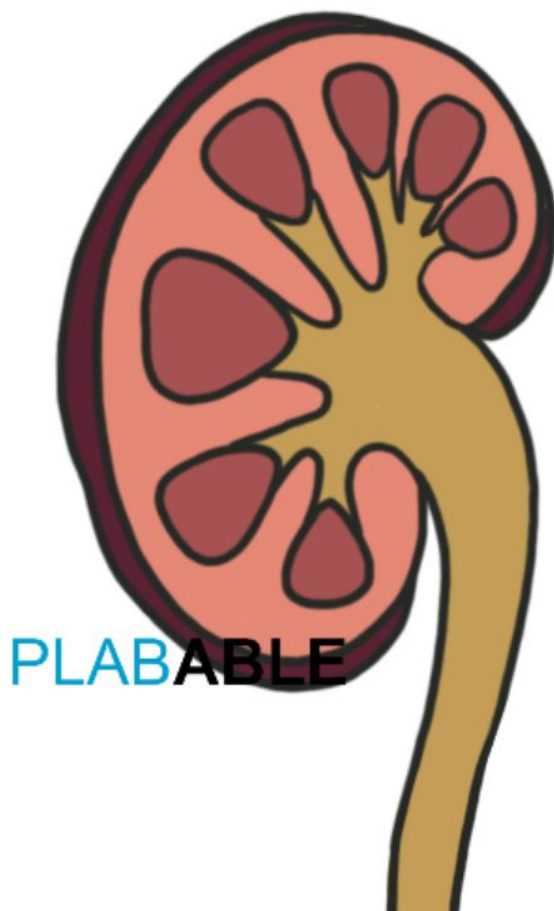


# PLABABLE

GEMS 

VERSION 3.0

## Nephrology





# Acute Kidney Injury

Definition:

Acutely raised creatinine with reduced urine output

Increase in serum creatinine of  $\geq 26.5 \mu\text{mol/L}$  from baseline within 48 hours

or

Increase in serum creatinine of  $\geq 50\%$  from baseline within 7 days

or

A reduction in urine volume below  $0.5\text{ml/kg/hr}$  for 6 hours



# Chronic Kidney Disease

Definition:

Chronically reduce eGFR and/or proteinuria

Persistent reduction in renal function (eGFR is less than 60 mL/min/1.73 m<sup>2</sup>) and/or proteinuria (urinary ACR is greater than 3 mg/mmol) lasting for at least three months.



# Goodpasture Syndrome

## Facts

It is a combination of:

- Rapidly progressive glomerulonephritis
- Pulmonary alveolar haemorrhage

It is an autoimmune disease

## Features

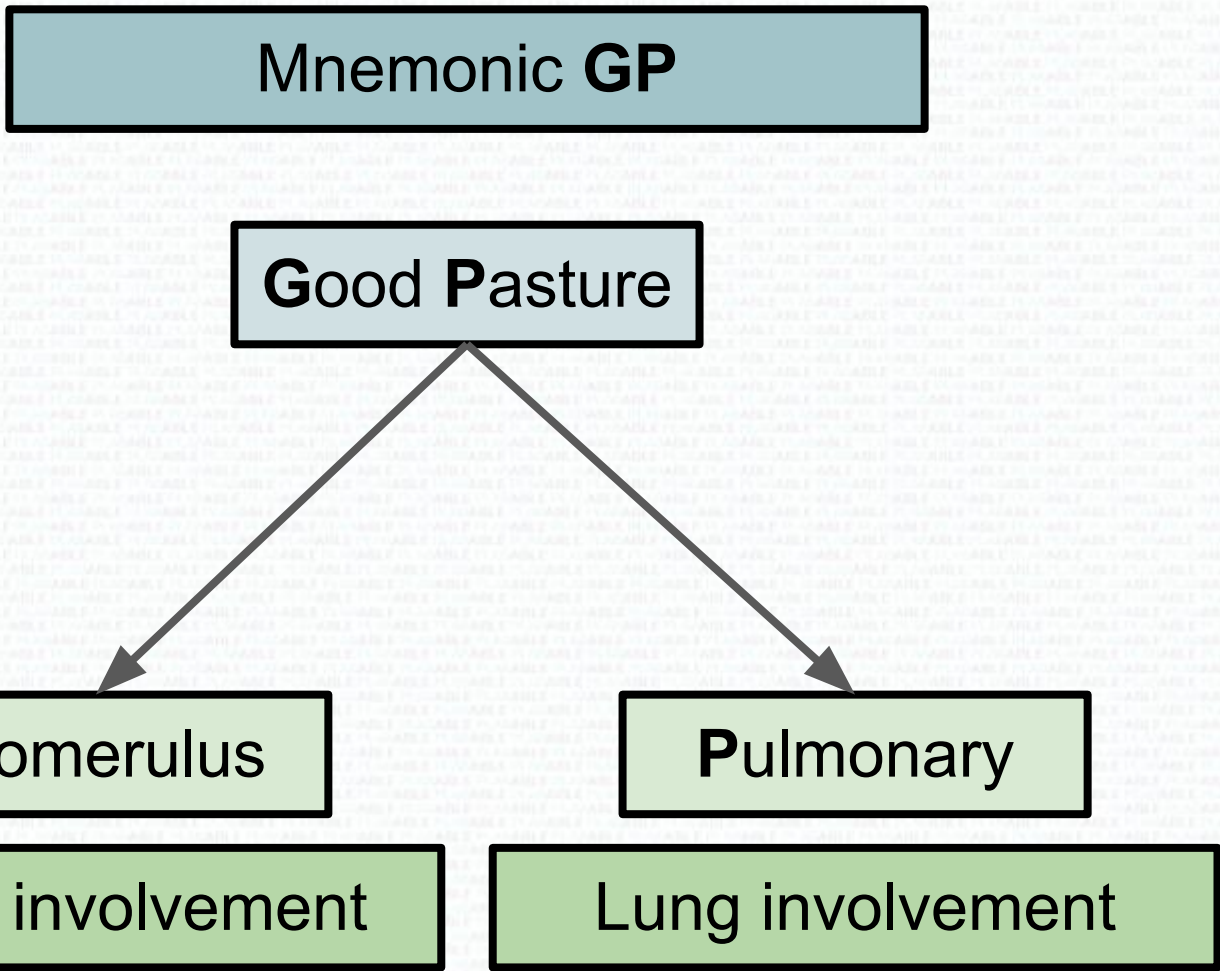
- Haematuria
- Hemoptysis
- Impaired renal function test

