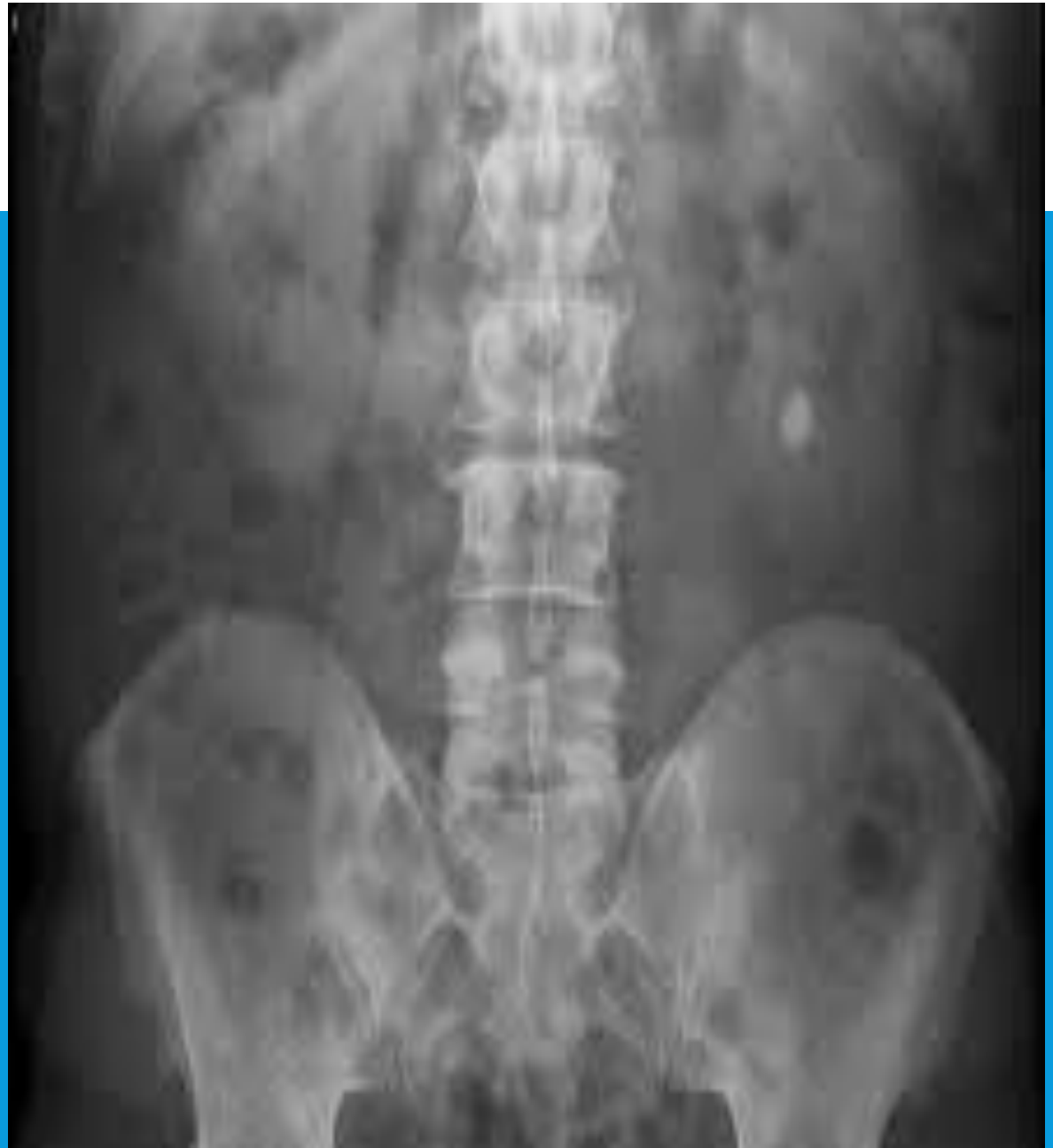
CASE SCENARIO-1

A 25-year-old female presented with recurrent severe pain in left lumber region for one year.

