Welcome to CME

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Key Concerns in a Preterm Baby:

Polyuria, Feeding Intolerance & Inadequate Growth

Case Summary

- **B/O Sharmin**
- **Baby girl**
- **Place of birth:** Ad-Din Hospital, **LUCS**
- **Gestational age:** 32 + 2 weeks
- **Birth weight:** 910 g

- **Maternal history:**
  - Antepartum hemorrhage (abruptio placentae)
  - Placental insufficiency
  - Oligohydramnios
  - Intrauterine growth restriction (IUGR)
- **At birth:** Baby **cried spontaneously**

Immediate Postnatal Management

- **Baby immediately placed on radiant warmer**
- **Positioned, dried, and stimulated**
- **APGAR scores:** 6/10 at 1 min, 8/10 at 5 min
- **SpO_2:** In room air → 90%
- **Post-resuscitation:**

  Shifted to NICU with proper wrapping and O_2 support

  En-route: Uneventful

Antenatal History-

- Mother: Sharmin, 30 years, Gr- 4^{th}, P-3+1, Housewife, B +ve
- Father: Aminul ,32 years,living abroad
- Her antenatal serological marker & HVS screening were negative
- Got antenatal corticosteroid (ANS) –two doses

AT NICU:

Baby was kept under radiant warmer

- Connected to Vitals monitor
- Temp recorded 36.8^0c
- R/R-50 breaths /min
- No grunting and Chest indrawing
- Respiratory Severity Score (RSS) - 3/10

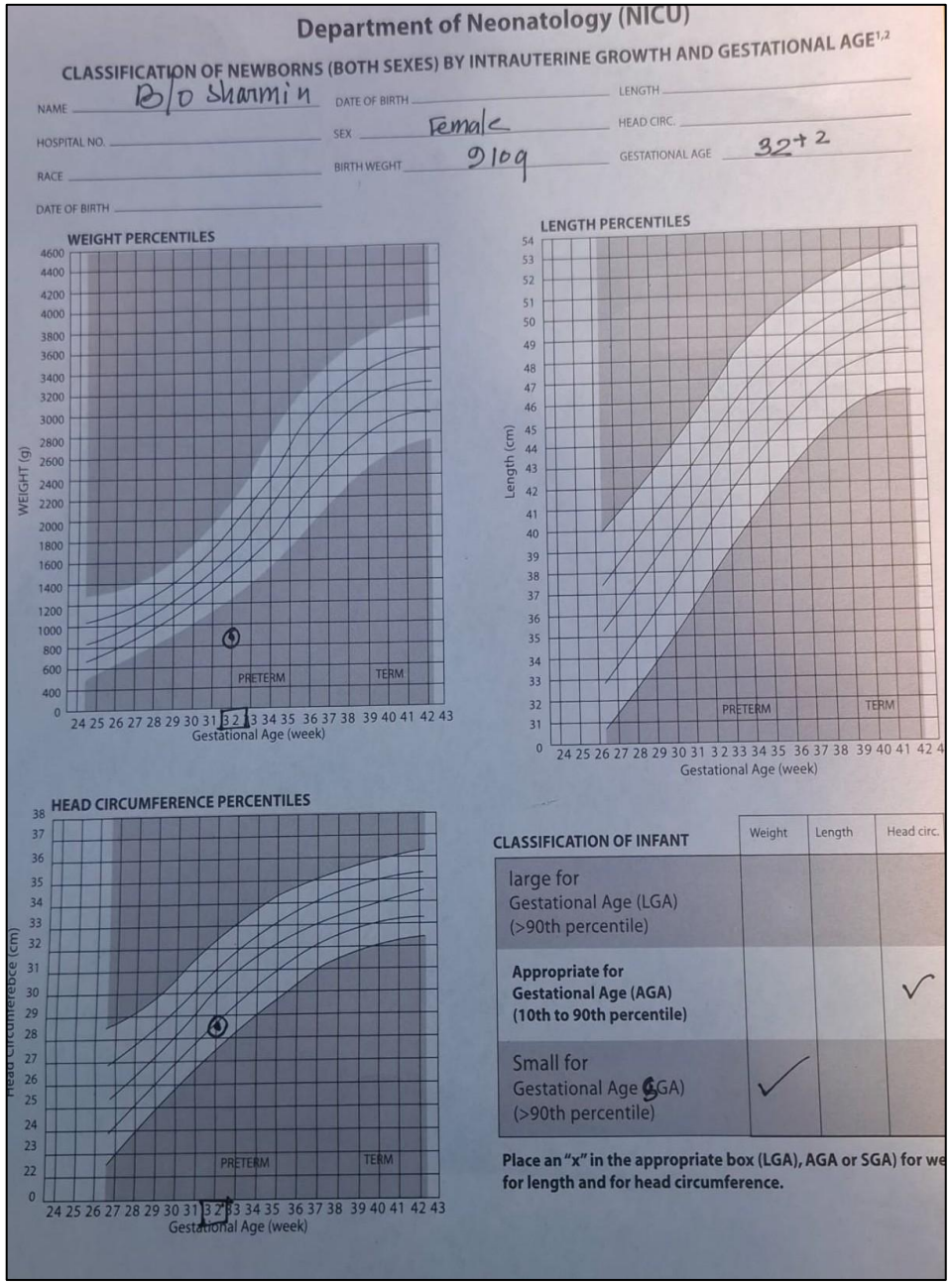
- PICTUTRE

At NICU

- SpO2-90 -92 %
- Good perfusion- Perfusion index(PI >1)
- Chest : AIR entry – good
- Precordium- S1 and S2 , no murmur.
- Reflex & activities- moderate
- Other systemic examination- Normal

Anthropometry

- Weight-910 g
- OFC- 28.7 cm



Initial Management

- Resp. support- Maintained SpO2 in room air
- Thermal care
- Fluid and electrolytes
- Antibiotics – as per NICU protocol
- Feeding started

Working Diagnosis:

- Moderate Preterm (32 +), ELBW (990g), SGA(IUGR)
- Presume Sepsis

Investigations sent:

- Blood gas analysis
- septic screening
- other relevant investigations

Peripheral line and **UVC** were done

Chest X-ray

Blood gas:

pH:7.34

pCO2: 45mmHg

pO2: 48mmHg

HCO3 :18.8mmol/L, Base deficit : 5.2

Supportive Management:

- **Counseling**
- Feed-1cc 4hrly
- IVF: 10% dextrose
- Amino acid- initially 3 g/kg then inerease
- Maintenance calcium
- Antibiotics: As per protocol
- Inj. Caffeine Loading and maintenance

- picture

Initial Investigation:

1.Septic panel:

- **CBC**:
  - Hb 16.6gm/dl
  - HCT:44%
  - TC: 8.38x10^3 /cumm
  - DC: N- 43.1%, L -42.6%, M- 13.5%, E- 0.7%
  - PC: 220000/ cmm

- **PBF :** Normal
- **Blood C/S-sent**
- **CRP-** 2.6 mg/L

Investigations cont…

- **S. Electrolytes**: Na-140mmol/L, K- 4.3mmol/L, Cl- 107 mmol/L
- **S. calcium**: 7.23 mg/dl
- **S. Mg**: 0.87 mmol/l
- **RBS:** 4.5 mmol/l
- **Vit D** : 28.84ng/ml
- **BUN-** 8.25 mg/dl
- **S.Cr-** 0.83 mg/dl

progress in NICU

At 24 hours :