## Investigations

1. Blood test **Most initial**  
→ Anti-glomerular basement membrane antibodies (anti-GBM antibodies)
2. Kidney biopsy **Most definitive**  
→ Shows crescentic glomerulonephritis
3. Chest X-ray / CT scan  
→ Shows patchy interstitial infiltration (intra-pulmonary bleeding)
4. Lung biopsy there are any lung involvement **Most definitive**



# Goodpasture Syndrome





# Goodpasture Syndrome & Differential Diagnosis

*These are the differentials for pulmonary renal syndrome (AKI with pulmonary involvement)*

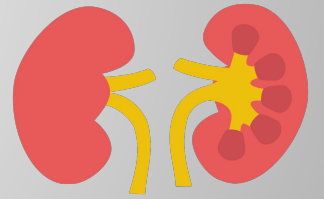
Condition	Features	Test
Alport syndrome	<ul style="list-style-type: none"><li>● Haemaproteinuria</li><li>● Haemoptysis</li><li>● Abnormal U&amp;E</li><li>● Loss of sight</li><li>● Loss of hearing</li></ul>	
Churg Strauss (Eosinophilic granulomatosis with polyangiitis)	<ul style="list-style-type: none"><li>● Asthma</li><li>● Eosinophilia</li></ul>	p-ANCA
Wegener’s granulomatosis (Granulomatosis with polyangiitis)	<ul style="list-style-type: none"><li>● Haematuria</li><li>● Nasal septum perforation</li><li>● Epistaxis</li></ul>	c-ANCA



# Itching

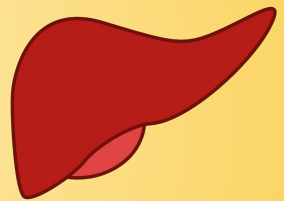
## Chronic Renal Failure

- Itching → ↑ Serum urea and/or ↑ serum phosphate
- Tiredness → ↓ Erythropoietin → Anaemia
- Peripheral oedema
- Hyperpigmentation



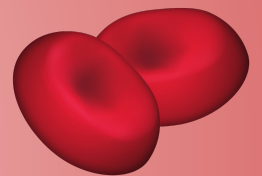
## Liver Failure

- Itching → ↑ Serum bilirubin
- Ascites
- Jaundice
- Bleeding → ↓ Clotting factor production



## Polycythemia rubra vera

- Itching
- Red skin → ↑ Haemoglobin
- Splenomegaly
- Burning sensation in fingers and toes
- Gout
- Stroke



## Scabies

- Itching
- Line tracks on skin (burrows)



# Rhabdomyolysis

## Facts

It is a condition of **dying of skeletal muscles** and as a result releasing:

- **Myoglobin**
- **Potassium**
- **Creatine kinase**

## Common scenarios

- Trapped for several hours under heavy object
- Fall followed by lying for long period of time on floor
- An elderly with frequent fall after acute kidney injury
- IV drug user lying on floor not moving for long
- Severe exertion or dehydration e.g. marathon
- Severe crash injury

## Features

- Myoglobinuria (*dipstick would pick up blood*)
- Hyperkalaemia
- AKI - Acute tubular injury
- ↑ Creatine kinase *Usually more than 2000 U/L*

## Summary

1. Prolonged immobilisation
  - Muscle ischaemia
  - Rhabdomyolysis
2. Myoglobin
  - Red colour due to haem



# Rhabdomyolysis

## Brain trainer:

An elderly man is found on the floor of his house. It is uncertain the duration he has been unconscious on the floor. On fluid resuscitation, he gains consciousness. His bloods show:

Creatine kinase 3523 U/L (25-195)

Serum urea 25 mmol/L (2.0-7)