X-ray KUB revealed-



ABG

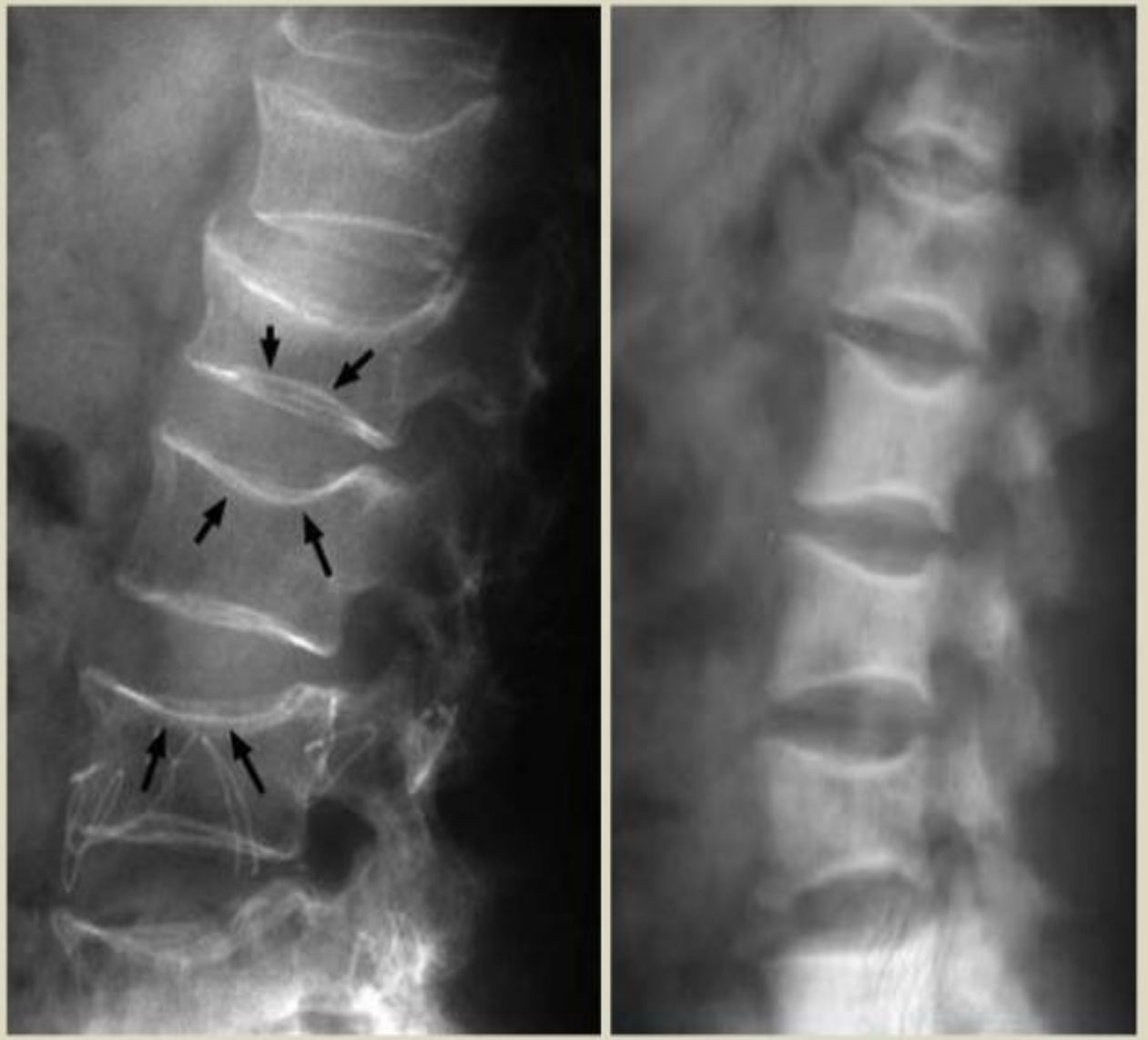
- ✓PH-7.2
- ✓HCO_3-10 mmol/L
- ✓$PaCO_2$-30 mmol/L
- ✓Na-136 meq/L
- ✓K-2 meq/L
- ✓Cl-114 meq/L
- ✓Anion Gap-14

Urinary PH-7.1

CASE SCENARIO-2

A 52-year-old male made recurrent visits in medicine OPD for low back pain.

X-ray L-S spine revealed



Osteomalacia with biconcave (fish vertebra) with endplate depression.

ABG

- ✓PH-7.25
- ✓HCO_3-12 mmol/L
- ✓$PaCO_2$-38 mmol/L
- ✓Na-138 meq/L
- ✓K-2.5 meq/L
- ✓Cl-116.5 meq/L
- ✓Anion Gap-12

Urinary PH-6.8

KEY FINDINGS IN TWO CASES

- Metabolic acidosis
- Urinary PH >5.5
- Hypercloraemia
- Normal Anion Gap
- Hypokalaemia

RENAL TUBULAR ACIDOSIS

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RENAL TUBULAR ACIDOSIS (RTA) SHOULD BE SUSPECTED WHEN

- There is a hyperchloraemic acidosis
- Normal anion gap
- No evidence of gastrointestinal disturbance.
- The urine pH is inappropriately high (> 5.5)
- Presence of systemic acidosis.

