients may present with symptoms related to the cause of the AIN.

In patient with drug-induced AIN:

- Signs of a generalised drug hypersensitivity reaction with ***fever, rash, joint pain and eosinophilia & eosinophiluria*** Withdrawal of the drug reverses the disease.

- Proteinuria Contrary to all other types of AIN, NSAID-induced AIN may cause nephrotic-range proteinuria

Rash, fever, and eosinophilia are frequently absent

3. Acute interstitial nephritis (AIN) causes AKI and its associated symptoms. Many patients are not oliguric, despite moderately severe renal impairment,
AIN should always be considered in patients with non-oliguric AKI.

However, approximately 50% of patients have oliguria.

Acute Interstitial Nephritis

C. Investigations & Diagnosis

1. Urinalysis (the initial test to do)

a. Eosinophils in the urine suggest the diagnosis, hansel's stain, giemsa stain

b. Mild proteinuria or microscopic hematuria may be present

c. may contain red and white blood cells

2.kidney function tests (increased BUN and Cr levels)

3. High fractional sodium excretion- >1%, which is in part indicative of tubular damage.

4. Increased serum eosinophils and IgE levels

5. Rarely biopsy the most accurate

Diagnosing AIN :

- look for recent infection,
- start of a new medication,
- rash, fever, general pains,
- signs/symptoms of AKI.

When do we suggest renal biopsy?

We suggest a kidney biopsy for the following patients :

- uncertainty for the diagnosis .
- Patients with any features (such as high-grade proteinuria) that cause the diagnosis of AIN to be uncertain.
- Patients who present with advanced (progressive) renal failure .
- No improvement after causative agent has stopped .

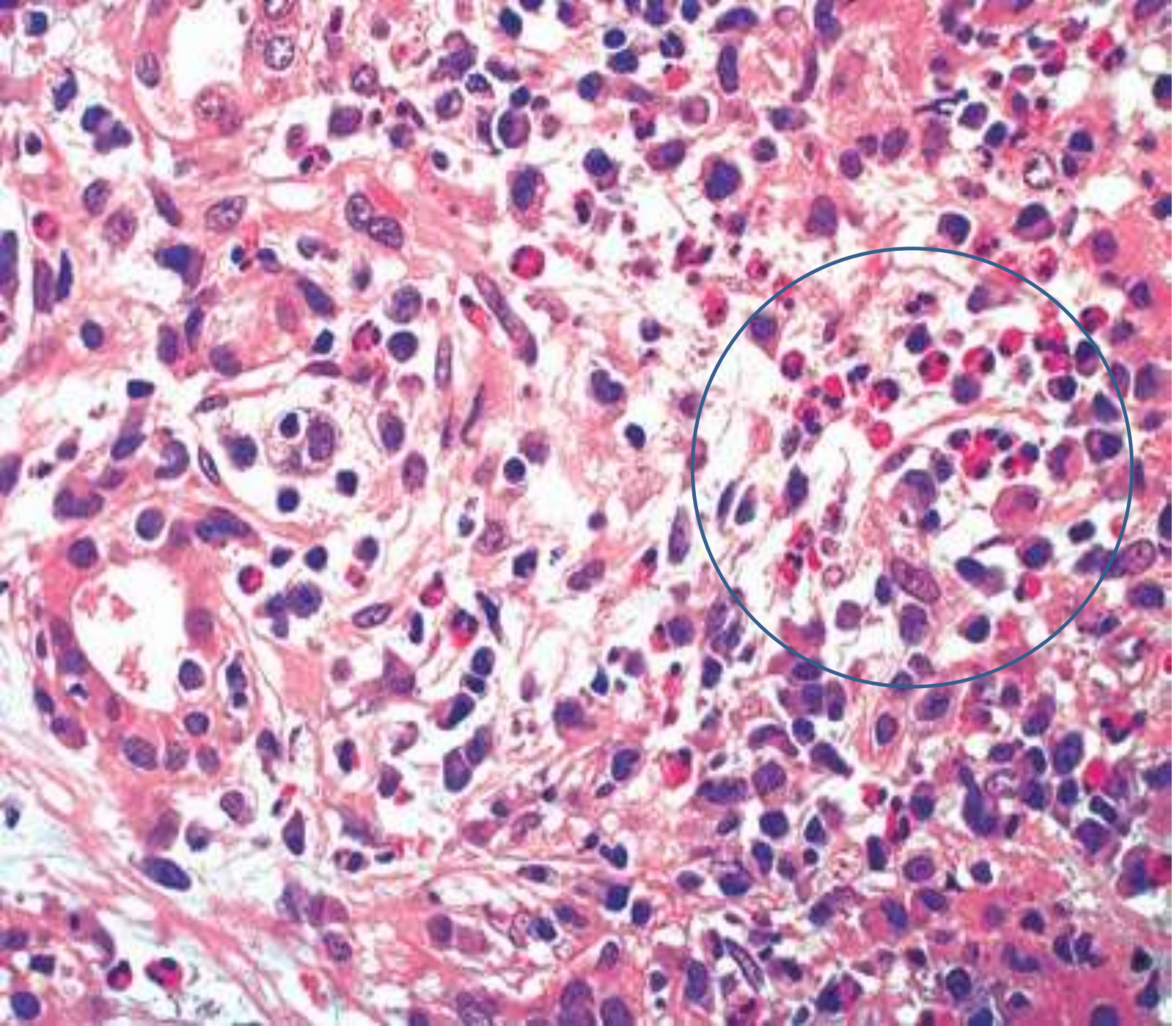
Histology

-The major histologic changes are:

- Interstitial edema
- Marked interstitial infiltrate consisting primarily of T lymphocytes and monocytes. Eosinophils, plasma cells, and neutrophils also may be found.

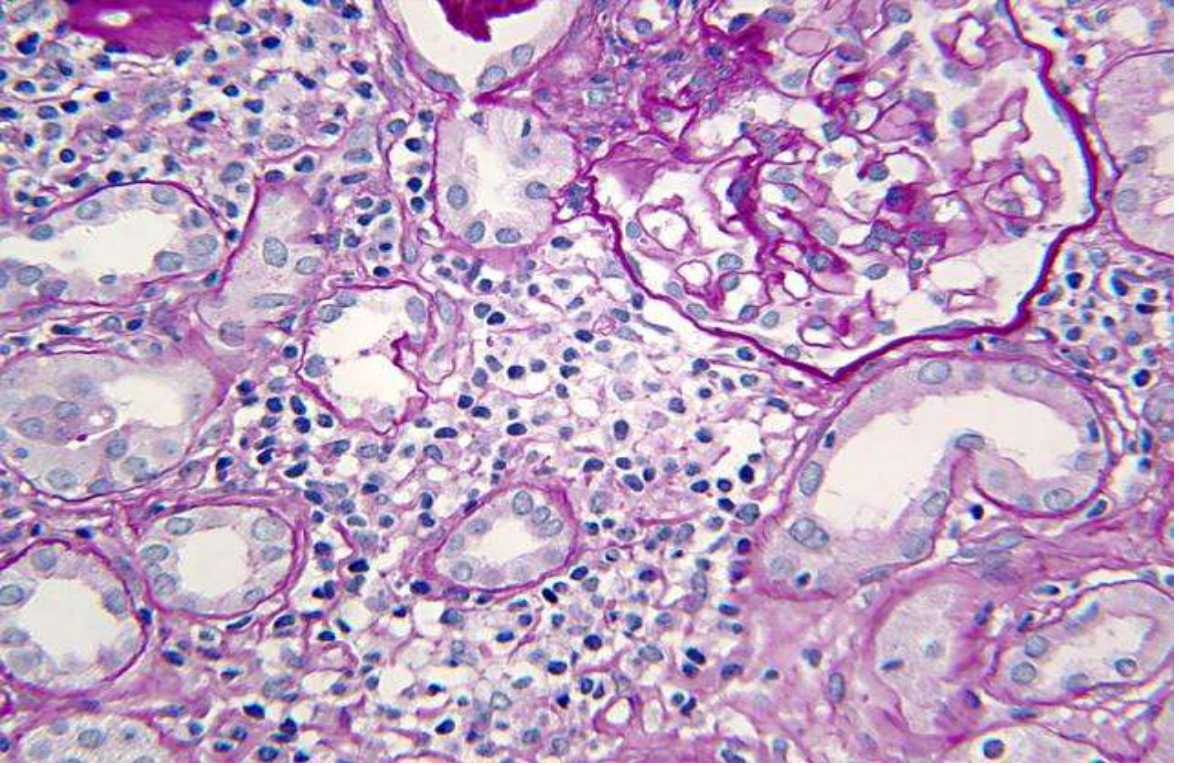
- Special histologic feature:
Granuloma: sarcoidosis, infection (e.g. Mycobacterium)

Note that it is often impossible to distinguish AIN from ATN based on clinical grounds alone. Renal biopsy is the only way to distinguish between the two, but is usually not performed.



In acute **drug-induced interstitial nephritis** biopsy shows a marked mononuclear and eosinophil infiltrate in the interstitium.

In the circle, note the mononuclear infiltrate (lymphocytes, macrophages, and plasma cells) and abundant **eosinophils**.

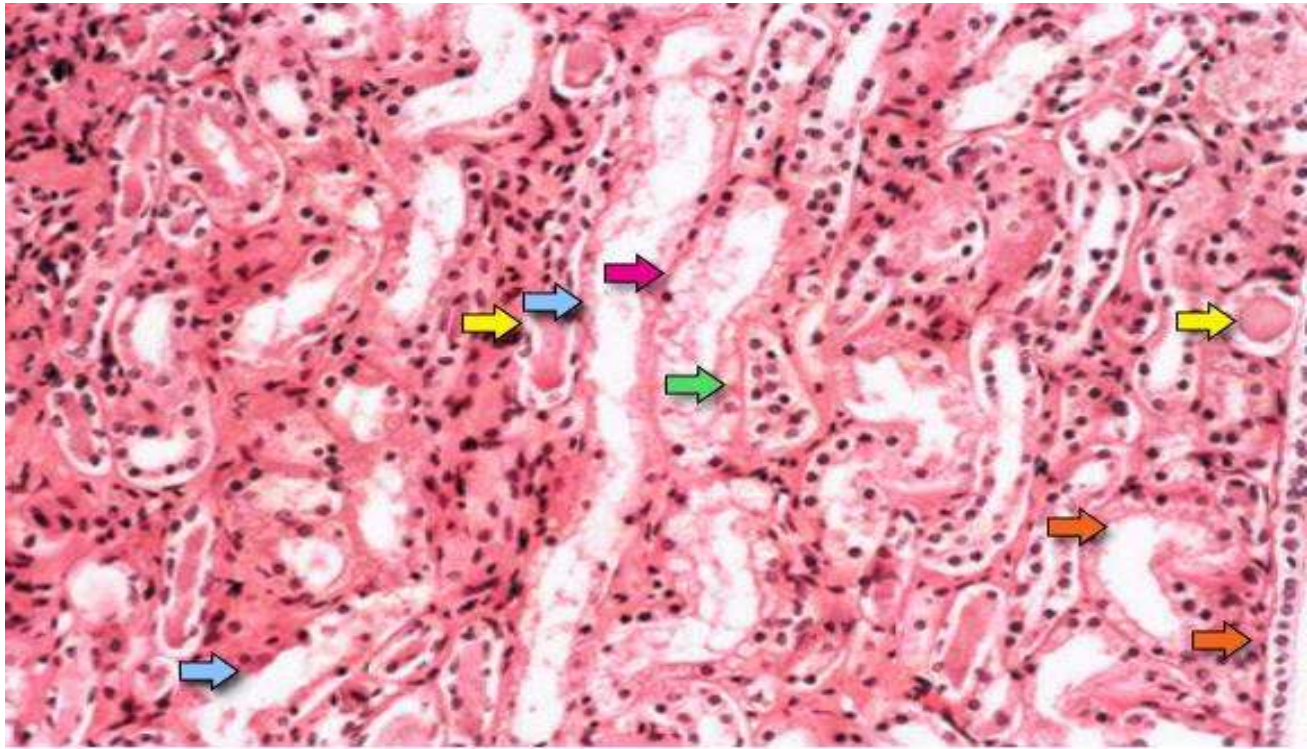


Acute Interstitial Nephritis

The renal cortex shows a diffuse interstitial, predominantly mononuclear, inflammatory infiltrate with **no changes to the glomerulus**. Tubules in the center of the field are separated by inflammation and edema, as compared with the more normal architecture in the right lower area