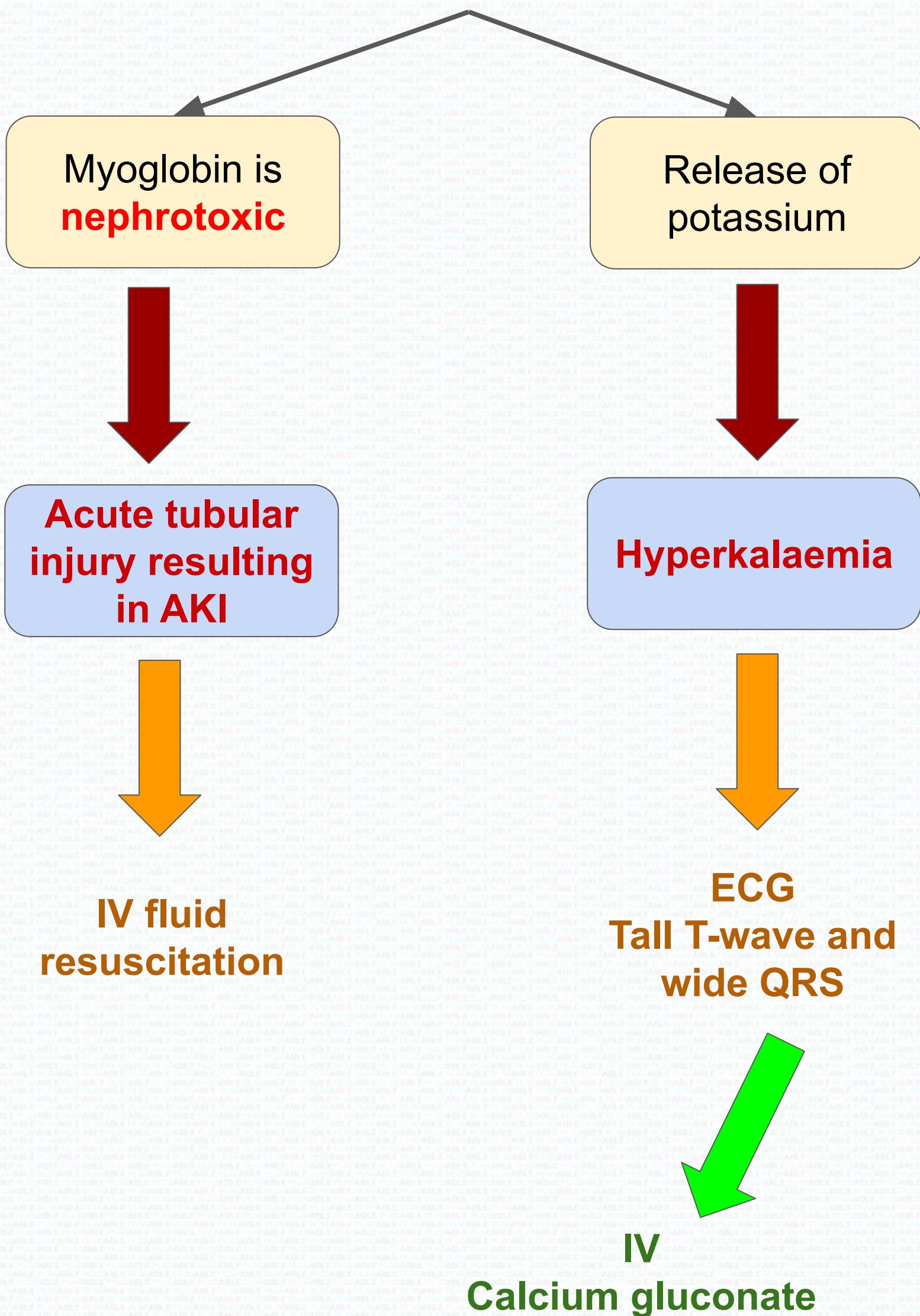
Serum creatinine 297  $\mu$ mol/L (70-150)

What is the most likely reason for the elevated creatine kinase?

→ Rhabdomyolysis



# Rhabdomyolysis Management





# Acute Tubular Necrosis

## Causes

- Massive haemorrhage  
→ Hypotensive shock
- Prolonged renal ischaemia  
→ Reduced renal perfusion  
→ Tubular necrosis

## Management

- Maintain hydration and perfusion
- Fluid balance management



Balance input and output to avoid fluid overload

Aim to avoid being in a negative fluid balance



# Interstitial Nephritis

## Features:

Acute kidney injury in a euvolaemic patient

- Allergy reaction
  - Drug as most common cause
  - Common drugs:
    - Penicillins
    - Proton pump inhibitors
- Mild eosinophilia
- Urine dipstick - bland

Means no protein or blood

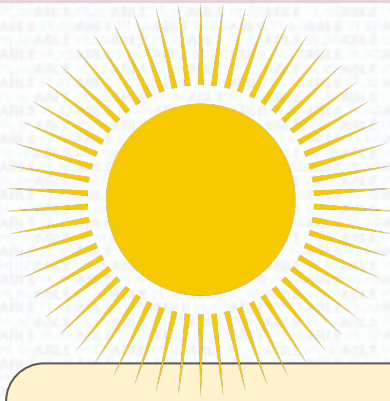
Definitive diagnosis is done from a renal biopsy