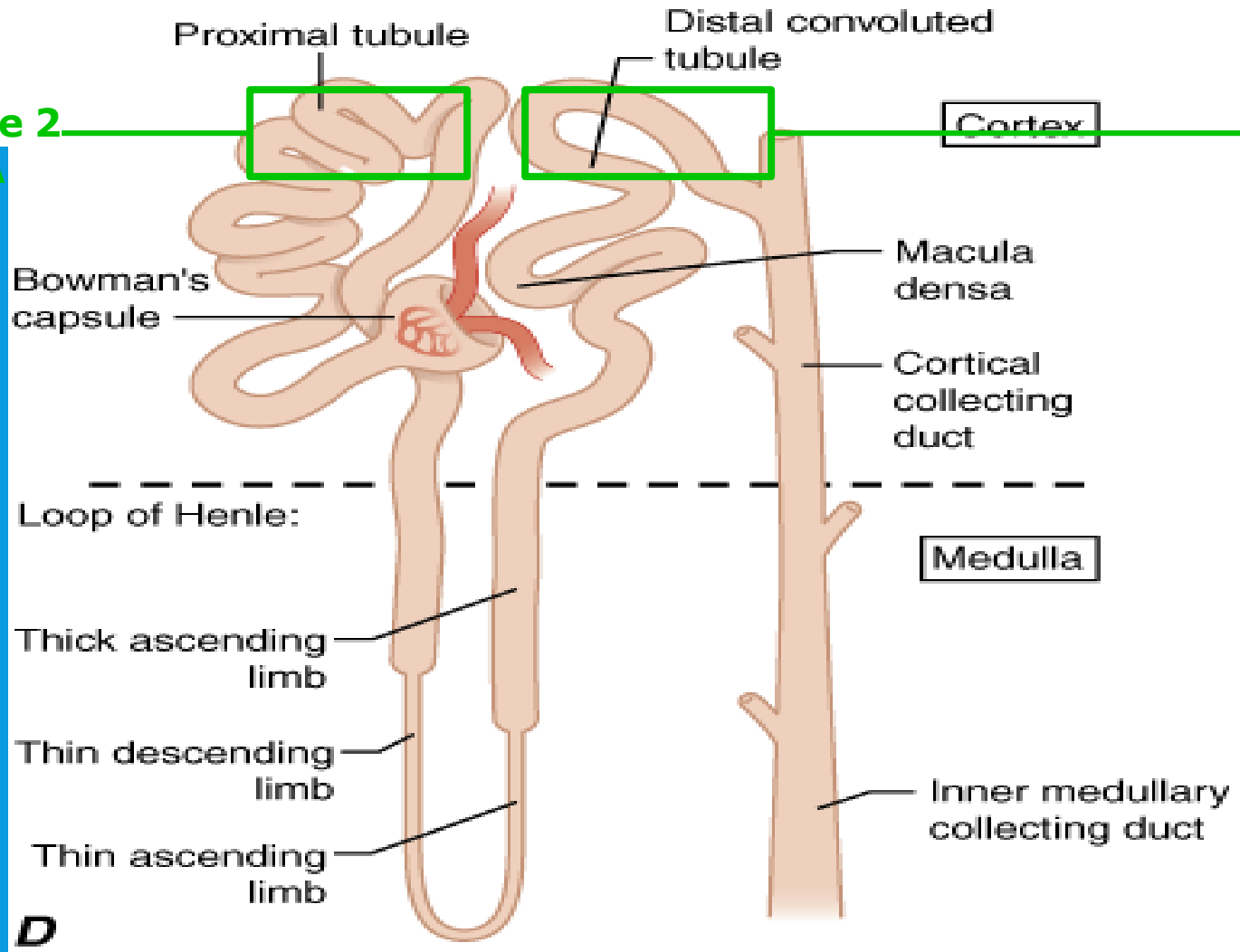
HISTORY

- First described in 1935
- Confirmed as renal tubular disorder in 1946
- Entitled as ‘Renal Tubular Acidosis’ in 1951

PATHOPHYSIOLOGY

- Impaired bicarbonate reabsorption in the proximal tubule (proximal RTA)
- Impaired acid secretion in the late distal tubule ,cortical collecting duct intercalated cells (classical distal RTA)
- Impaired sodium reabsorption in the late distal tubule or cortical collecting duct, which is associated with reduced secretion of both potassium and H+ ions (hyperkalaemic distal RTA).