- Temp- Normal
- RR- 48b/min
- HR- 136b/min
- SpO2 – 90-95%
- No signs of respiratory distress, cardiac murmur
- No abdominal distension
- Feed Tolerated

Day 3:

- Feeding – increased acc. to feeding protocol.
- Phototherapy started- for hyperbilirubinemia
- Vitals- Normal
- A systolic murmur present
- Reflexes and activities- Moderate

At day-4

- Feeding intolerance
- Abdominal distension
- No vomiting
- No signs of respiratory distress, cardiac murmur, or hepatosplenomegaly
- **Baby developed polyuria- 4.8 ml/kg/day**
- Reflex & Activities – decreased

Hospital course- Day 5

Fluid intake & Output monitoring

- **Daily intake–output chart**
  - Note **persistent polyuria**
  - Current status: **Negative fluid balance**
- **Daily weight monitoring**
  - 11 % **weight loss (910 g – 810 g)** → suggests significant fluid deficit

IV Fluid Management

- IV fluids **adjusted according to ongoing losses**
- Balance fluid replacement with:
  - Urine output trends
  - Weight changes
  - Serum electrolytes
  - Blood gas results

Interventions

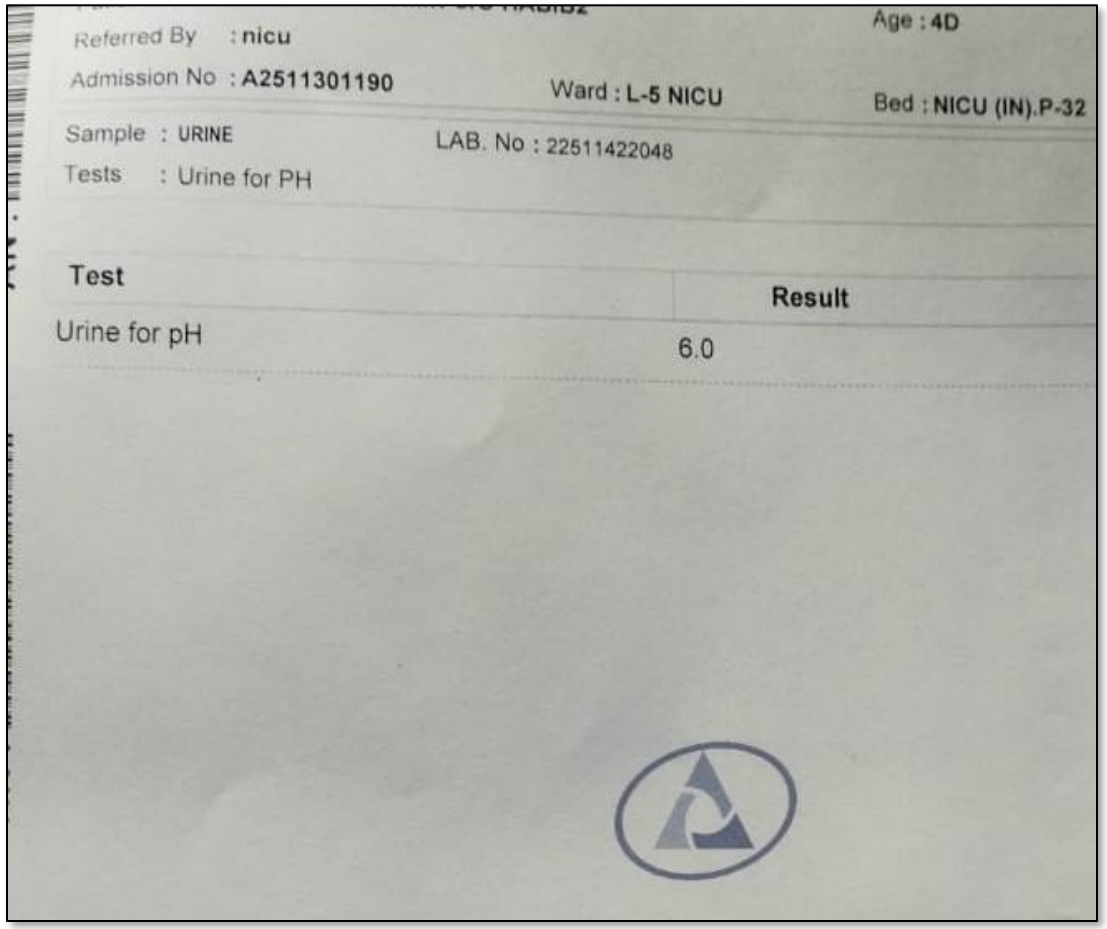
- Blood gas
- Urine PH
- Urine Electrolytes
- S. Electrolytes
- Repeat septic profile

Blood Gas

Test	Result	Normal Range	Interpretation
pH	7.18	7.35–7.45	Low
HCO_3^-	14.3 mmol/L	17-21 mmol/L	Low
pCO_2	27 mmHg	35–45 mmHg	Low
Na^+	145 mmol/L	135–145	Normal
K^+	3.4 mmol/L	3.5–5.5	Normal
Cl^-	117.5mmol/L	98–110	High
Anion gap	13.7 mmol/L	8–16	Normal

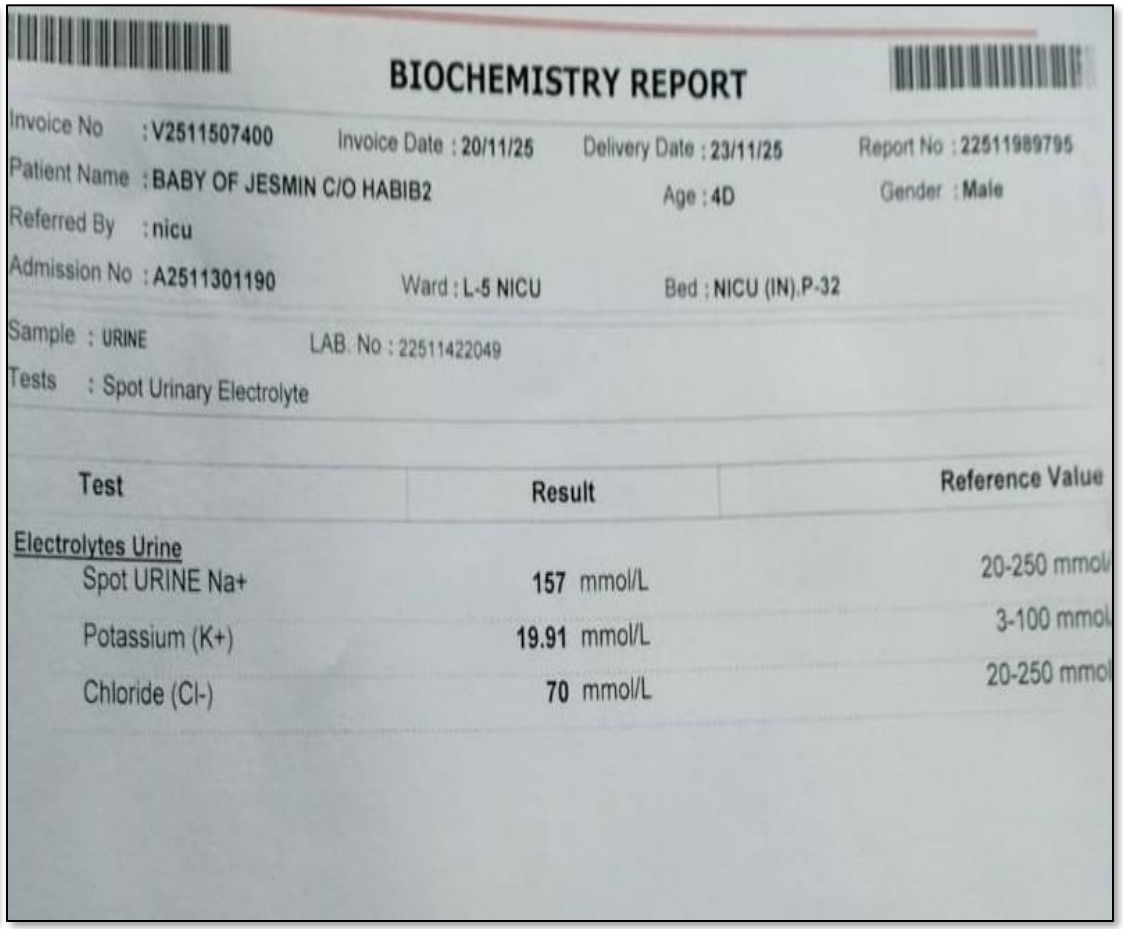
Hyperchloremic metabolic acidosis with Normal anion gap