This will show tubular inflammation with lymphocytes and eosinophils

Treatment: Oral Prednisolone



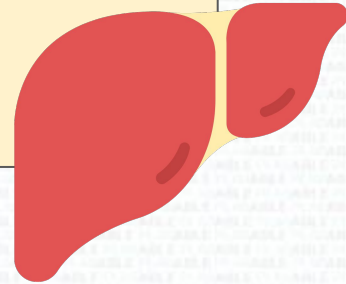
# Vitamin D Deficiency in CKD



Sunlight and Skin

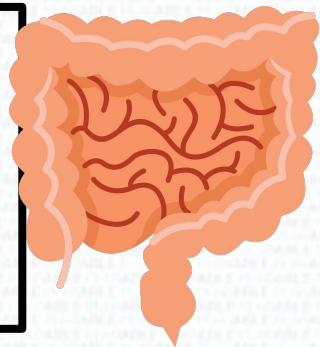


**1st Hydroxylation in liver**  
25-Hydroxycholecalciferol



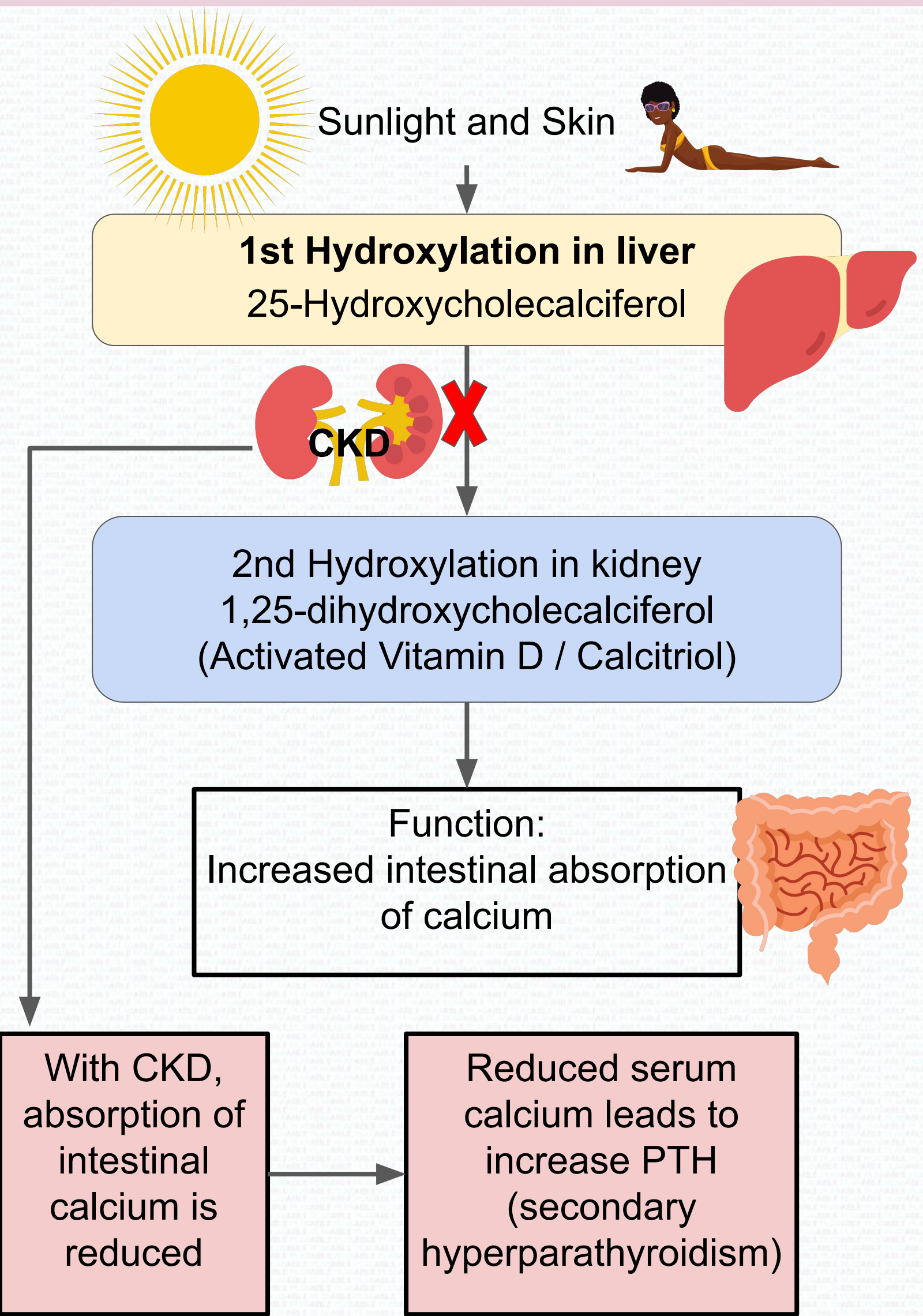
**2nd Hydroxylation in kidney**  
1,25-dihydroxycholecalciferol  
(Activated Vitamin D / Calcitriol)

**Function:**  
Increased intestinal absorption  
of calcium





# Vitamin D Deficiency in CKD





# Pyelonephritis

## Contributing factors

Pregnancy, stone, vesico-ureteric reflux, diabetes

## Symptoms

- Dysuria
- Frequency
- Urgency
- Lower abdominal pain
- Fever
- Rigors
- Loin or back pain

*(Costovertebral angle tenderness known as renal angle tenderness)*

Lower UTI

Pyelonephritis

## ACUTE

Sudden development:

- Fever
- Rigors
- Loin pain

## CHRONIC

- Hypertension
- Repeated UTI  
→ Renal scarring
- No active infection



# Management Of Pyelonephritis

## Investigation

- **Urinalysis** shows blood, protein, nitrite, leukocyte esterase
- **Urine culture and sensitivity** ideally **before** commencing antibiotics

In acute pyelonephritis:  
**Start empirical antibiotics immediately** once sample has been sent.

***\*E.Coli = Most common cause of UTI\****



# Management Of Pyelonephritis

We have decided to just give you 2 antibiotics to remember which we believe is enough for the exam

Co-amoxiclav

Cefalexin

Generally a 7 day course



# Proteinuria

## Facts

Protein 1+ and above in dipstick test

- Can be normal (5% in healthy individuals)
- Can occur in relation to fever, post-exercise, seizure, CCF, severe acute illness, extreme cold



**If no symptoms and healthy**

→ Repeat the test



**If still high**



→ 24hrs urine collection

→ urinary albumin creatinine ratio  
/ protein creatinine ratio

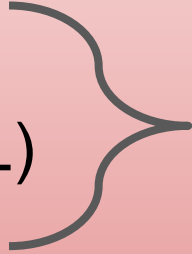
**DO NOT** refer to renal clinic because of a ONE  
'isolated positive dipstick'

Further investigation before a referral is always  
required!