Renal Tubular Acidosis



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: http://www.accessmedicine.com

CAUSES OF PROXIMAL RENAL TUBULAR ACIDOSIS

- Inherited- Fanconi's syndrome,Cystinosis,Wilson's disease
- Paraproteinaemia
- Myeloma
- Amyloidosis
- Hyperparathyroidism
- Heavy metal toxicity-Lead, cadmium and mercury poisoning
- Drugs-Carbonic anhydrase inhibitors,Ifosfamide

CHARACTERISTICS OF PROXIMAL RENAL TUBULAR ACIDOSIS

- Wasting of amino acids, phosphate and glucose(Fanconi's syndrome), as well as bicarbonate and potassium.
- Lower the urine pH when the acidosis is severe
- Plasma bicarbonate levels have fallen below 16 mmol/L since distal H+ secretion mechanisms are intact.

CLINICAL MANIFESTATIONS

- Failure to thrive, growth retardation.
- Polyuria, Polydipsia
- Dehydration (due to sodium, H_2O Losses)
- Rachitic Manifestations.
- Irritability, anorexia

CAUSES OF CLASSICAL DISTAL RENAL TUBULAR ACIDOSIS

- Inherited
- Autoimmune diseases-Systemic lupus erythematosus,Sjögren's syndrome
- Hyperglobulinaemia
- Toxins and drugs-Toluene,Lithium,Amphotericin

CHARACTERSTICS OF CLASSICAL DISTAL RTA

- Acid accumulation is relentless and progressive
- Mobilisation of calcium from bone
- Osteomalacia with hypercalciuria, renal stone formation and nephrocalcinosis.
- Potassium is lost in classical distal RTA.

CLINICAL MANIFESTATIONS

- Failure to thrive, Growth retardation.
- Polyuria, Polydipsia
- Nephrocalcinosis, Nephrolithiasis
- Rachitic manifestations (later in childhood),Osteomalacia(in adult)
- Weakness,Transient paralysis(due to hypokalaemia)

CAUSES OF HYPERKALAEMIC DISTAL RENAL TUBULAR ACIDOSIS

- Hypoaldosteronism (primary or secondary)
- Obstructive nephropathy
- Renal transplant rejection
- Drugs-Amiloride,Spironolactone

CHARACTERSTICS OF TYPE 4 RTA

- Most commom type
- Impaired Aldosterone secretion or distal tubule resistance to Aldosterone
- Impaired function of Na^+/K^+-H^+ (Cation exchange mechanism)
- Decreased H^+ and K^+ secretion →plasma buildup of H^+ and K^+(Hyperkalaemia)
- Urine pH < 5.5 (because the distal tubule H+ pump functions normally)
- Renal function may be impaired.

CLINICAL MANIFESTATIONS OF TYPE 4 RTA

- Growth retardation.
- Polyuria, polydipsia, dehydration.
- Signs and symptoms of obstructive uropathy and features of pyelonephritis.
- Bone diseases are usually absent.

INVESTIGATIONS

First line investigations

- Complete Blood Count
- Blood sugar
- Urine R/M/E
- S.Creatinine
- Arterial Blood Gas Analysis
- S.Electrolytes
- Urinary PH
- NH_4Cl acidification test
- Furosemide test

Second line investingations

- USG of KUB
- S.Phosphate
- S.Amino acid
- S.caeruloplasmin
- 24 hours urinary copper
- S.Protein electrophoresis
- Tissue exam for amyloidosis
- S.calcium,PTH
- Plasma renin,aldosterone,ARR

MANAGEMENT

- Identification and correction of the underlying cause.
- Controlling diarrhoea, diabetes mellitus, correcting shock, stopping drugs that might cause the condition
- Dialysis to remove toxins.
- Intravenous fluids

MANAGEMENT

- Rapid correction of acidosis can induce hypokalaemia or a fall in plasma ionised calcium
- Indication of the use of bicarbonate infusions
- ✓Where the underlying disorder cannot be readily corrected
- ✓ Acidosis is severe ($H^+ > 100$ nmol/L, $pH < 7.00$)
- ✓Associated with evidence of tissue dysfunction.

MANAGEMENT

- Supplements of sodium and potassium bicarbonate are usually also necessary in types 1 and 2 RTA to achieve a target plasma bicarbonate level of above 18 mmol/Land normokalaemia.
- In type 4 RTA, loop diuretics, thiazides or fludrocortisone (as appropriate to the underlying diagnosis) may be effective in correcting the acidosis and the hyperkalaemia.

KEY MESSAGE

High index of suspicion to diagnose

THANK YOU