Urine pH and Electrolytes:



Referred By : nicu
Age : 4D
Admission No : A2511301190
Ward : L-5 NICU
Bed : NICU (IN).P-32
Sample : URINE
LAB. No : 22511422048
Tests : Urine for PH

Test	Result
Urine for pH	6.0



BIOCHEMISTRY REPORT

Invoice No : V2511507400
Invoice Date : 20/11/25
Delivery Date : 23/11/25
Report No : 22511989795
Patient Name : BABY OF JESMIN C/O HABIB2
Age : 4D
Gender : Male
Referred By : nicu
Admission No : A2511301190
Ward : L-5 NICU
Bed : NICU (IN).P-32
Sample : URINE
LAB. No : 22511422049
Tests : Spot Urinary Electrolyte

Test	Result	Reference Value
Electrolytes Urine		
Spot URINE Na+	157 mmol/L	20-250 mmol
Potassium (K+)	19.91 mmol/L	3-100 mmol
Chloride (Cl-)	70 mmol/L	20-250 mmo

Urinary Electrolytes & pH

Urine pH	**6.0**	**<5.5 (should be low in acidosis)**	**Inappropriately high**
Urinary Electrolytes	Na – 157 K - 19.9 CL- 70		
Urine anion gap	106.9		Positive
Renal ultrasound	• Normal		

- Hyperchloremic metabolic acidosis with Normal anion gap with normal renal function indicate – suggestive of **Renal tubular Acidosis**

Therapeutic measures included:

- **Initial – Intravenous** Correction followed by
- **Oral sodium bicarbonate supplementation** at 3-5 mEq/kg/day in 4 divided doses.
- **Adequate fluid and electrolyte management.**
- **Exclusive expressed breast milk** feeding continued with gradual increments.
- **Regular monitoring** of weight, serum electrolytes, and acid-base status.

Clinical Progress & Follow-up

- **Improvement noted by Day 10–12:**
  - Increased activity
  - Better feeding tolerance
  - U/O- 3-4 ml/kg/d
- **Repeat BG :**
  - **pH:** 7.33
  - **HCO_3^-:** 18 mmol/L
  - **pCO_2:** 35 mmHg
- **Nutrition & Growth:**
  - Feeding well tolerated
  - Gradual, steady weight gain

Complications and Clinical Course

- **Blood culture positive for *Acinetobacter***
- Developed **DIC with thrombocytopenia** → managed with **FFP transfusion**
- **Echocardiography on Day 7:**
  - ASD (3 mm)
  - Moderate pulmonary hypertension
- Started on **Inj. Milrinone**
- **ROP screening-Zone III, Immature no plus (B/L)**

NICU course

- SpO_2 maintained in room air from day 19

- **PRBC transfusion (10 ml/kg**) and **Darbepoetin (3 doses)** given for neonatal anemia.

- Weight gain started from day 19

- Reached full enteral feeds by day 24

MNCU..

- Initiated Kangaroo Mother Care (KMC)
- Baby is feeding well and gaining weight
- Mother trained in newborn care and feeding techniques
- Feeding pattern established: exclusive breastfeeding or supplemented feeds
- Mother trained in feeding techniques, volume, frequency, and burping.

Outcome and Follow-Up:

- The infant was discharged at **38 days of age** weighing **1350 kg**, with normal hydration, good feeding, and improved biochemical profile.
- Parents were counseled on the need for **regular follow-up** to monitor growth, renal function.
- At 2 months follow-up, growth parameters were appropriate for corrected gestational age, and no recurrence of acidosis was observed.

Discharge Diagnosis

- Mod Preterm , ELBW, SGA (IUGR)
- **Transient Renal Tubular Acidosis of Prematurity**
- Neonatal Anaemia
- Acinetobacter sepsis
- DIC
- ROP: Zone III , Immature no plus.
- CHD (ASD- 3 mm)

Renal Tubular Acidosis

RTA refers to a group of disorders in which the **kidneys fail to maintain acid-base balance** due to impaired acid secretion or bicarbonate reabsorption in the renal tubules — despite normal glomerular function.

Metabolic Acidosis

- A primary **decrease in plasma HCO_3^-** leading to **low blood pH (<7.35)**.
  ↓ HCO_3^- (primary change)
- **↓ pH** (<7.35)
- **Compensatory ↓ pCO_2** → hyperventilation/Kussmaul breathing)
- **Anion gap (AG): Normal 8-16**
  - **High AG** → accumulation of acids (lactic acidosis, ketoacidosis, toxins)
  - **Normal AG** → HCO_3^- loss or impaired renal acid excretion (diarrhea, RTA)

High Anion Gap Metabolic Acidosis (HAGMA)

- **Accumulation of unmeasured acids** in the blood (organic acids, toxins).
- These acids **consume HCO_3^-**, lowering its concentration.