# Nephrotic Syndrome

## Features

1. Proteinuria ( $> 3\text{g}/24\text{hr}$ )
  2. Hypoalbuminemia ( $< 30\text{g/L}$ )
  3. Oedema
  4. Hyperlipidaemia
  5. Hypercoagulable state - loss of antithrombin III
  6. Predisposition to infection - loss of immunoglobulins
- 
- Defines nephrotic syndrome

## Investigations

1. Urinary protein:creatinine ratio / 24 hr urinary protein levels
  2. Blood test - Albumin level
  3. Renal biopsy - definitive diagnostic test
- \*Foamy or frothy urine = High protein\*

## Complications

- Thromboembolism
- Sepsis
- Pre-renal AKI (usually from over-diuresis)



# Common Cause Of Nephrotic Syndrome

## In Children

Most common

→ **Minimal change nephropathy**

## In Adults

Most common

→ **Membranous glomerulonephritis**

Second most common

→ **Focal segmental glomerulonephritis**

## Others

- Diabetic nephropathy
- Myeloma cast nephropathy
  - Amyloidosis

Most common cause for nephrotic syndrome in adult > 40 years old is **membranous glomerulonephritis**



# Minimal Change Nephropathy

*Minimal change diseases always account for nephrotic syndrome*

## Features

- Common in children
- Nephrotic syndrome
- Normotensive
- Renal biopsy

Proteinuria > 3g/day  
Oedema  
Hypoalbuminaemia

→ Electron microscopy shows **fusion of podocytes**

*We do not really do this for children as it is invasive, we just treat with steroids*

## Example

**6 years old boy** presented with progressively:

- **Swelling** of face, scrotum and legs
- Urine is **frothy**
- **Fusion of podocytes** shows on electron microscopy

**RED** are all the hints!



# Minimal Change Disease

## Brain trainer:

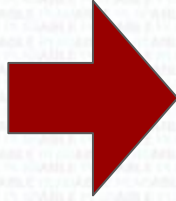
A boy presents with serum albumin 20 g/L (35-50), oedema and urine dipstick positive for protein. What should you check before referring this boy to nephrology?

➔ **Urine protein:creatinine ratio**

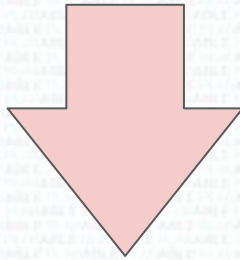


# Hypovolaemia

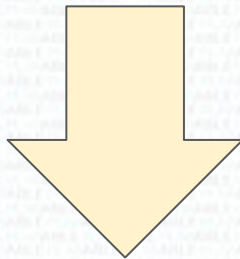
Vomiting  
+  
Diarrhoea



Dehydration  
+  
Hypokalaemia  
(↓K)



**Acute kidney injury (AKI)**



Significantly raised serum creatinine



Kidney unable to excrete potassium,  
creatinine and urea



**Hyperkalaemia**  
+

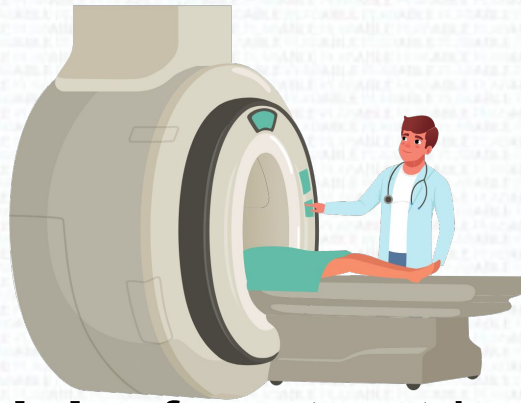
↑ **Serum urea**



↑ **Creatinine**



# Contrast Induced Nephropathy



To **reduce risk** of contrast in CT scan  
or any contrast study

Drink plenty of  
fluid

Stop  
nephrotoxic  
drugs if renal  
function  
abnormal  
e.g. Metformin,  
NSAIDs

IV fluid with normal saline  
(0.9% NaCl)  
*(Probably the same efficacy  
as just drinking oral fluids)*

- Pre- and post-contrast
- Particularly in high risk patient
- e.g. Elderly with diabetes, CKD stage 4 and above, CCF



# Blood Gas Abnormality

Acidaemic

**pH < 7.35**

1

Alkalaemia

**pH > 7.45**

2

Respiratory acidosis  
**PaCO<sub>2</sub> > 6.0 kPa**

*(Respiratory  
compensation for  
metabolic alkalosis)*

Respiratory alkalosis  
**PaCO<sub>2</sub> < 4.7 kPa**

*(Respiratory  
compensation for  
metabolic acidosis)*

3

Metabolic acidosis  
**Bicarbonate  
< 22 mmol/L**

*(Renal compensation  
for respiratory alkalosis)*

Metabolic alkalosis  
**Bicarbonate  
> 26 mmol/L**

*(Renal compensation  
for respiratory acidosis)*

**CO<sub>2</sub> is an acid. Bicarbonate is an alkali**



# Hypertension In Chronic Kidney Disease

Patient has both **CKD** and **HTN**

Is the patient also diabetic?  
(Age/ethnicity doesn't matter)

**Non-Diabetic**

**Diabetic**

**START AN **ACEI OR ARB** IF  
eGFR > 30 OR**

**ACR > 30**

**ACR  $\geq$  3**



**Consider the following situations:**

**If eGFR is < 30**

- Use ACEI/ARB **with caution** (lower dose)

**If ACR is <30 or <3**

- This is **NOT** a contra-indication for ACE/ARB use
- Therefore continue use of drug if there are other indications