Acute Tubular Necrosis

. Features suggesting acute tubular necrosis are the patchy or diffuse denudation of the renal tubular cells with **loss of brush border** (blue arrows); **flattening of the renal tubular cells** due to **tubular dilation** (orange arrows); intratubular **cast formation** (yellow arrows); and sloughing of cells, which is responsible for the formation of granular casts (red arrow). Finally, intratubular obstruction due to the denuded epithelium and cellular debris is evident (green arrow);



Acute Interstitial Nephritis

D. Treatment

1. withdrawal of the drug .

Some patients with drug-induced AIN recover following withdrawal of the drug alone,.

2. Treat infection if present .

3. Steroids may help

high-dose corticosteroids (prednisolone 1 mg/kg/day) accelerate recovery and may prevent long-term scarring.

4. Dialysis

sometimes necessary but is usually only short-term. Other specific causes should be treated, if possible

PROGNOSIS

- Recovery of renal function was observed in the great majority of cases of AIN , either with discontinuation of the offending drug or with steroids therapy..

- Acute dialysis maybe required, but only approximately 10 percent of patients remain dialysis dependent.

- Recovery of kidney function is often incomplete, with persistent elevation of the serum creatinine concentration in up to 40 percent of patients. The final serum creatinine concentration did not correlate with the maximum value during AIN.

- Clinical indicators of a decreased likelihood of recovery include prolonged renal failure (greater than three weeks), AIN associated with NSAID use, and certain histologic findings (including interstitial **granulomas**, interstitial **fibrosis**, and tubular **atrophy**) on kidney biopsy.

CHRONIC INTERSTITIAL NEPHRITIS (CIN)

By : Abdullah almomani

chronic interstitial nephritis is characterized by :

- 1) renal dysfunction
- 2) fibrosis
- 3) infiltration of the renal parenchyma by lymphocytes, plasma cells and macrophages.
- 3) tubular damage

CIN is caused by:

Acute Interstitial Nephritis	If Persistent
Glomerulonephritis	Varying degrees of interstitial inflammation occurs with glomerulonephritis
Immune	Sarcoidosis Systemic lupus erythematosus Sjogren's syndrome
Toxic	Aristolochia in herbal medicine Lead, cadmium
Drugs	Analgesic abuse, cyclosporine, cisplatin
Infection	Consequence of severe pyelonephritis
Congenital/developmental	Vesico-uretral reflux sickle cell disease

Analgesic-Abuse Nephropathy

- Analgesic-abuse nephropathy had been associated with ***Phenacetin***, a key ingredient in the OTC meds, but this has been removed from the U.S market.
- Use of the ***acetaminophen*** (major metabolite of phenacetin) particularly **in combination with** ASA (acytelsalicyclic acid, aspirin) is associated with increased risk of chronic interstitial nephritis, especially if it's further combined with mixtures containing **caffeine** or ***codeine***. Chronic use of NSAIDs is associated with renal failure in some patients.

Analgesic-Abuse Nephropathy

- Typically occurs in a patient with a history of frequent pain and **presents with: low urine specific gravity** (loss of the concentrating ability), minimal **proteinuria**, **sterile pyuria** and **elevated creatinine**.
- **Papillary necrosis** is the **most common** ultimate consequence.
- **Diagnosis: noncontrast CT** of the kidney may show the papillary necrosis, but not always

Treatment: ttt is **supportive** and with **discontinuation of the offending analgesic.**

Reflux Nephropathy

- This condition, which was previously known as chronic pyelonephritis, is a specific type of chronic interstitial nephritis associated with **vesico-ureteric reflux (VUR)** in early life and with the appearance of **scars in the kidney**.
- Reflux nephropathy is thought to be due to chronic reflux of urine from the bladder into the ureters, in association with recurrent UTI in childhood. It was previously assumed that ascending infection was necessary for progressive renal damage in patients with VUR but there is evidence to suggest that renal scars can occur, even in the absence of infection.
- Susceptibility to VUR has **a genetic component** and may be associated with renal dysplasia and other congenital abnormalities of the urinary tract. It can be connected with outflow obstruction, usually caused by urethral valves, but usually occurs with an apparently normal bladder.

Reflux Nephropathy

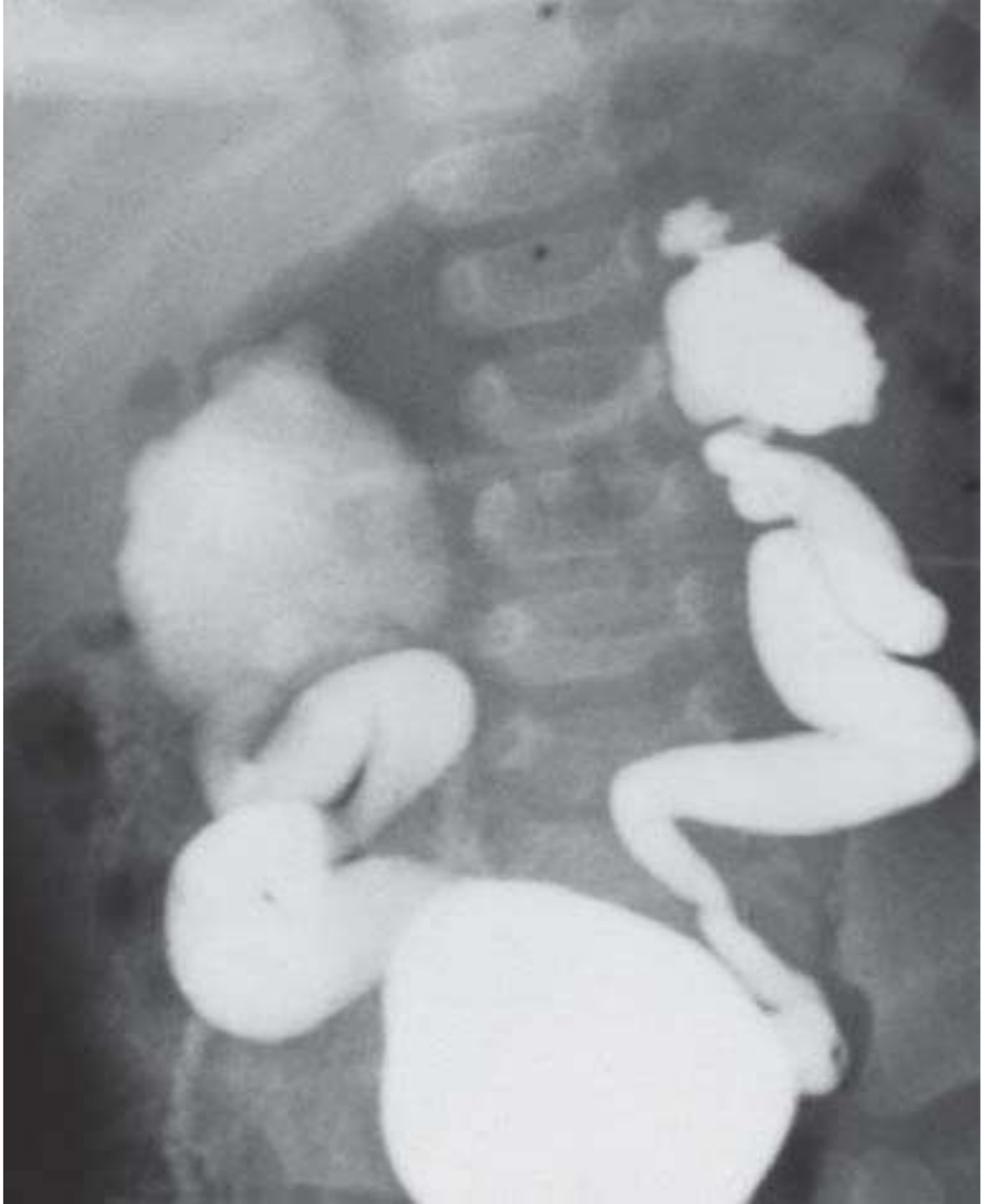
- ***Clinical features:***
  -The **renal scarring and dilatation** are asymptomatic and the patient may present at any age with **hypertension** (sometimes severe), **proteinuria** or **features of CKD.**

  -Symptoms arising from the urinary tract may be present and include **frequency of micturition**, **dysuria** and **aching lumbar pain.**

  -Unilateral scars are associated with compensatory hypertrophy of the contralateral kidney.