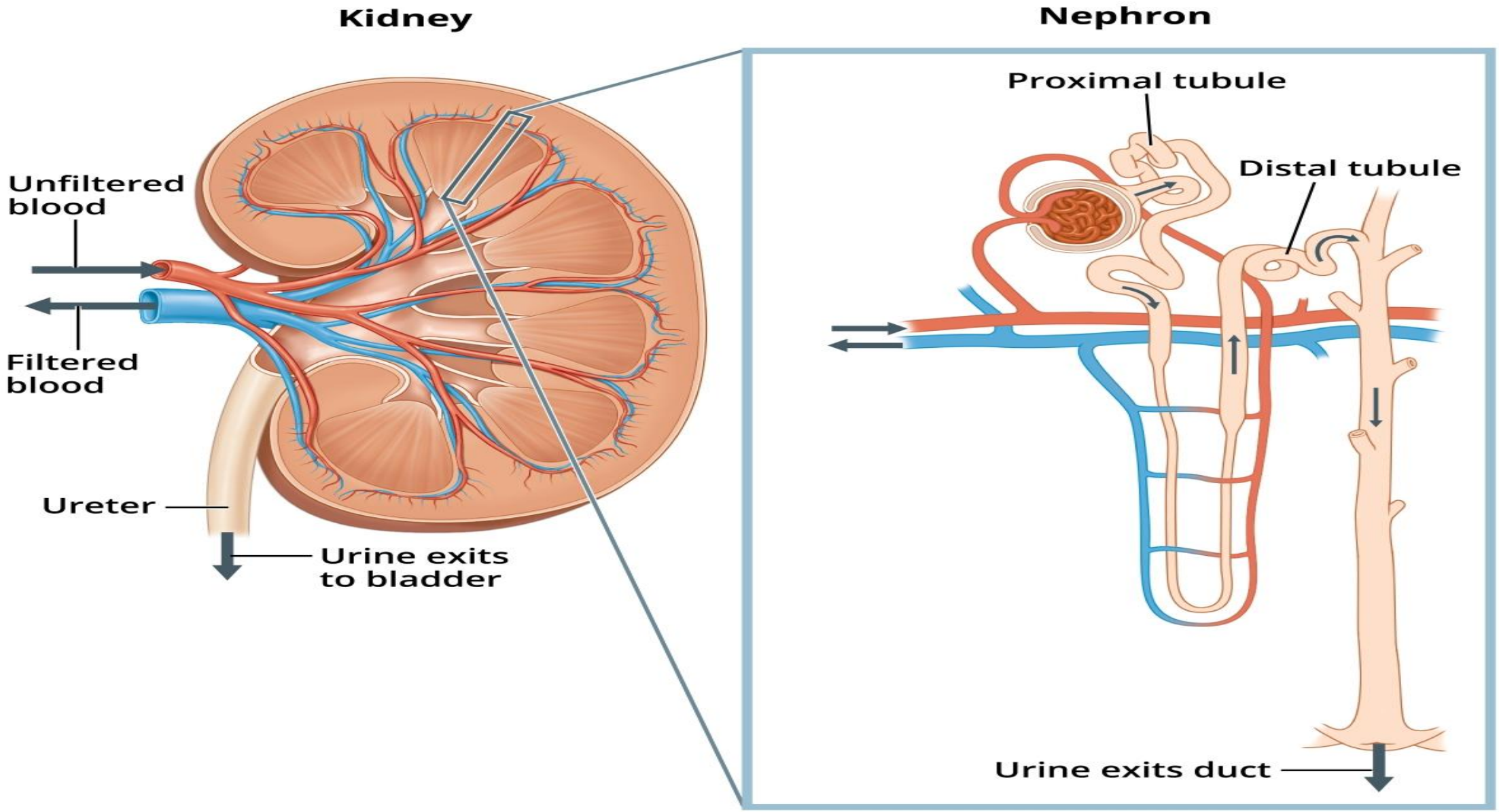
- **Anion gap ($AG = Na^+ – [Cl^- + HCO_3^-]$) increases**.
  Uraemia (renal failure)
  Diabetic ketoacidosis
  Lactic acidosis
  Salicylates

Normal Anion Gap Metabolic Acidosis (NAGMA)

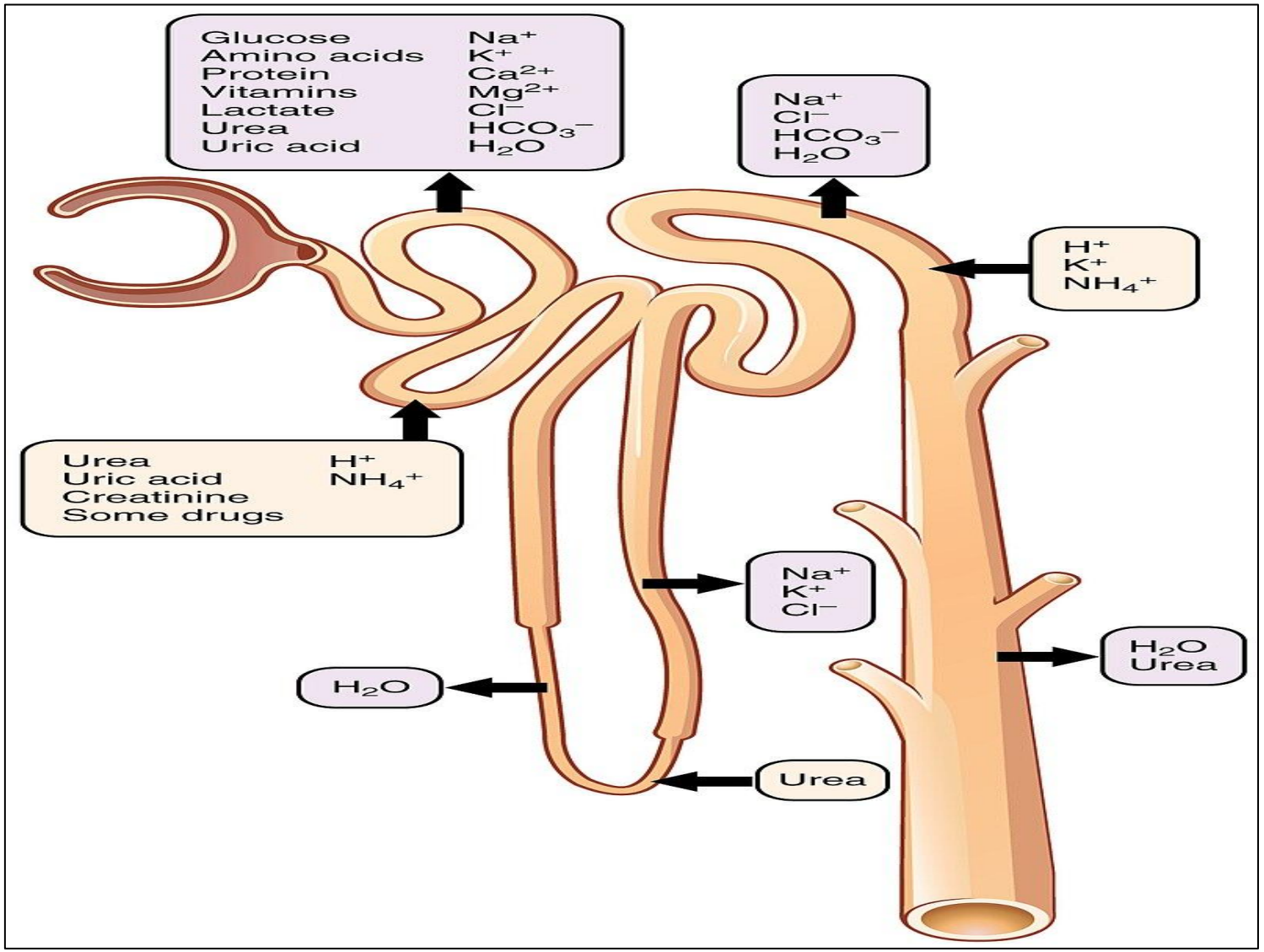
- **Mechanism:**
- Caused by **loss of HCO_3^-** or **impaired renal H^+ excretion**.
- **Chloride (Cl^-) rises to replace lost HCO_3^-** ("hyperchloremic acidosis").
- **Gastrointestinal loss of HCO_3^-** → diarrhoea, pancreatic fistula
- **Renal tubular acidosis (RTA):**
  - Type 1 (distal), Type 2 (proximal), Type 4 (hypoaldosteronism)
- **Drugs** → e.g., acetazolamide (bicarbonate wasting), aminiglycosisde
- **Key point:** AG normal because lost HCO_3^- is replaced by Cl^-.