# Types Of Glomerulonephritis

<p><b>Presented with nephritic syndrome</b></p> <ul style="list-style-type: none"><li>● <b>Haematuria</b></li><li>● <b>Hypertension</b></li></ul>	<p><b>Presented with nephrotic syndrome</b></p> <ul style="list-style-type: none"><li>● <b>Proteinuria</b></li><li>● <b>Oedema</b></li></ul>
<p><b>Rapid progressive glomerulonephritis (Crescentic glomerulonephritis)</b></p> <ul style="list-style-type: none"><li>● <b>Rapid onset</b></li><li>● <b>Often presented as AKI</b></li><li>● <b>Caused by Goodpasture → Haematuria → Hemoptysis Or ANCA +ve vasculitis</b></li></ul>	<p><b>Minimal change diseases</b></p> <ul style="list-style-type: none"><li>● <b>Accounted for 80% of nephrotic syndrome</b></li><li>● <b>Good response to steroid</b></li><li>● <b>Fusion of podocytes show in electron microscopy via renal biopsy</b></li><li>● <b>Mostly idiopathic or Hodgkin's or steroid induced</b></li></ul>



# Types of Glomerulonephritis

## Presented with nephritic syndrome

- Haematuria
- Hypertension

## Presented with nephrotic syndrome

- Proteinuria
- Oedema

## IgA nephropathy (Berger's diseases)

- Young adult with haematuria 1-2 days after URTI

## Membranous glomerulonephritis

- Presentation:
  - Proteinuria
  - Nephrotic syndrome
  - CKD
- Idiopathic or caused by infection, rheumatoid drugs or malignancy
- $\frac{1}{3}$  resolved
- $\frac{1}{3}$  response to cytotoxics
- $\frac{1}{3}$  develops to CKD



# Types of Glomerulonephritis

<p><b>Presented with nephritic syndrome</b></p> <ul style="list-style-type: none"><li>● <b>Haematuria</b></li><li>● <b>Hypertension</b></li></ul>	<p><b>Presented with nephrotic syndrome</b></p> <ul style="list-style-type: none"><li>● <b>Proteinuria</b></li><li>● <b>Oedema</b></li></ul>
<p><b>Mesangioproliferative glomerulonephritis</b></p> <ul style="list-style-type: none"><li>● Young adult with haematuria 1-2 days after URTI</li></ul>	<p><b>Focal segmental glomerulosclerosis</b></p> <ul style="list-style-type: none"><li>● Idiopathic or secondary to HIV/heroin</li><li>● Presentation:<ul style="list-style-type: none"><li>→ Proteinuria</li><li>→ Nephrotic syndrome</li><li>→ CKD</li></ul></li></ul>



# When To Suspect Chronic Kidney Disease?

**Anaemia**

**Hypocalcaemia**

**Hyperphosphatemia**

**Small Kidney on  
ultrasound < 9 cm**



# Diabetes and Chronic Kidney Disease

## Brain trainer:

An elderly man with a background of hypertension, diabetes mellitus type 2 and chronic kidney disease attends the GP surgery for a check up. His BP is 180/100 mmHg. He is taking regular amlodipine. There is significant proteinuria on two separate occasions. Which medication is likely to slow down the renal disease process?

→ **ACE inhibitor**



# Hypocalcaemia

## Symptoms

- Tingling
- Numbness
- Paresthesia
- Involuntary spasm/cramps

CKD can cause

1,25 dihydroxyvitamin D3 deficiency

→ ↓ Calcium absorption

→ Hypocalcemia



# Autosomal Dominant Polycystic Kidney Diseases

## Features

- Haematuria
- Hypertension
- Loin or flank pain

Often associated with intracranial aneurysm

## Investigation

- Ultrasound kidney, ureters and bladders

**Genetic testing** → Best not to select in the exam as there are very specific criteria for its use in specialist centres

**Autosomal dominant**

= 50% of offspring (1st generation) will be affected

**Can lead to progressive CKD**



# Haemolytic Uremic Syndrome (HUS)

## In children

1. Eating undercooked contaminated food
2. E.Coli → Produce verotoxin
3. Profuse **diarrhoea**
4. **Bloody** diarrhoea
5. After 2-14 days  
→ **Uremia (Acute renal failure)**
6. + Features of anaemia e.g fatigue, pallor

## Features

1. Haemolytic anaemia (Haemolysis)
2. Uremia (Acute renal failure)  
→ Haematuria  
→ Proteinuria  
→ ↑ Urea and creatinine
3. Thrombocytopenia (Low platelets)

## Treatment

- IV Fluid
- ± Blood transfusion
- ± Dialysis
- Plasma exchange (in very severe case only)