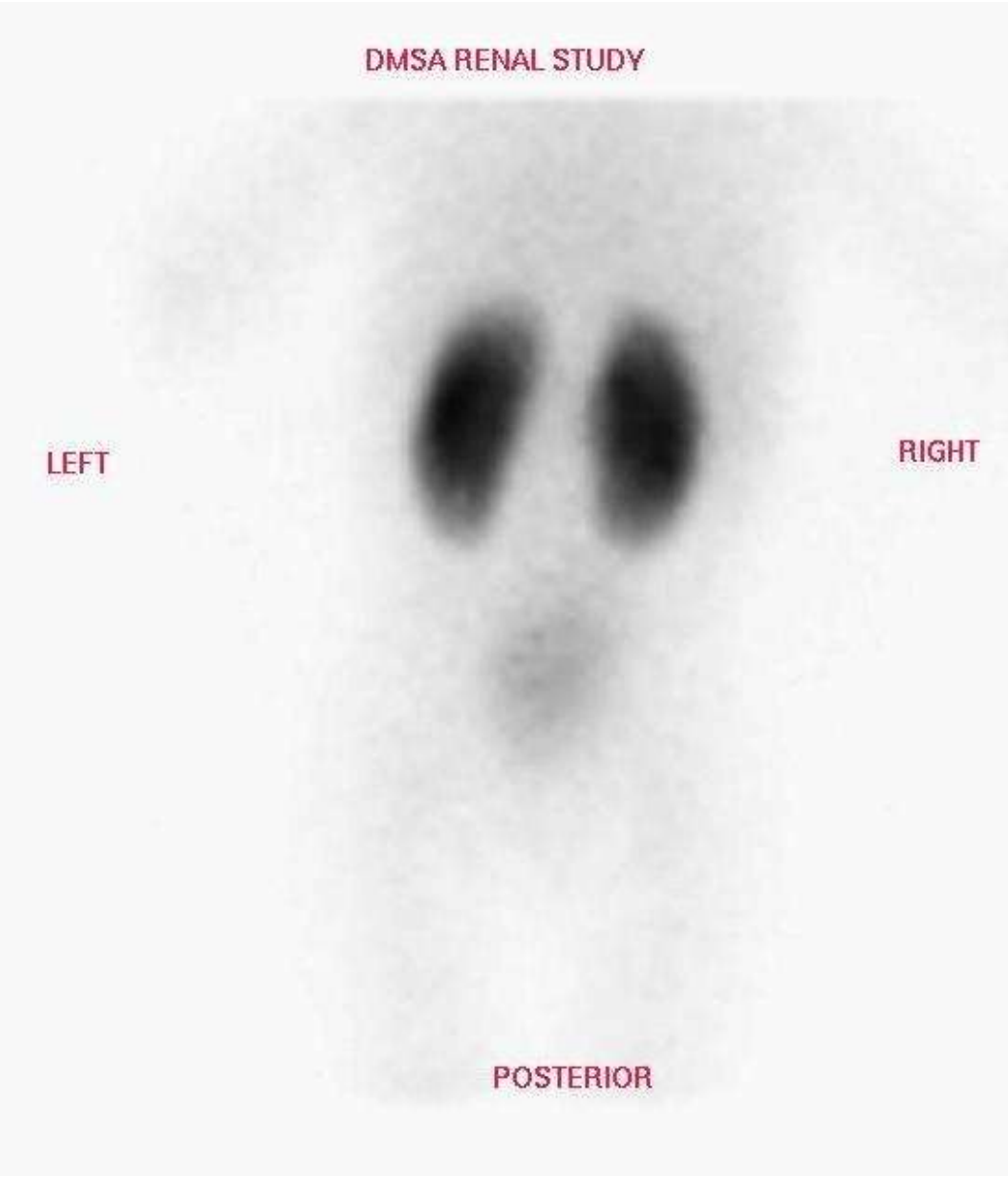
Reflux Nephropathy

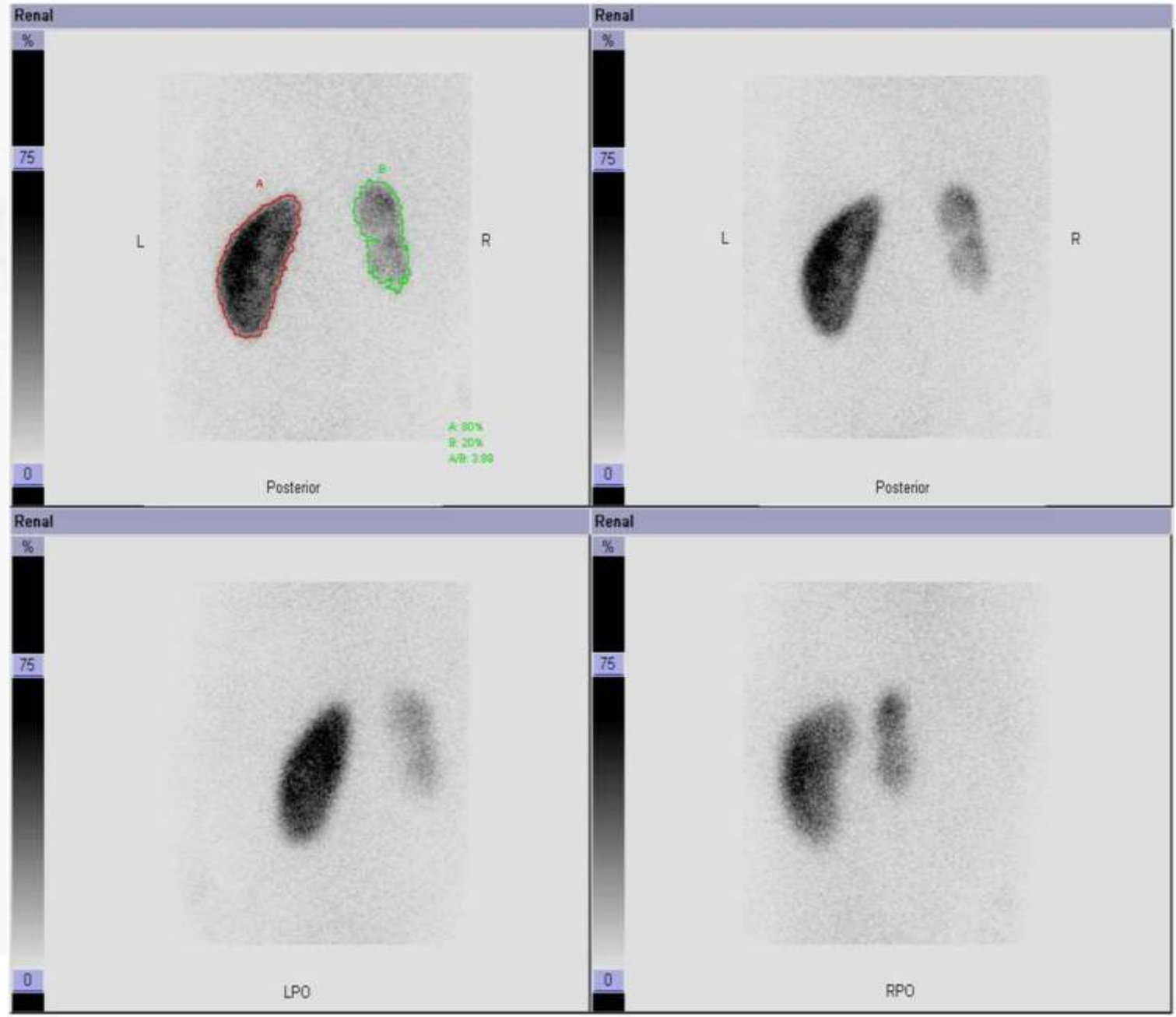
- ***I*nvestigations**
  1) **MCUG (micturating cystourethrography)**: is the way by which we can **diagnose VUR** and that is done by filling the bladder with contrast media through a urinary catheter and images are taken during and after micturition.
  2) **Ultrasound**: has a **poor sensitivity** and capable of detecting significant obstruction and scarring.
  3) **MRI and CT**: may be useful in patients **follow-up and assessing disease progression.**
  4) **DMSA scan**: are the **most sensitive test.**



Vesico-ureteric reflux shown by micturating cystogram. The bladder has been filled with contrast medium through a urinary catheter. **After micturition, there was gross vesico-ureteric reflux into widely distended ureters and pelvicalyceal systems**

DMSA RENAL STUDY





Reflux Nephropathy

- ***Management:***
  - Infection, if present, should be treated; if recurrent, it should be prevented with prophylactic therapy, as described for UTI.

  -As most childhood reflux tends to disappear **spontaneously** and trials have shown small or no benefits from anti-reflux surgery, such intervention is now less common.

Sickle-cell nephropathy

- Improved survival of patients with sickle-cell disease means that a high proportion now live to develop chronic complications of **microvascular occlusion**.
- In the kidney, these changes are most pronounced in the medulla, where **the vasa recta** are the site of **sickling** because of **hypoxia** and hyper tonicity.
- Loss of urinary concentrating ability and **polyuria**. **Papillary necrosis** may also occur.
- A minority of patients **develop ESRD**. Patients with sickle trait have an increased incidence of unexplained non-visible haematuria.

Chronic Interstitial Nephritis

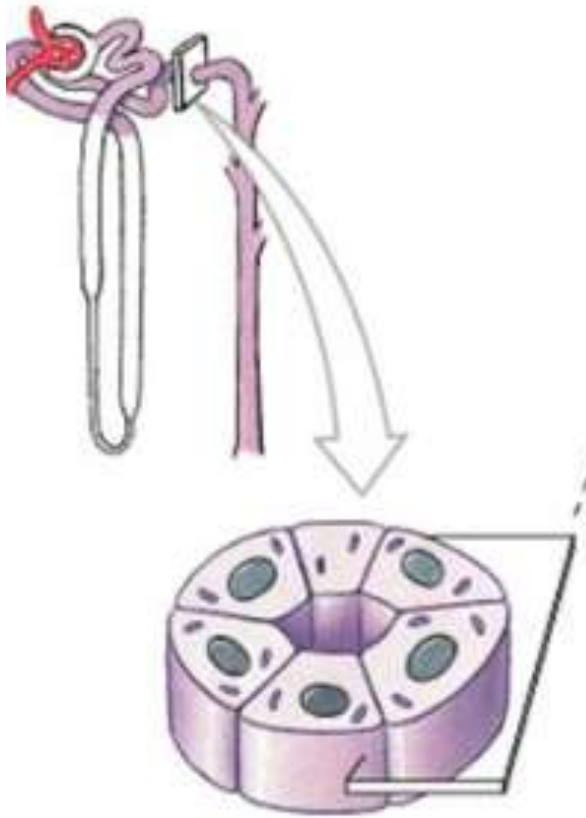
- ***Clinical features :***
  - **Most patients** with CIN **present in adult life with CKD**, **hypertension** and **small kidneys**. **Urinalysis abnormalities** are non-specific.
  -**A minority** present with **salt-losing nephropathy**, characterized **hypotension**, **polyuria** and features of **sodium and water depletion**.

  -People with CIN have an **impairment of urine-concentrating** ability and sodium conservation, which puts them at **risk of AKI** due to salt and water depletion during an acute illness.
- ***Management:***
  Management is **supportive** in nature, with **correction of acidosis and hyperkalemia**; **replacement of fluid** and electrolytes, as required; and **renal replacement therapy** if irreversible renal damage has occurred.