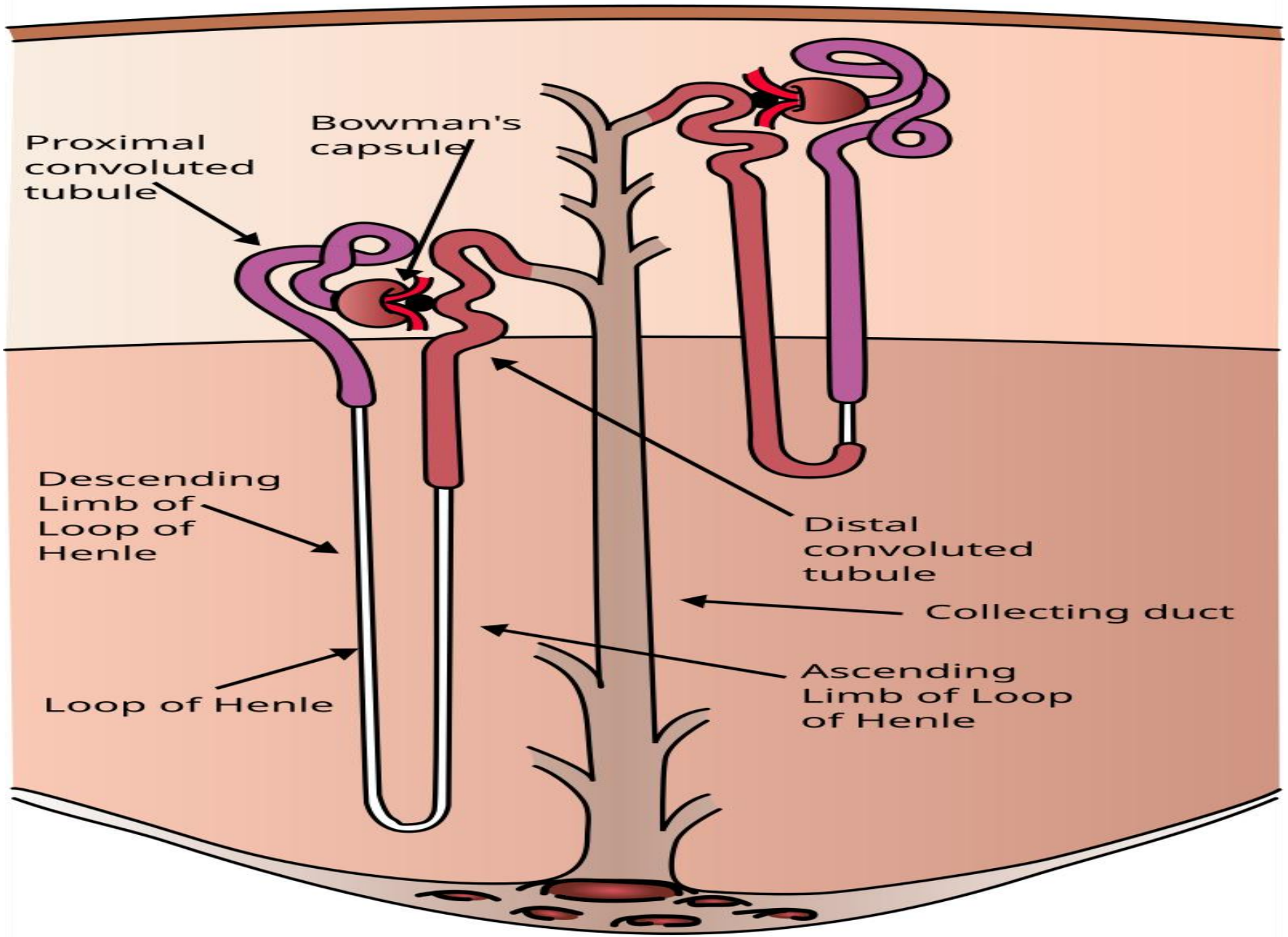
Normal Acid-Base Physiology in the Kidney

- The kidneys maintain **acid-base balance** by:
- **Reabsorbing almost all filtered bicarbonate (HCO_3^-)**
- **Excreting daily acid load (H^+) produced by metabolism**
- **Generating new bicarbonate** to replace what is lost in buffering acids


Kidney
Nephron
Unfiltered blood
Filtered blood
Ureter
Urine exits to bladder
Proximal tubule
Distal tubule
Urine exits duct

Renal tubular electrolytes absorption and secretion




Proximal convoluted tubule
Bowman's capsule
Descending Limb of Loop of Henle
Loop of Henle
Distal convoluted tubule
Collecting duct
Ascending Limb of Loop of Henle

Molecular mechanisms of Bicarbonate reabsorption in proximal tubule (PT)

1. In the tubular lumen (urine side)

bicarbonate (HCO_3^-) combines **secreted H^+ → carbonic acid (H_2CO_3)**.

H_2CO_3 is converted to **$CO_2 + H_2O$**

CO_2 diffuses **into the proximal tubule cell**.