# Haemolytic Uremic Syndrome (HUS)

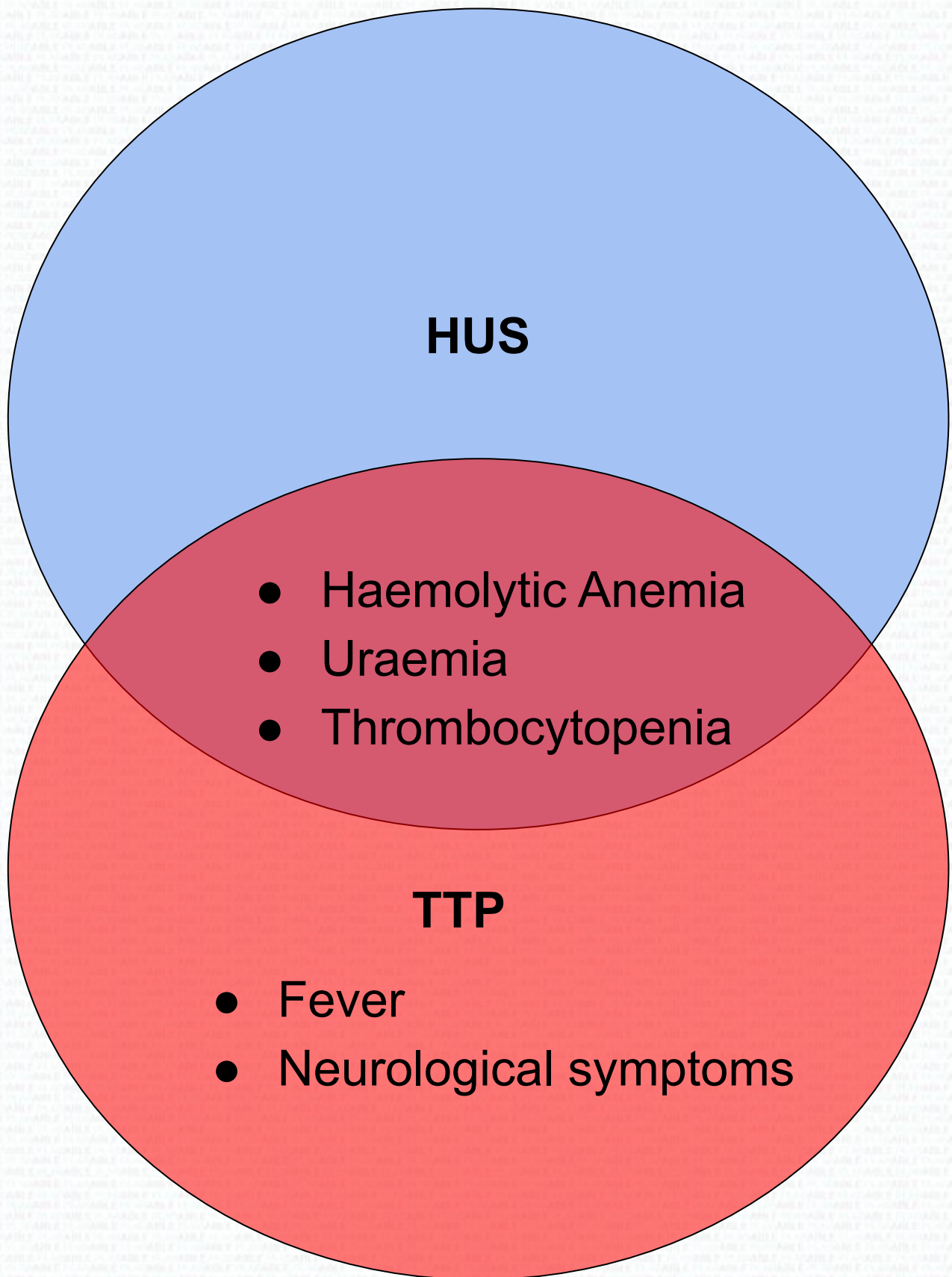
**NEVER** give antibiotics as it releases more toxin by killing *E.Coli*

Suspect **thrombotic thrombocytopenic purpura** if they have features of HUS **plus fever and neurological manifestation**

<i>Diseases</i>	<i>Features</i>	<i>Investigation</i>
<b>Polycystic Kidney Diseases (ADPKD)</b>	<ul style="list-style-type: none"><li>● Haematuria</li><li>● Hypertension</li></ul>	Ultrasound
<b>Goodpasture Syndrome</b>	<ul style="list-style-type: none"><li>● Haematuria</li><li>● Hemoptysis</li></ul>	Anti-GBM antibodies
<b>Wegener’s (Granulomatosis with Polyangiitis)</b>	<ul style="list-style-type: none"><li>● Haematuria</li><li>● Hemoptysis</li><li>● Nasal/Sinus symptoms</li></ul>	c-ANCA
<b>Haemolytic Uremic Syndrome (HUS)</b>	<ul style="list-style-type: none"><li>● Haematuria</li><li>● Bloody Diarrhoea</li></ul>	



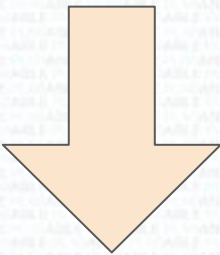
# Haemolytic Uremic Syndrome (HUS)



Suspect **thrombotic thrombocytopenic purpura** if they have features of HUS plus **fever** and **neurological symptoms**



# Haematuria After A Upper Respiratory Tract Infection



IgA glomerulonephritis	Post-streptococcal glomerulonephritis
1-2 <b>days</b> after URTI	1-2 <b>weeks</b> after URTI
Main Features: <ul style="list-style-type: none"><li>● Haematuria</li></ul>	Main Features: <ul style="list-style-type: none"><li>● Proteinuria</li></ul>
Patient factor: <ul style="list-style-type: none"><li>● Young males</li></ul>	Patient factor: <ul style="list-style-type: none"><li>● ↓ Complement (C3) Level</li><li>● Humps on electron microscopy in renal biopsy</li></ul>
Causative organism → Group A beta-haemolytic Streptococci ( <i>Streptococcus Pyogenes</i> )  Treatment → Mainly supporting as it is self-limiting	



# Abnormal Kidney Size Causes

## Large Kidney

- ADPKD  
→ Multiple cysts makes kidney bigger
- Obstructive uropathy  
→ Due to stone or enlarged prostate  
→ Hydronephrosis  
→ ↑ Size

## Small Kidney

- Hypertensive renal diseases  
→ Small and scarred kidney
- Bilateral renal artery stenosis
- Chronic pyelonephritis
- Chronic glomerulonephritis



# Small Kidneys

While there are many causes of small kidneys. We would like you think of these few which are commonly tested.