RENAL TUBULAR ACIDOSIS

by : ghaith alhanahena

Kidney tubules and PH regulation-buffer



1. Carbonic acid–bicarbonate buffer system
2. Phosphate buffer system
3. Ammonia buffer system

Tubular fluid in lumen

Metabolic generation in tubule cell

Peritubular fluid

Capillary

CO_2 + H_2O

Carbonic anhydrase

H_2CO_3

1 CO_2 + H_2O ⇌ H_2CO_3 ⇌ HCO_3^- + H^+

2 $H_2PO_4^-$ ⇌ HPO_4^{2-} + H^+

3 NH_4^+ ⇌ NH_3 + H^+

Na^+ K^+ Cl^- H^+ HCO_3^-

Cells of PCT, DCT, and collecting system

(a)

KEY

- = Leak channel
- = Countertransport
- = Active transport
- = Exchange pump
- = Cotransport
- = Reabsorption
- = Secretion
- = Diffusion

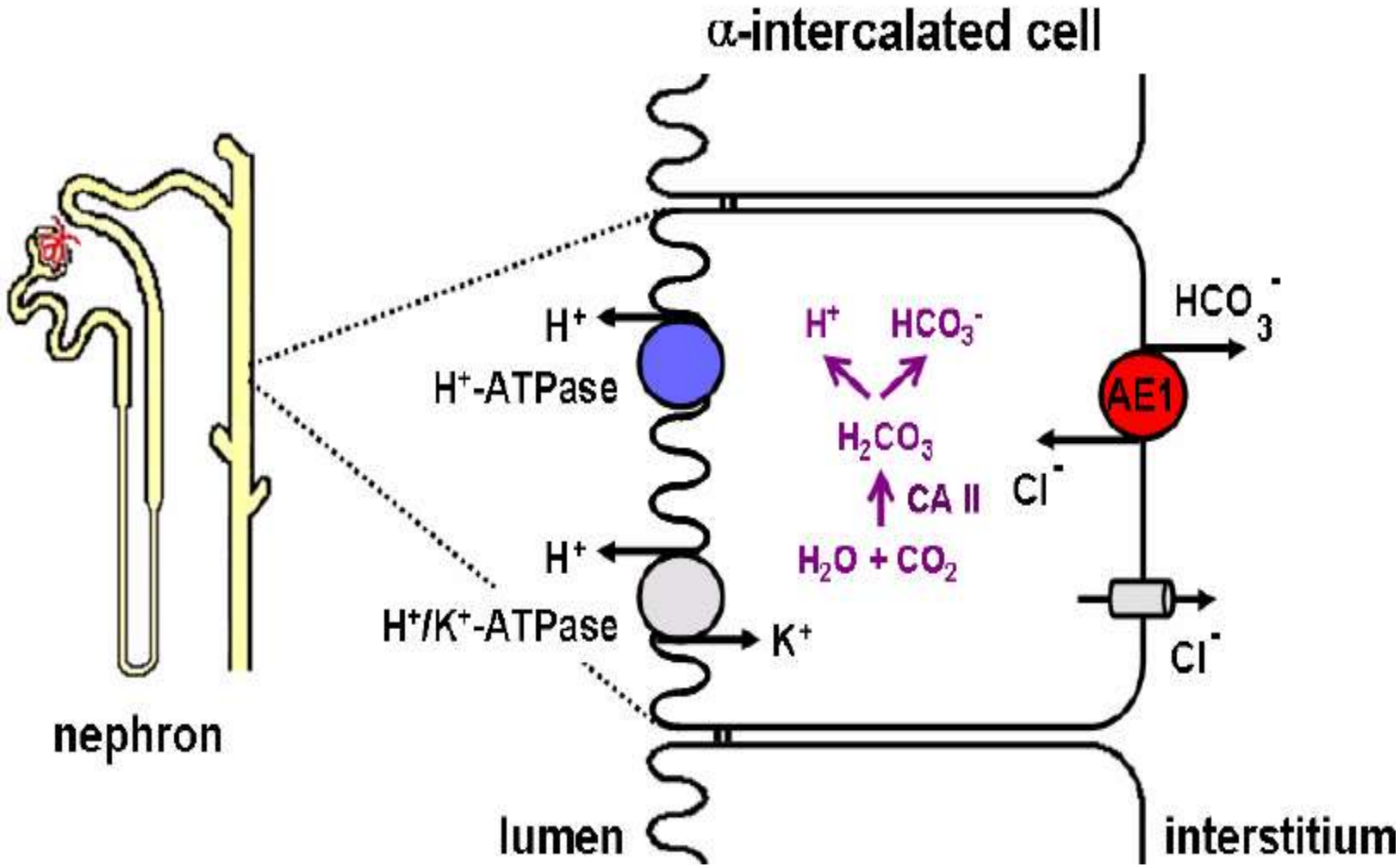
Renal Tubular Acidosis

A. General characteristics :

1. Renal tubular acidosis (RTA) is a disorder of the renal tubules that leads to a non-anion gap hyperchloremic metabolic acidosis. Glomerular function is normal

2. It is characterized by a decrease in the proton excreted in the urine, leading to acidemia and urine alkalosis.

3. There are three types of RTA (types 1, 2, and 4). (Type 3 RTA is a term that is no longer used.)


α-intercalated cell
nephron
H+
H+-ATPase
H+
H+/K+-ATPase
K+
H+ HCO3-
H2CO3
CA II
H2O + CO2
HCO3-
AE1
Cl-
Cl-
lumen
interstitium

Type 1 (distal)

1. **The defect is an <u>inability to secrete</u> H+ at the distal tubule** (therefore new bicarbonate cannot be generated). This inability to acidify the urine results in metabolic acidosis. Although normally the urine pH can be as low as 4.7, in distal RTA the urine pH cannot be lowered below 6, regardless of the severity of metabolic acidosis.
2. **<u>It leads to increased excretion of ions</u>** (sodium, calcium, potassium, sulfate, phosphate), with the following effects:

a. Decrease in ECF volume.

b. Hypokalemia.

c. Renal stones/nephrocalcinosis (due to increased calcium and phosphate excretion into alkaline urine).

d. Rickets/osteomalacia(decrease Ca, phosphate) in children.

3. **<u>Leads to hypokalemic, hyperchloremic, nonanion gap metabolic acidosis</u>**.

Type 1 (distal) cont.

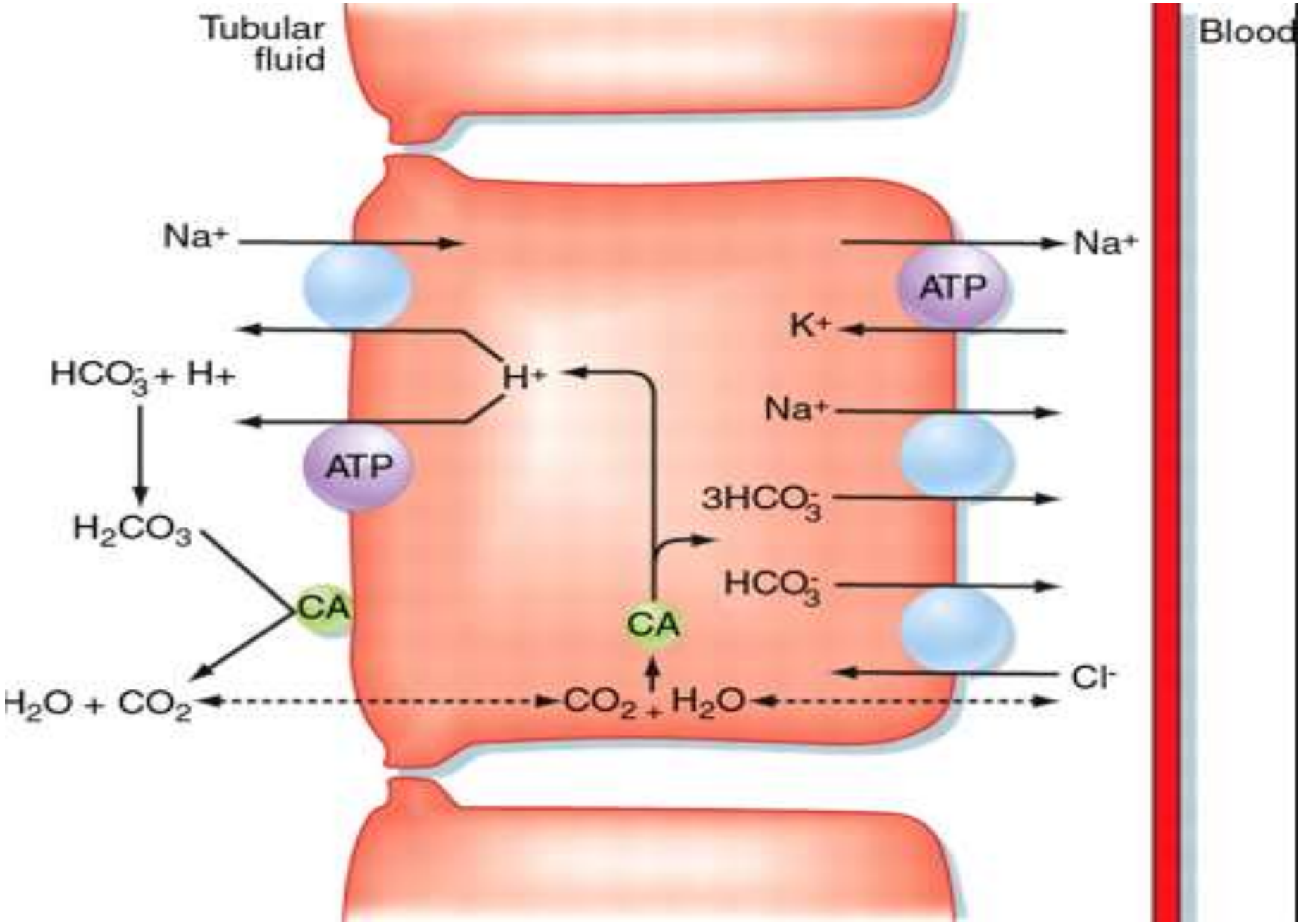
4. Symptoms are secondary to nephrolithiasis and nephrocalcinosis. Up to 70% of patients have kidney stones.