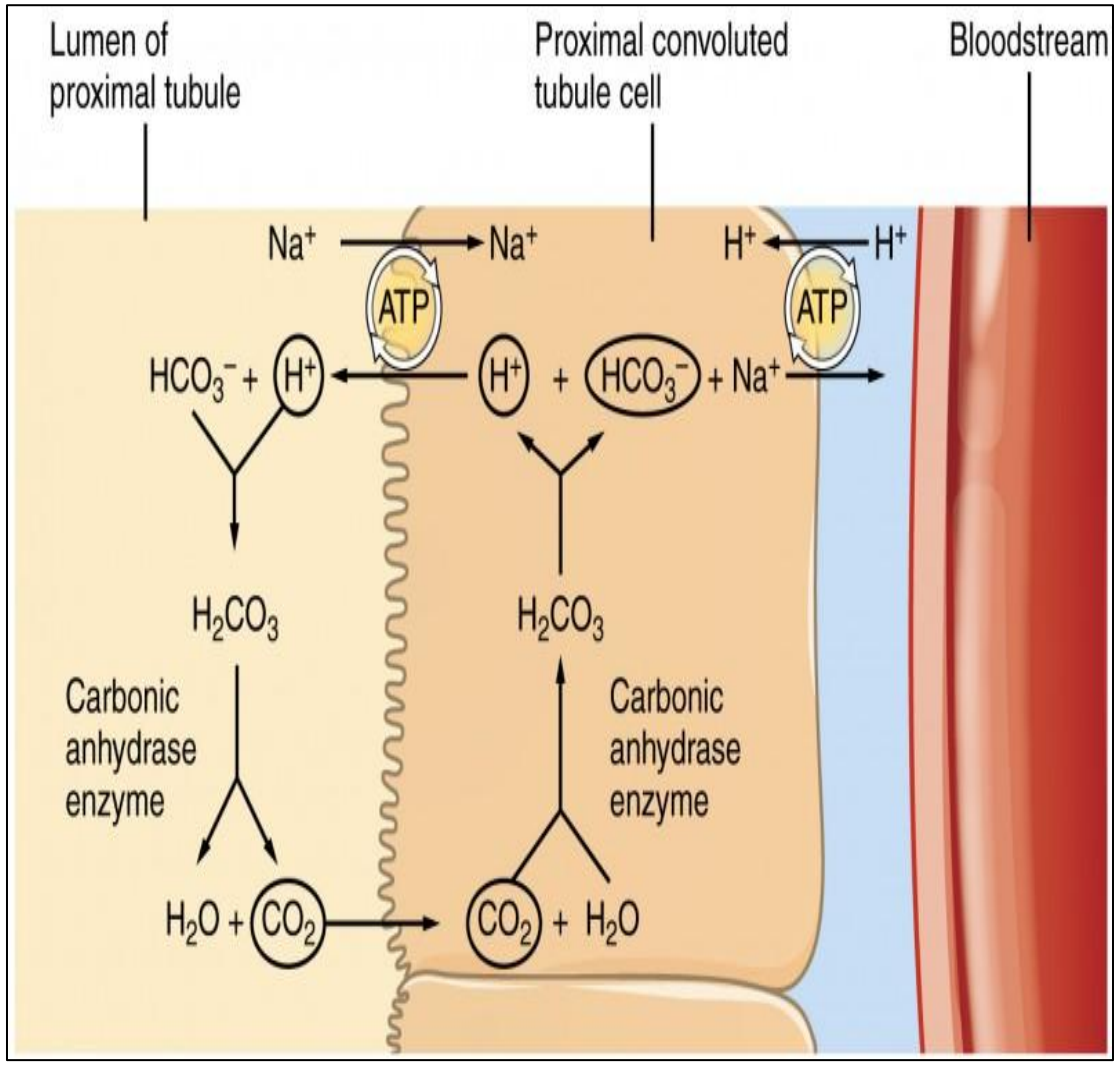
2. Inside the proximal tubule cell

$CO_2 + H_2O$ **to H_2CO_3 → $H^+ + HCO_3^-$**.

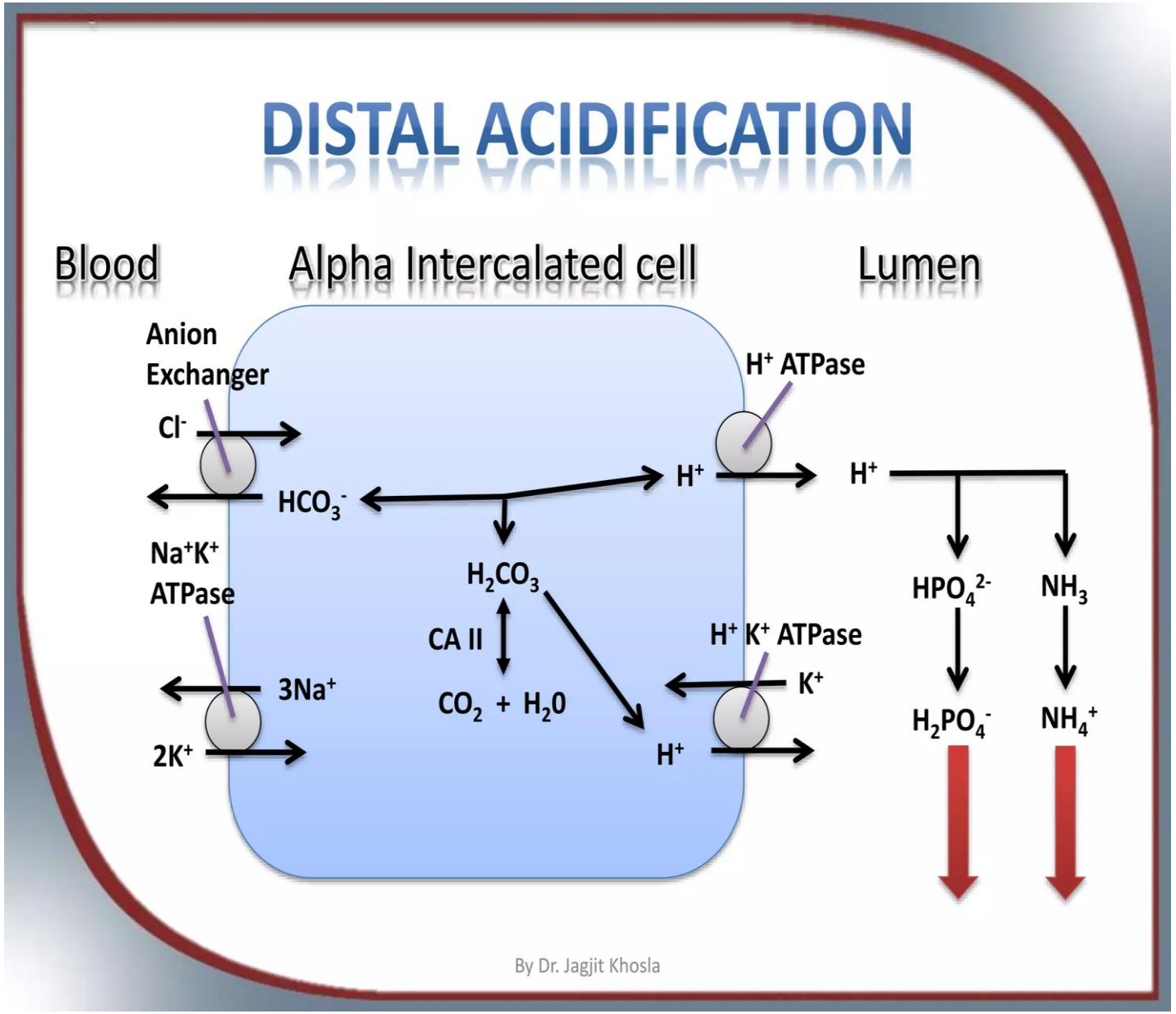
H^+ is recycled back into the lumen

3. Basolateral transport to blood

HCO_3^- together with Na^+ via a **Net result:** Reabsorption of HCO_3^- from urine into blood.



Urine Acidification in Distal Tubule



Nephron Site	Main Process	Mechanism	Purpose
Proximal tubule	Reabsorb filtered HCO_3^-	Na^+/H^+ exchange, carbonic anhydrase	Prevent HCO_3^- loss
Thick ascending limb	Some H^+ secretion	Na^+/H^+ exchange	Fine-tuning
Distal tubule / Collecting duct	Secrete H^+, generate new HCO_3^-	H^+-ATPase, H^+/K^+ ATPase	Final acid excretion
Proximal + distal nephron	Ammonium handling	NH_4^+ excretion	Long-term acid balance

When This Fails → RTA

Defect	Site	Result
↓ HCO_3^- reabsorption	Proximal tubule	**Type 2 (Proximal RTA)**
↓ H^+ secretion	Distal tubule	**Type 1 (Distal RTA)**
↓ Aldosterone or response	Collecting duct	**Type 4 (Hyperkalemic RTA)**

RTA in Neonates

- May be **transient** (due to tubular immaturity)
- Or **persistent** (due to inherited or acquired causes)
- **Preterm infants**, especially **VLBW babies**, are more prone to acid–base disturbances because of:
  - Reduced nephron maturity
  - Limited bicarbonate reabsorption

RTA in neonates :

- **Prematurity-related tubular immaturity** (most common)
- **LBW infants with perinatal asphyxia** → tubular ischemia
- **Nephrotoxic drugs:** Aminoglycosides, NSAIDs, diuretics
- **Congenital disorders:** Rare, e.g., inherited distal RTA
- **Sepsis or hypovolemia** → transient tubular dysfunction

Etiology

RTA is due to-

- Primary (Hereditary)

  Autosomal dominant – isolated distal RTA (milder form)

  Autosomal recessive – severe, early-onset, often with sensorineural deafness

- Aquired- any condition that affects kidneys ability to absorp filtered bicarbonate, or excrete ammonia or titrable acid.