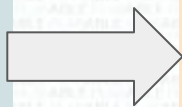
## Findings

Hypertensive renal diseases



Long term high blood pressure

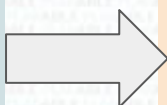
Bilateral renal artery stenosis



ACEi often worsen creatinine acutely

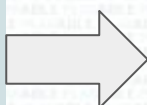
Remember, unilateral renal artery stenosis results in only ONE kidney being small

Chronic pyelonephritis



Recurrent upper UTIs  
Scarring of renal parenchyma

Chronic glomerulonephritis



Look out for the absence of the other diagnostic options above + proteinuria



# Abnormal Kidney Size Causes

## Examples:

Patient with hypertension with elevated creatinine, u/s shows bilateral small kidneys

→ Hypertensive renal diseases

Patient with enlarged prostate presented with prostatism (urinary frequency, post-void dripping, hesitancy)

→ Obstructive uropathy + Hydronephrosis

→ Ultrasound will show LARGE kidneys

Patient with dilated pelvicalyceal system and is young

→ Reflux nephropathy



# Haemodialysis

## Indications:

Persistent high potassium	Acidosis
Pulmonary oedema, pericarditis	Fluid overload with anuria

## Example:

Patient had renal transplant and presented with above features?

Causes:

**Transplant rejection or Host-versus-graft:**

- ✗ Treat hyperkalemia
- ✗ Give IV calcium gluconate followed by insulin and glucose

∴ These **DO NOT** treat all the above features as kidney does not work, potassium and toxin will accumulate again.  
Extra fluid can worsen fluid overload problem

**BEST treatment: Haemodialysis**



# Pseudohypoparathyroidism

Remember the levels!

- PTH raised
- Calcium levels low/normal
- Phosphate raised

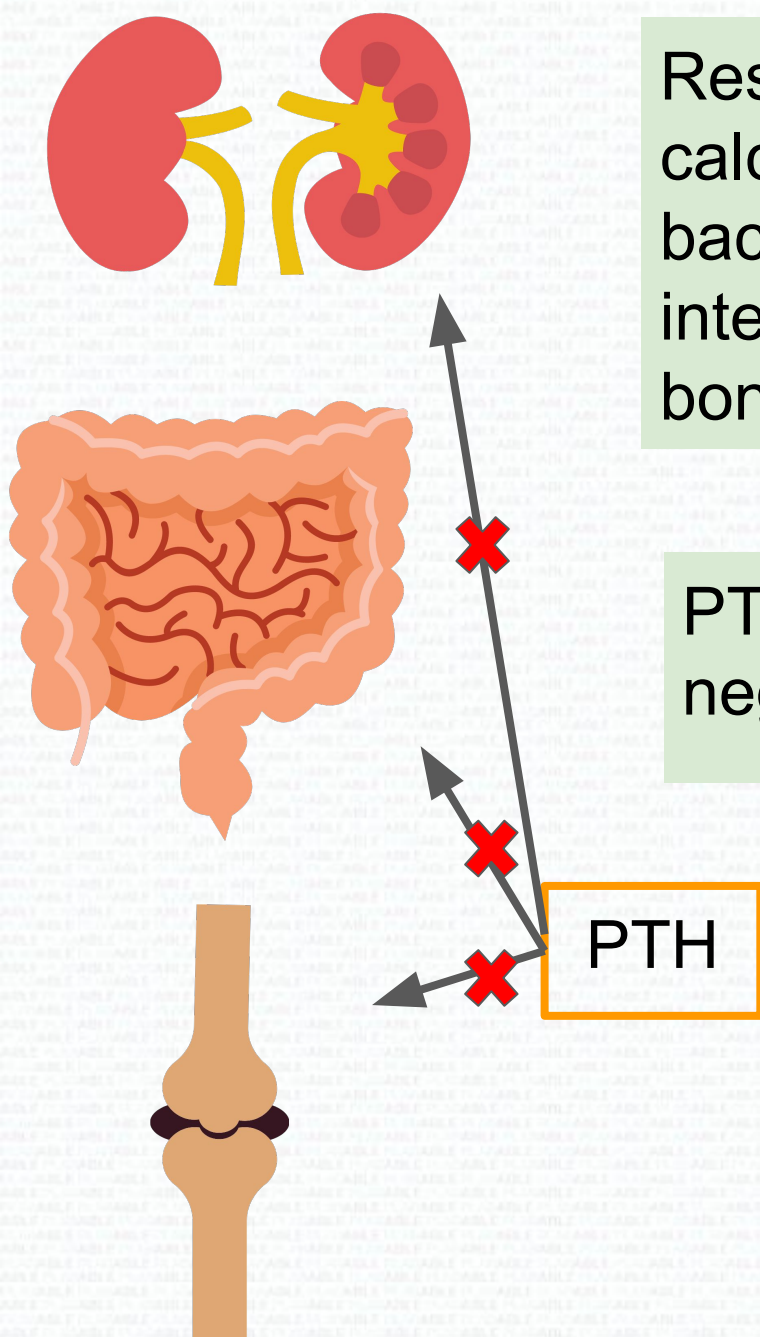
Pseudohypoparathyroidism  
= **PTH resistance** in end organ (*e.g. bone and kidneys*)



Results in not enough calcium being absorbed back into serum from intestine, kidney and bone



PTH rises because of negative feedback





# Adynamic Bone Disease

Disease due to low bone turnover

- Oversuppression of PTH (e.g. overreplacement of calcium and vitamin D)

Remember the levels

- In context of end stage renal disease
  - Normal PTH
  - Normal or low calcium levels
  - Normal or raised phosphate
  - Raised ALP



Vitamin D and calcium tablets



Suppresses PTH



Reduces bone turnover



Bone pain



# Image Attribution

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