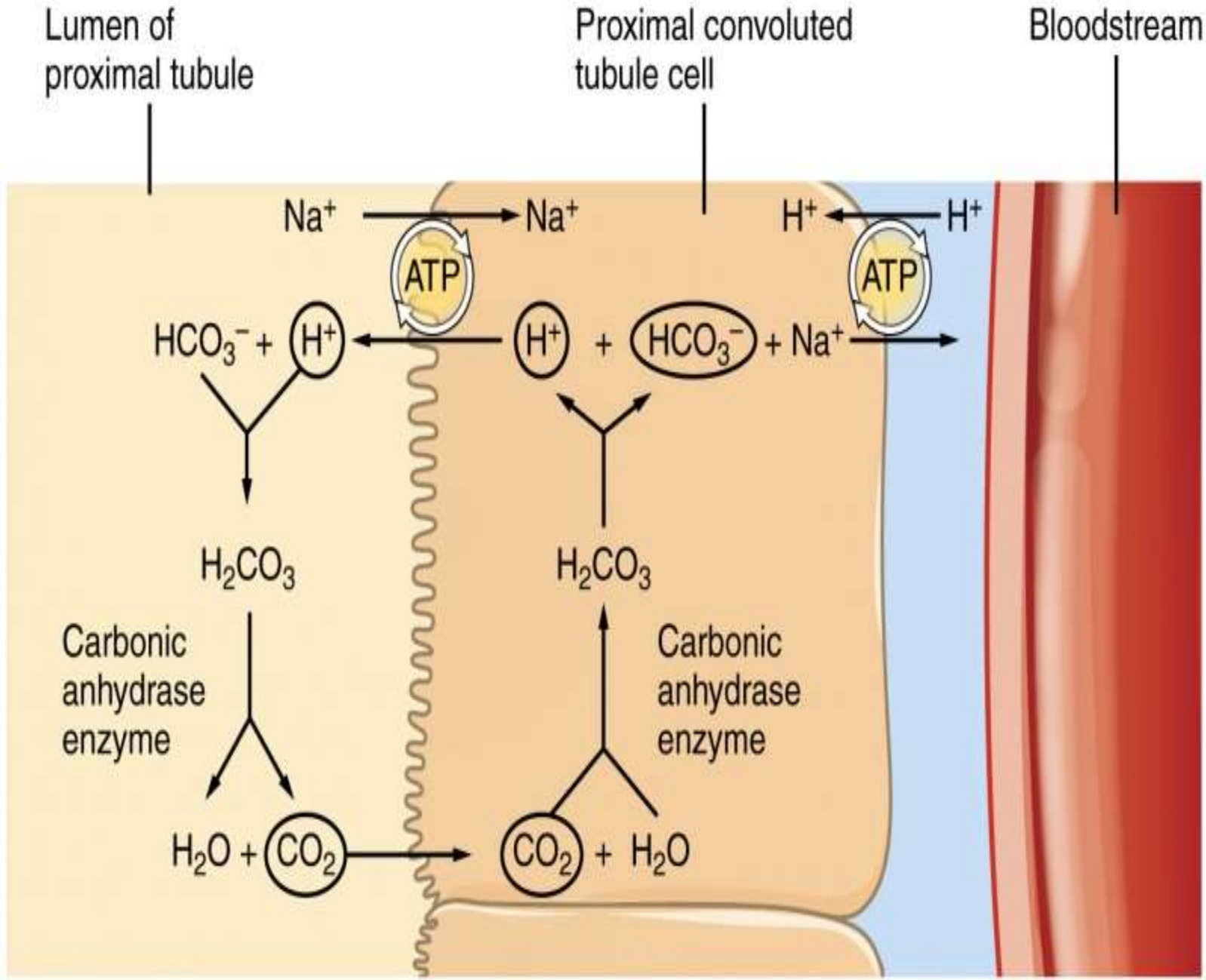
5. Causes:
congenital, multiple myeloma, nephrocalcinosis, nephrotoxicity (e.g., amphotericin B toxicity), autoimmune diseases (lupus, Sjögren syndrome), medullary sponge kidney, and analgesic nephropathy

6. Treatment :

a. Correct acidosis with sodium bicarbonate. This can also help prevent kidney stones, which is a major goal of therapy.

b. Administer phosphate salts (promotes excretion of protons).


Tubular fluid
Blood
Na+
Na+
ATP
K+
HCO3- + H+
H+
Na+
ATP
3HCO3-
H2CO3
HCO3-
CA
CA
Cl-
H2O + CO2
CO2 + H2O


Lumen of proximal tubule
Proximal convoluted tubule cell
Bloodstream
Na+
Na+
H+
H+
ATP
ATP
HCO3− + H+
H+ + HCO3− + Na+
H2CO3
H2CO3
Carbonic anhydrase enzyme
Carbonic anhydrase enzyme
H2O + CO2
CO2 + H2O

Type 2(proximal)

1. The defect is an inability to reabsorb HCO3- at the proximal tubule, resulting in **increased excretion of bicarbonate in the urine** and metabolic acidosis. The patient also loses K+ and Na+ in the urine

2. Characterized by hypokalemic, hyperchloremic non-anion gap metabolic acidosis (as in type 1 RTA)

3. Causes :

a. Fanconi syndrome (in children)

b. Cystinosis, Wilson disease, lead toxicity, multiple myeloma, nephrotic syndrome, amyloidosis

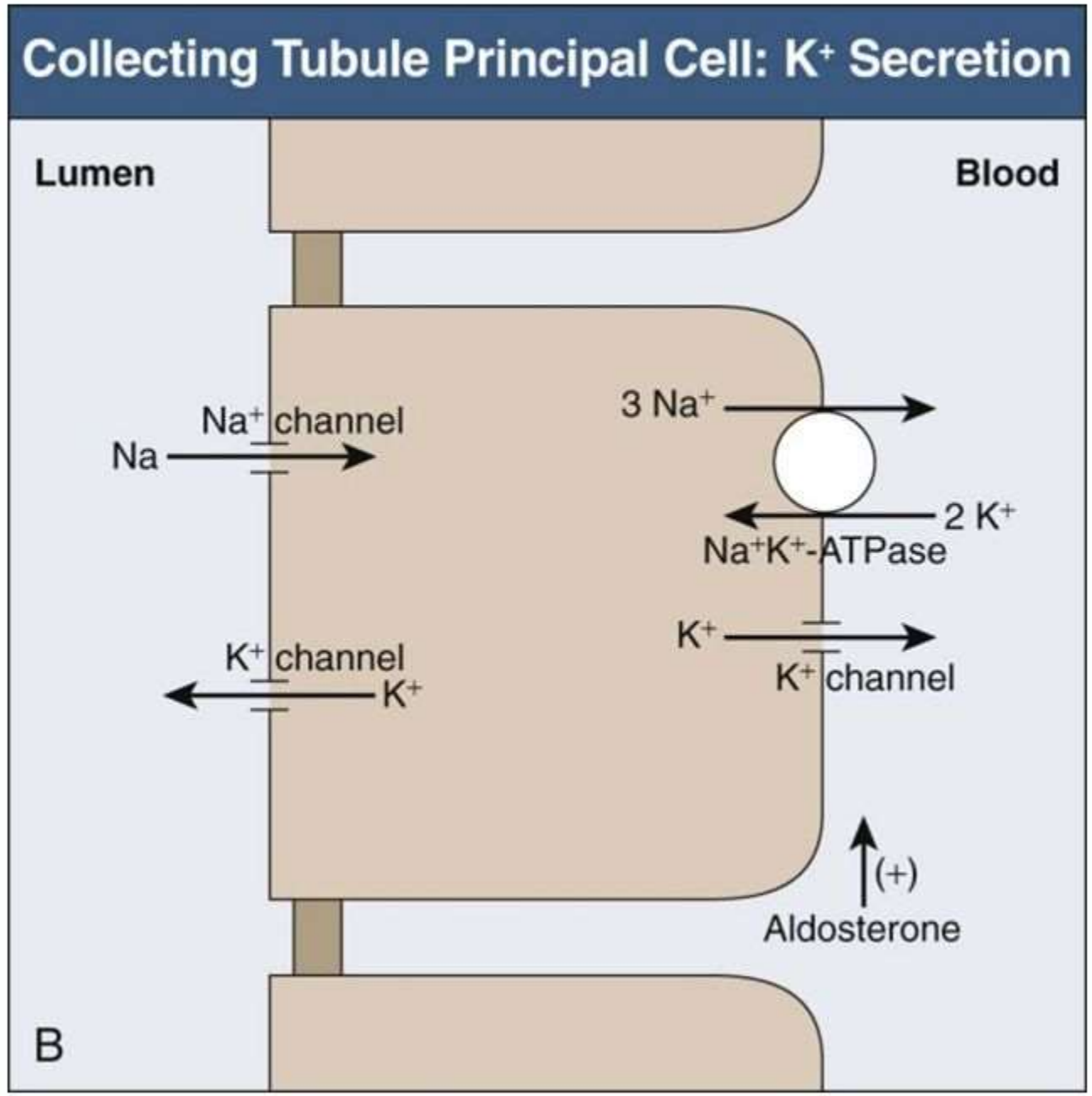
c. The excretion of monoclonal light chains is a common feature, so multiple myeloma should always be ruled out in a patient with proximal RTA

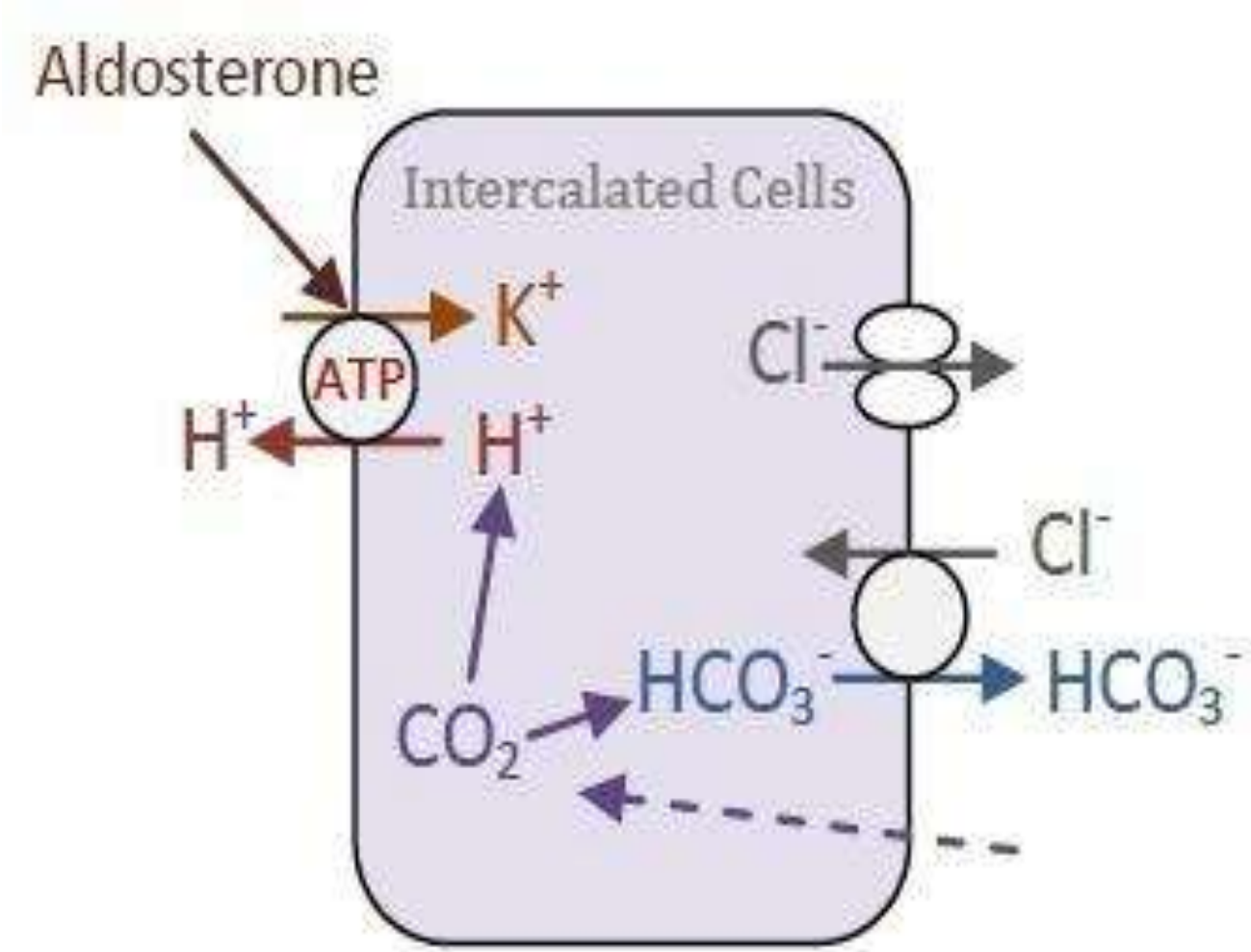
Type 2 (proximal) cont.