In **preterm neonates**, the renal tubules are **functionally immature**, leading to:

- Decreased bicarbonate reabsorption
- Reduced acid excretion
- Limited ability to concentrate urine

Hence, **a degree of "Transient RTA"** is common in preterm infants, especially those <34 weeks gestation.
This usually **improves with maturation** (by 4–8 weeks postnatal age).

Why different from older children

- Neonatal kidneys have:
  - Lower **GFR** and tubular flow.
  - Immature **distal acidification** capacity.
  - Reduced **NH_4^+ excretion** due to limited glutamine metabolism.
- Therefore, they **retain acid less efficiently** early in life → UAG remains slightly positive.

Pathophysiology in Preterm/LBW Babies

- **Tubular Immaturity:**
  - Both proximal and distal tubular transporters are underdeveloped.
  - Full acidification capacity matures around 32–36 weeks gestation.

- **Limited Buffering Capacity:**
  - Low muscle mass and bone buffering → worsening acidosis

- **Stress Factors:**
  - Perinatal hypoxia, sepsis, or nephrotoxic drugs can transiently worsen tubular dysfunction.

Types of RTA in Preterm/LBW Infants

1. Distal (Type 1) RTA – Impaired Acid Secretion

- **Defect:** The distal tubule (collecting duct) cannot adequately secrete hydrogen ions (H^+) into urine.
- **Mechanism:**
  - Immature H^+-ATPase and H^+/K^+-ATPase pumps in intercalated cells
  - Inability to lower urine pH below 5.5 despite systemic acidosis
  - Leads to **hyperchloremic metabolic acidosis**

2.Proximal (Type 2)

- **Defect:** The proximal tubule cannot reabsorb filtered bicarbonate (HCO_3^-) efficiently.
- **Mechanism:**
  - Immature Na^+/H^+ exchanger and bicarbonate transporters
  - Bicarbonate is lost in urine → metabolic acidosis
  - Once serum bicarbonate stabilizes at a lower level, distal acidification can partially compensate, so urine pH fall
- **Consequences:**
  - Hypokalemia due to increased distal K^+ secretion
  - May be associated with **Fanconi syndrome** (glucosuria, aminoaciduria, phosphaturia)

3. Type 4 RTA – Hyperkalemic RTA

- **Defect:** Impaired distal tubular acidification due to aldosterone deficiency or resistance.
- **Mechanism:**
  - Reduced aldosterone → decreased Na^+ reabsorption and H^+/K^+ excretion
  - Leads to mild metabolic acidosis
- **Consequences:**
  - Hyperkalaemia
  - Mild acidosis
  - Usually less effect on growth and bones

Clinical Presentations of RTA in Preterm Babies

1.Growth and Feeding Issues

- Poor weight gain despite adequate nutrition
- Failure to thrive appear similar to feeding intolerance or chronic illness
- Polyuria
- Feeding difficulties / vomiting
- Lethargy and irritability

.

Clinical features cont..

- **Dehydration**

  Due to polyuria and renal salt wasting (especially in proximal RTA).

- **Developmental delay**

  Chronic acidosis and malnutrition affect overall neurodevelopment.

Respiratory Manifestations

- **Tachypnea**
- **Apnea / periodic breathing**
- **Respiratory distress or increased work of breathing**

Electrolyte-Related Manifestations

- Muscle weakness, constipation,
- Muscle hypotonia, bone demineralization
- Bradycardia, lethargy, potential cardiac conduction issues

.)

Chronic / Late (If Untreated)

Manifestation	Mechanism
Severe growth retardation	Persistent acidosis and malnutrition
Rickets / osteomalacia	Chronic bone buffering
Recurrent nephrolithiasis	Alkaline urine, hypercalciuria
Chronic kidney disease (rare)	Due to nephrocalcinosis and progressive tubular injury

Initial Evaluation — Confirm Metabolic Acidosis

Step 1- Blood Gas analysis -

↓ pH (<7.35), ↓ HCO_3^-, $PaCO_2$

Confirms **metabolic acidosis**

Serum electrolytes

↓ HCO_3^-, ↑ Cl^-, K^+ ↓ (Type 1 & 2) / ↑ (Type 4)

Step2 - Calculation anion gap

Anion Gap (AG) = Na^+ - (Cl^- + HCO_3^-)

Normal (8–16 mmol/L)

Interpretation:

➞ If **normal AG metabolic acidosis** and **normal renal function** → **suspect RTA**

Step-3 Urine Studies — Localization (Distal vs Proximal)

- Urine PH and urinary Electrolytes

- Urine PH

- UAG $= (Urine\ Na^{+} + Urine\ K^{+}) - Urine\ Cl^{-}$

Normal older child : –20 to +20

Term neonate (1st week): +10 to +30

Preterm neonate -: +20 to +40

Step-3 Urine Studies — Localization (Distal vs Proximal)

Test	Type 1 (Distal)	Type 2 (Proximal)	Type 4 (Hyperkalemic)	Purpose
Urine pH	>5.5 (cannot acidify urine)	<5.5	<5.5	Key screening test
Urine anion gap (UAG) = $(Na^+ + K^+) – Cl^-$	**Positive** (↓ NH_4^+ excretion)	Positive	Positive	Reflects urinary NH_4^+ excretion (acid excretion)

Age group	Expected UAG (mmol/L)	Comments
Term neonates (first week)	**+10 to +30 mmol/L**	Tubular acidification immature → low NH_4^+ excretion → mildly **positive UAG** is normal
After 1–2 weeks of age	**0 to –20 mmol/L**	As renal acidification matures, NH_4^+ excretion increases → **UAG becomes neutral or negative**
Preterm neonates	**More positive (up to +40 mmol/L)**	Immature distal nephron → reduced acid excretion

Assess Renal Function and Rule Out Other Causes

Test	Findings	Purpose
Serum creatinine, BUN	Normal (usually)	Excludes renal failure
Urinalysis	No glycosuria/proteinuria (unless Fanconi syndrome)	To exclude glomerular disease
Urine ketone/lactate	Negative	Exclude keto/lactic acidosis

Step 4: Imaging & Additional Tests

- **Renal ultrasound:** nephrocalcinosis or stones (especially distal RTA)
- **Bone X-ray:** rickets / bone demineralization
- **Genetic / metabolic testing:** if hereditary RTA or syndromes suspected

Treatment Principles

Regardless of type, the main goals are:

- Correct and maintain acid–base balance (i.e., normalize serum bicarbonate)
- Correct associated electrolyte abnormalities (potassium, phosphate, etc)
- Preserve growth, bone health (prevent rickets/osteomalacia)
- Prevent complications (nephrocalcinosis, kidney stones, chronic kidney disease)
- Identify & treat underlying causes when present

1. Supportive :

- Ensure adequate hydration
- Nutritional support
- Promote growth and bone mineralization
- Avoid acidogenic drugs

  e.g., carbonic anhydrase inhibitors
- Treat rickets if present
- Vitamin D and phosphate as indicated

2. Correction of Acidosis - Alkali Therapy

.