4. Nephrolithiasis and nephrocalcinosis do not occur (as they do in type 1 RTA)

5. Treatment: treat the underlying cause

a. Do not give bicarbonate to correct the acidosis because it will be excreted in the urine.
b. Sodium restriction increases sodium reabsorption (and thus bicarbonate reabsorption) in the proximal tubule


Collecting Tubule Principal Cell: K+ Secretion
Lumen
Blood
Na+ channel
Na
3 Na+
2 K+
Na+K+-ATPase
K+
K+ channel
K+ channel
K+
(+)
Aldosterone
B


Aldosterone
Intercalated Cells
K+
ATP
H+
H+
Cl-
Cl-
HCO3-
HCO3-
CO2

Type 4

1. This can result from any condition that is associated with hypoaldosteronism, or increased renal resistance to aldosterone.

2. It is common in patients with interstitial renal disease and diabetic nephropathy.

3. It is characterized by decreased Na+ absorption and decreased H+ and K+ secretion in the distal tubule.

4. Unlike other types of RTA, **type 4 results in hyperkalemia** and acidemia (although a non-anion gap metabolic acidosis still occurs).

5. Nephrolithiasis and nephrocalcinosis are rare.

Types

Type 1 (Distal)

• Inability to secrete H+
• Urine pH >6
• Nephrolithiasis and nephrocalcinosis **do** occur
* hypokalemia

Type2(Proximal)

• **inability to reabsorb HCO_3^-**
• **increased bicarbonate excretion**
• Nephrolithiasis and nephrocalcinosis do **not** occur
. hypokalemia

Type 4 (collecting tubule)

• Decreased Na+ absorption and H+ and K+ secretion in distal tubule
• Results in **hyperkalemia** and acidemia
• Nephrolithiasis and nephrocalcinosis are **rare**
. hyperkalemia

type 3 (Mixed)

• Rare autosomal recessive disorder : carbonic anhydrase II defciency

•Characteristcs of type I and II

ACUTE TUBULAR NECROSIS

by : thamer alshameri

Acute Tubular Necrosis

- Definition :

is a medical condition involve the death of tubular epithelial cells that form the renal tubules of kidneys/is a acute renal failure associated with reversible injury to tubular epithelium.

- This is the most common cause of RENAL-AKI 85% of the cases
- Usually recovers within 6 weeks

There are two types of ATN:

• Ischemic ATN : the most common cause of ATN & it may be due to decreased blood flow caused by :
severe hemorrhage , hypotension , dehydration,
shock , severe renal vasoconstriction and other procedures .

• Nephrotoxic ATN : caused by drugs (aminoglycosides/antifungal) , heavy metals , mushroom poisoning , organic solvents and radiographic contrast agents .

Risk Factors

-(antineoplastic)
-Surgery (Aortic surgery)
-Labor complications
- Sepsis (caused by infection)
-Amphotericin B
-Aminoglycosides (gentamycin)
-Shock
- Radiocontrast agents
-Jaundice
-Hypotension

ATN THREE phases:

- Initiation phase : characterized by acute decrease in GFR to very low level, with sudden increase in serum creatinine and BUN

- Maintenance phase: characterized by sustain decrease in GFR , with continue increase in creatinine and BUN (Oliguria : < 450 ml \ 24 hours OR Anuria : < 100 ml / 24 hours)

- Recovery phase : characterized by restore tubular function and gradual increase urine volume, with gradual decrease in creatinine and BUN

CLINICAL PRESENTATION OF ATN:

1-Early Oliguric phase

• Followed later on by Polyuric diuretic phase, due to loss of renal tubular medullary urine concentration function.

2-URAEMIC-Symptoms,

•Anorexia, Nausea, Vomiting, Hiccups, Pruritis, Drowzness, Muscle twitching

•Apathy, Confused, Fit, Coma.

3-Metabolic acidosis-Hyperkaelemia.

ATN-histopathology:

- Structural renal tubular cells damage .
- tubular cells effacement-flat-with necrosis.
- Prox. tubular obstruction
- Tubular dilatation
- Leucocytes infilteration

Diagnosis:

• BUN test: (essential)
mostly high BUN result from low blood flow to kidneys caused by e.g dehydration or heart failure
• Other investigations include :
- Physical examination + auscultation(lungs\ heart)
- kidneys biopsy
- urinalysis (find brown casts of epithelial cells in urine)
- urine sodium (kidneys can’t absorp Na+ and Na+ escape >40)
- Urine osmolarity Very Low due to death of tubules (can’t absorp water)

Treatment:

• There is no specific therapy for ATN to reverse the renal failure , when only the underlying cause must be corrected.

• Restricting fluid intake , taking medicine to control potassium level , taking medicine to remove fluids from body (Diuretics)

• Temporary dialysis can remove excess waste and fluids and improve symptoms and control kidney failure (dialysis necessary if potassium level is dangerously high) .

• Dialysis can be needed in the following cases : decrease mental status , fluid overload , increase potassium , decrease urination

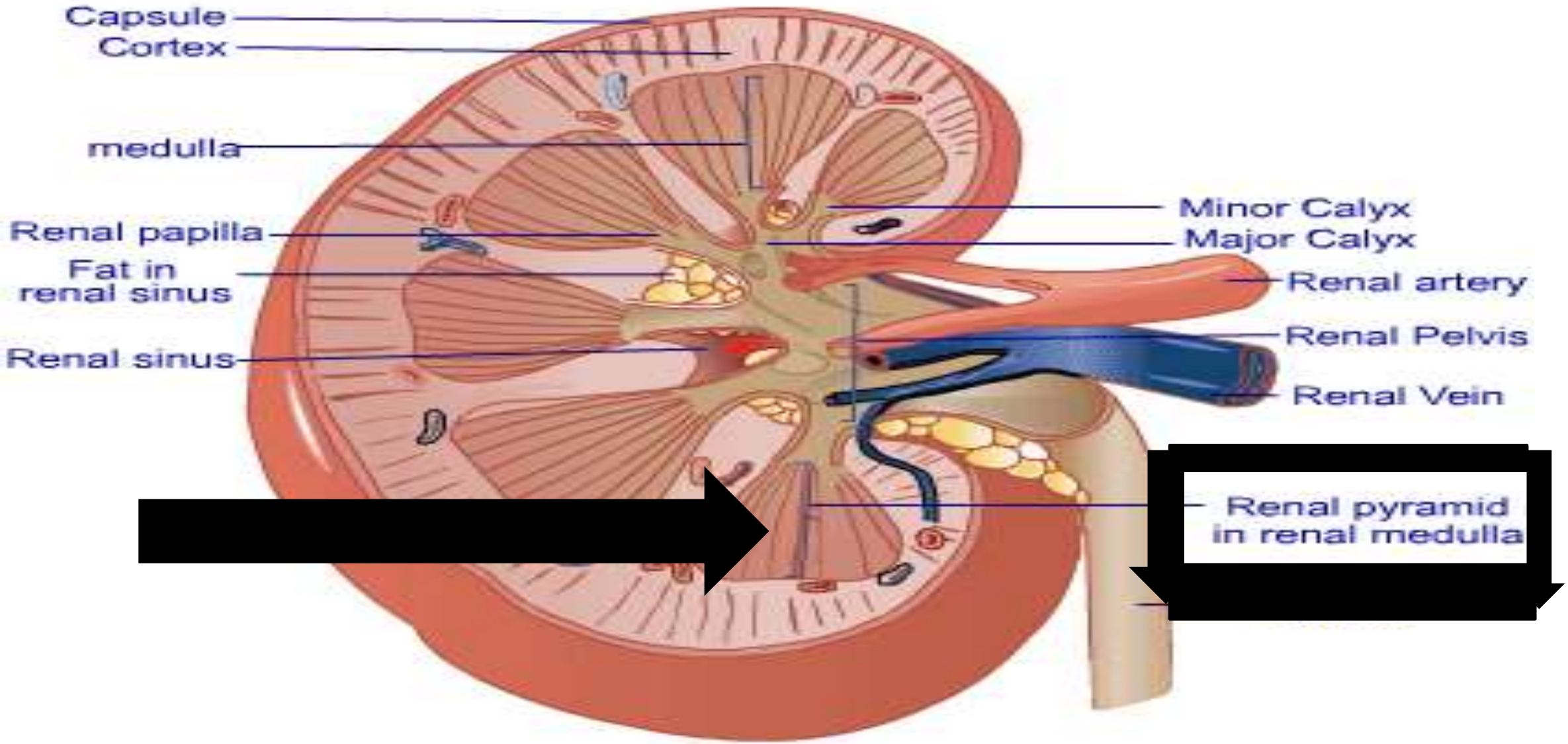
Prevention:

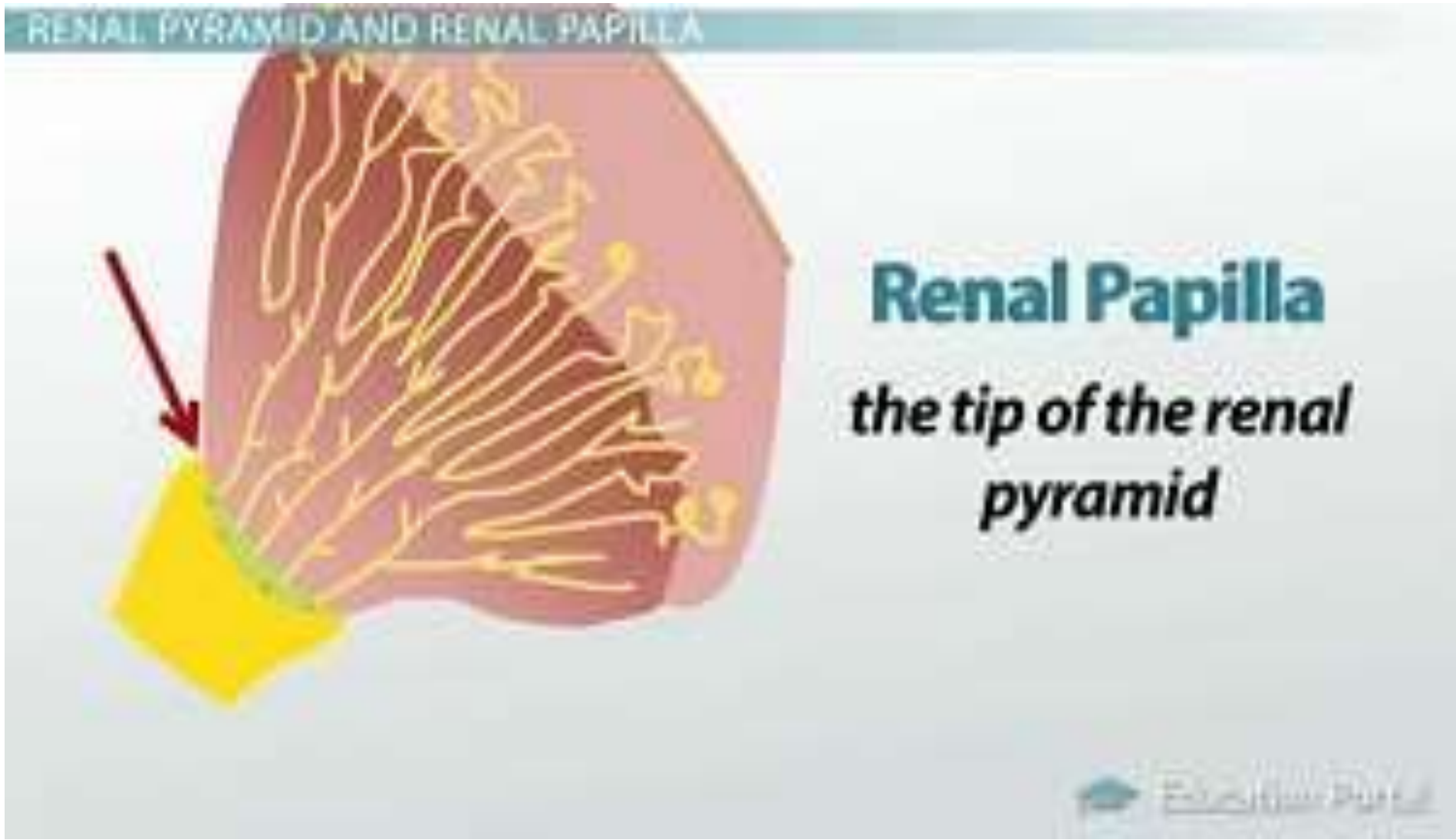
- Treat hypotension \ hypoperfusion for kidneys
- Ensure Cross matched blood transfusion
- Manage DM , liver disorders and heart problems .
- Drink a lot of water after having contrast dyes to allow them to be removed from body

Renal papillary necrosis

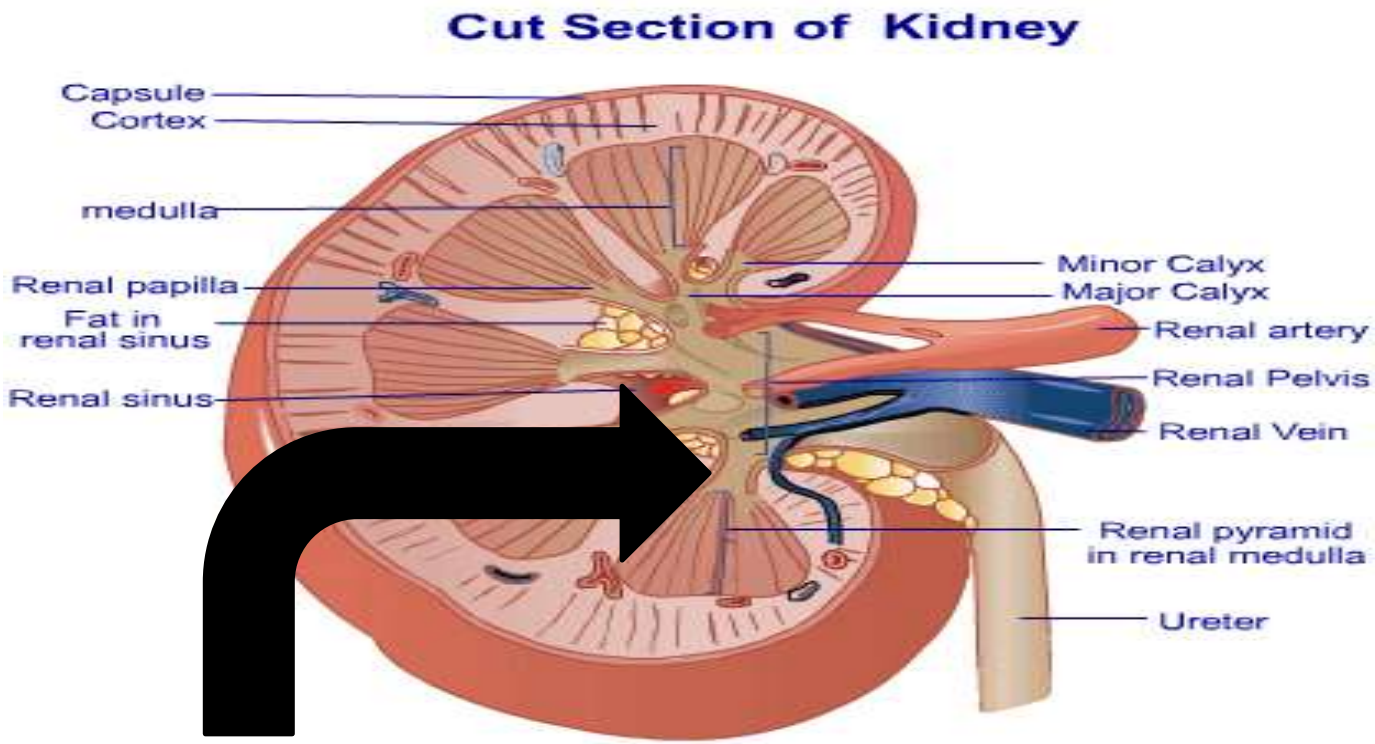
by : soud alsuwait

Cut Section of Kidney

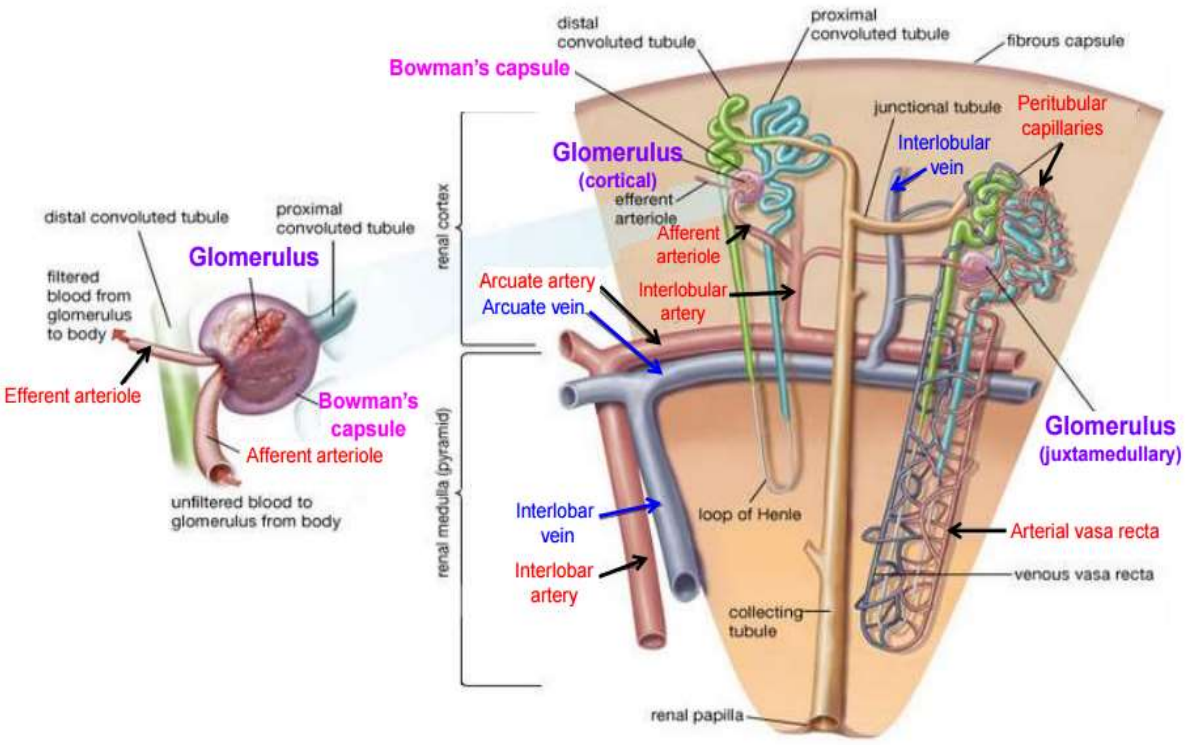


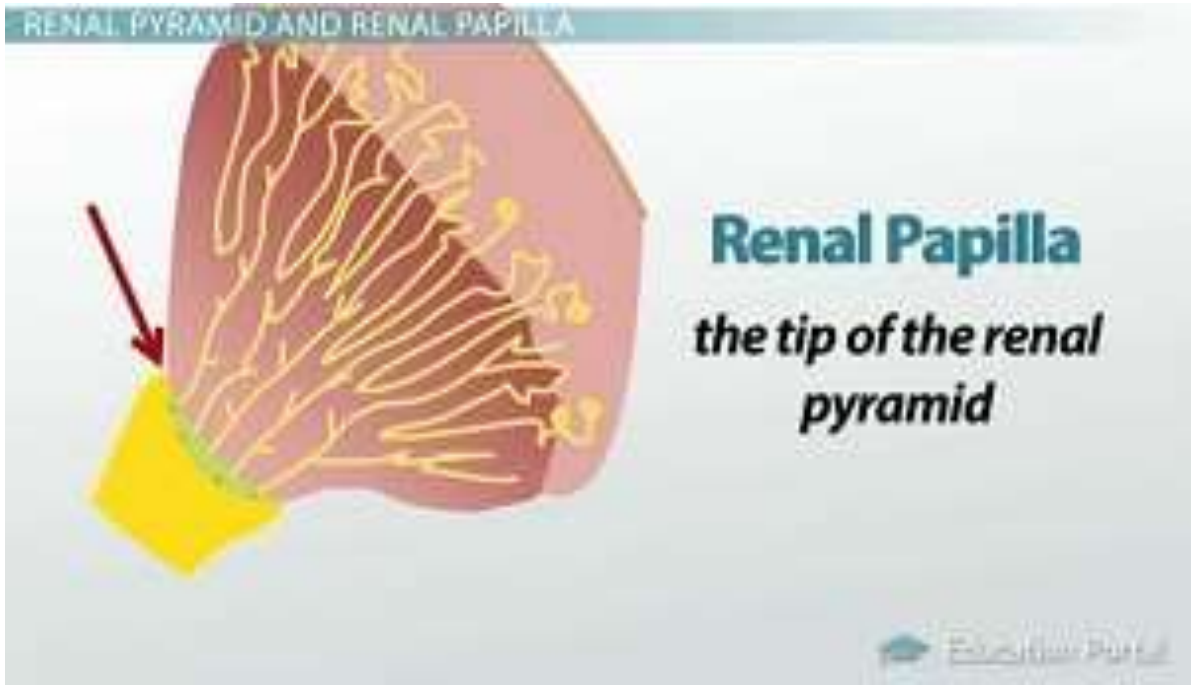

RENAL PYRAMID AND RENAL PAPILLA
Renal Papilla
the tip of the renal pyramid

The renal papillae are the areas where the openings of the collecting ducts enter the kidney and where urine flows into the ureters



The renal papillae lie within a ***hypertonic environment*** in the renal medulla, at the end of the vasa recta



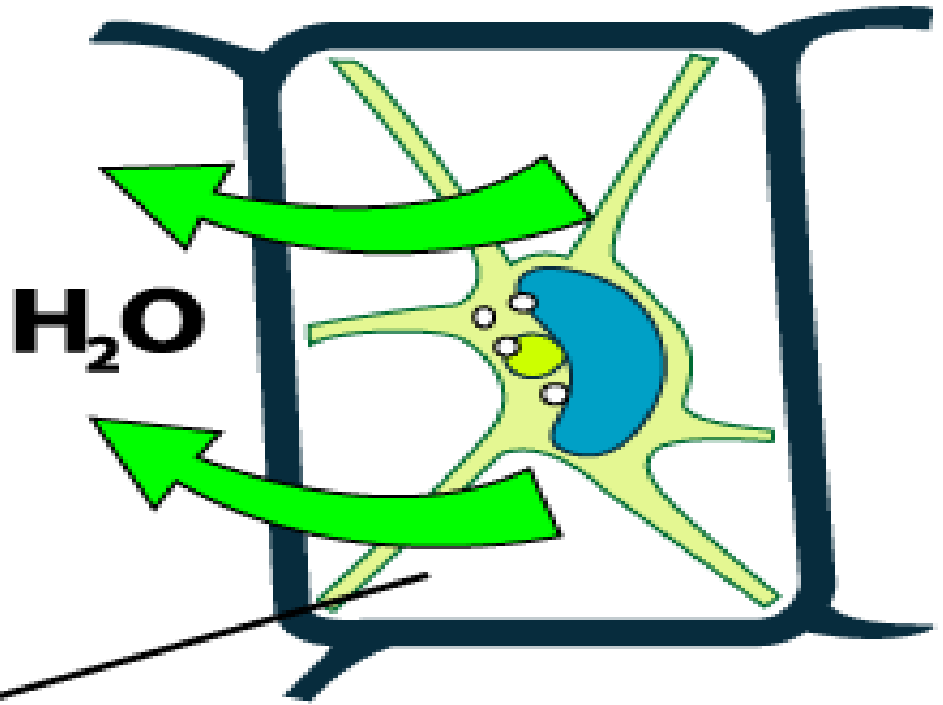


TO KNOW THIS IS VERY IMPORTANT

- ***Rapid review about hypertonic enviroment***
- -there is a higher concentration of dissolved
- salt or sugar outside of the cell
- H_20 will diffuse "out" from the cell and the cell will dehydrate and shrink and cellular metabolism will cease. This phenomenon is called **plasmolysis**

Hypertonic

Water is transported
out from the cell



Solute concentration
inside the cell is LOWER

Plasmolyzed

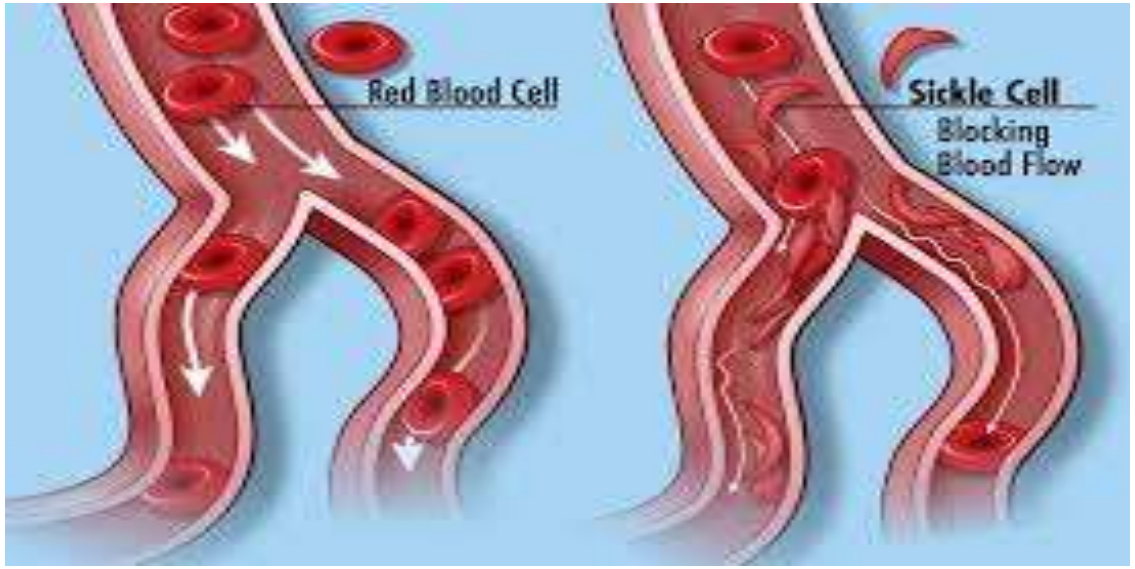
- ***So due that hypertonic envirnoment the renal***
- ***papilla are susceptible to ischaemic damage***
- and can undergo ***necrosis*** when their ***vascular supply is impaired***

So now we can determine some by logic causes

- **1)** Diabetes mellitius
- High serum of glucose leads to nonenzymatic glcosylation of the vascular basment membrane resulting in hyaline arteriolosclerosis
- ***DM cause injury to the small vessels in the –body -> impaired blood supply-> ischemia-> necrosis**

So now we can determine some by logic causes

- 2)Sickle cell disease
- As we see in th pic due
- The sickle cell shape
- There will be a blocking
- To the blood flow



- -> microvascular occlusions -> infarcts
- -> impaired flow-> ischemia->necrosis

So now we can determine some by logic causes

- **3)Analgesic nephropathy:**
- long-term ingestion of NSAID.
- Eg. Aspirin
- -we know that NSAID are prostaglandin
- Inhibitors so the they will induce
- “vasoconstriction” →(on long term use)
- Also will lead to impaired blood flow
- Ischemia-> necrosis

Other causes

- 4)pyelonephritis
- is a common suppurative inflammation of the kidney and renal pelvis caused by bacterial infection , Characterized by renal abscess formation within the renal parenchyma

  If the pus unable to drainage maybe because obstruction and reach the renal papillae will cause papillary necrosis
- but it is difficult to determine whether this is cause of papillary necrosis or a complication

Other causes

- 5) Cirrhosis - destruction of liver cells that can also indirectly damage kidney cells
- 6) Renal vein thrombosis - a blood clot in the veins of the kidney that can impair blood flow
- 7) Systemic vasculitis - an infection of blood vessels, specifically those in the kidneys
- 8) Tuberculosis - an infection to the lungs that can also impact kidney cells
- 9) Obstruction - a blockage in the blood vessels leading to the kidneys

RPN-renal papillary necrosis

- Typically affects adults (>60 years)

- **The clinical presentation is variable.**

Some patients are asymptomatic and clinically silent, whereas others present with renal colic and renal impairment as necrosed papillae slough off and cause ureteric obstruction.

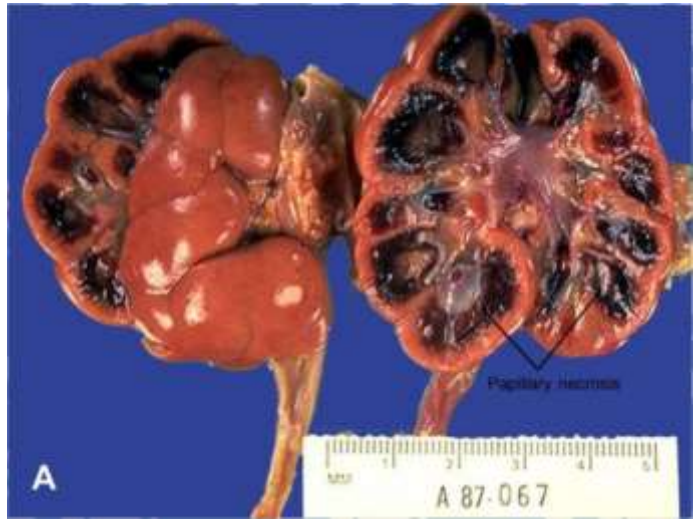
Con.t

-Urinalysis may be normal but, more frequently haematuria and sterile pyuria are present. Significant proteinuria is unusual, unless there is

renal failure.

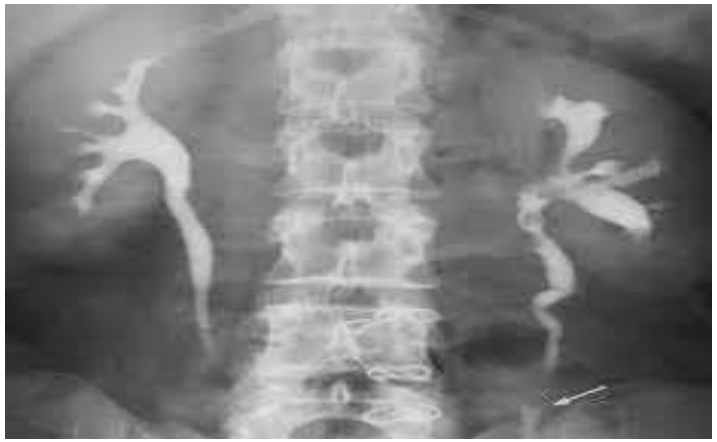
- Bilateral in 70% of cases

- Gross A : necrosis of renal papillae/medullary portion



- Histology B :
-
- Because the necrosis is induced by ischemia, it typically has a coagulative appearance (ghost cells).

- The imaging method of choice to make the diagnosis is ***pyelography***
- -Pyelography involves direct injection of contrast medium into the collecting system



Management and prevention

- -Management is based on relieving obstruction, where present, and withdrawal of the offending drugs.
- - Controlling diabetes or sickle cell anemia may reduce your risk. To prevent renal papillary necrosis from analgesic nephropathy, follow your provider's instructions when using medicines, Do not take more than the recommended dose without asking your provider