RTA Type	Target Serum HCO_3^- (mmol/L)	Alkali Dose
Type 1 (Distal RTA)	18–22	**3–5 mmol/kg/day** sodium bicarbonate or citrate
Type 2 (Proximal RTA)	18–20	**5–15 mmol/kg/day**, may require higher dose due to bicarbonate loss
Type 4 (Hyperkalemic RTA)	18–20	**1–3 mmol/kg/day**; mild correction only

Treatment Based on Severity of Acidosis

A. Mild Acidosis

- **HCO_3^- = 16–18 mmol/L**
  pH ≥ 7.30
- Usually **no bicarbonate** required.
- **Ensure adequate fluids & calories**
- Monitor HCO_3^- every 48–72 hours.

B. Moderate Acidosis

HCO_3^- = 12–16 mmol/L
pH 7.20–7.29

- **Treatment:**

1.Oral Alkali Therapy (preferred): Sodium bicarbonate:

1–2 mEq /kg/day divided every 6–8 hours

2. Potassium

- Only if **K^+ < 4 mEq/L**
- K-citrate preferred in distal RTA, but in prematurity RTA **Na-citrate alone** often sufficient.
- **Monitoring:**
- Check HCO_3^- every 24–48 hours until stable.

C. Severe Acidosis

- HCO_3^- < 12 mmol/L
  pH < 7.20

- **1. IV Sodium Bicarbonate (acute correction)**
  $$\text{mEq } HCO_3^- = 0.3 \times \textit{weight (kg)} \times (\textit{Desired } HCO_3^- - \textit{Measured } HCO_3^-)$$

Raise only to 15–16 mmol/L acutely

- Give **slowly over 30–60 min** to avoid:
  - IVH
  - Hypernatremia
  - CO_2 retention

2. Then switch to oral Na-citrate or $NaHCO_3$

3–5 mEq/kg/day divided every 6 hours.

Target HCO_3^-

Severity	HCO_3^-	Treatment	Target HCO_3^-
Mild	16–18	Observe, fluids	18–20
Moderate	12–16	Oral $NaHCO_3$ / Na-citrate 1–3 mEq/kg/day	18–20
Severe	<12	IV $NaHCO_3$ acutely → oral alkali	18–20

Preferred oral alkali preparations

- **Sodium bicarbonate ($NaHCO_3$):** 1 mEq = 1 mmol HCO_3^-
- **Shohl's solution (sodium citrate–citric acid):** 1 mL ≈ 1 mmol HCO_3^-
- **Polycitra (sodium/potassium citrate):** 1 mL ≈ 1 mmol HCO_3^-

Sodium citrate is often preferred in infants — less gastric irritation and easy dosing.

3. Potassium Supplementation

Type	Potassium Status	Treatment
Distal RTA (Type 1)	Usually low (hypokalemia)	Add **potassium citrate** 1–2 mmol/kg/day
Proximal RTA (Type 2)	Low or normal	**K^+ supplementation** often required
Type 4 RTA	High (hyperkalemia)	Restrict K^+ intake; may need **loop diuretic** or **fludrocortisone**

Treatment Duration

Type	Expected Duration
Transient RTA of Prematurity	2–4 weeks, until renal maturation
Secondary RTA (due to illness, drugs, etc.)	Until underlying condition resolves
Primary (genetic) RTA	Lifelong alkali therapy

Hyperkalemic RTA (Type 4, rare in neonates)

- **Problem:** Aldosterone deficiency or resistance → impaired H^+/K^+ excretion
- **Treatment:**
  - Correct hyperkalemia: dietary restriction or potassium binders if needed
  - Alkali therapy (bicarbonate) for acidosis
  - Investigate and treat underlying cause of aldosterone deficiency/resistance

Follow-up & Monitoring Plan for Preterm RTA

1. During Hospital Stay

- **Acid–Base Status (blood gas)** → **every 48–72 hours** until stable
- **Electrolytes**
- Na^+, K^+, Cl^- → **every 2–3 days**
- K^+ may drop early → monitor closely
- **Weight & Growth**

  Daily weight

  Feeding tolerance

2. After Discharge (Outpatient Follow-up)

At 1 week

- Serum electrolytes, bicarbonate
- Weight check

Every 2 weeks until 2 months

- Serum bicarbonate
- Potassium
- Growth parameters (wt, length, HC)

At 2–3 months

- If HCO_3^- has normalized → taper alkali
- If acidemia persists → evaluate for inherited RTA

When to Stop Treatment

- Shohl’s solution / bicarbonate can be **stopped** when:
- Serum HCO_3^- > **18–20 mEq/L** Baby is thriving
- No tachypnea, no metabolic acidosis
- Usually around **4–12 weeks** of age.

Complications

*Usually **no long-term complications***

- **Failure to thrive (FTT)**
- **Poor weight gain**, feeding difficulty
- **Delay in length/height gain**
- **Neurodevelopmental delay** (if chronic & untreated)

Kidney & Urinary Complications

- **Distal RTA (Type 1) – high risk**
- **Nephrocalcinosis**
- **Nephrolithiasis** (rare in neonates, more in older infants)
- **Medullary calcification**
- **Chronic kidney disease (CKD)** if prolonged untreated
- **Recurrent UTIs**

Electrolyte Complications

Hypokalemia

- Muscle weakness, hypotonia
- Arrhythmias (rare but possible)
- Poor feeding from GI hypotonia
- Worse in **proximal RTA** due to high bicarbonate losses.

Hyperkalaemia (Type 4 RTA)

- Risk of **life-threatening arrhythmias**
- Apnoea in severe neonates

Prognosis of

Transient RTA of Prematurity

- *Usually appears* in **very preterm neonates (<34 weeks)**
- Improves with maturation of renal tubules
- **Most cases resolve by 4–8 weeks of age**
- Some may take up to **10–12 weeks** for full resolution
- Very rare to persist beyond **3–4 months**

Transient RTA of Prematurity

With appropriate bicarbonate therapy and nutrition:

- **Normal growth** expected
- **No long-term developmental issues**
- Failure to thrive is usually temporary and reversible

Renal Outcomes

- **No progression to chronic kidney disease**
- **No nephrocalcinosis** (unlike distal RTA)
- No increased risk of kidney stones
- No long-term tubular dysfunction

When to Suspect Poor Prognosis or Alternative Diagnosis

- If RTA persists **beyond 3–4 months**, consider:
- Inherited proximal RTA
- Fanconi syndrome
- Metabolic disorders (galactosemia, tyrosinemia)
- Underlying renal dysplasia

Summary

- **Alkali therapy** (bicarbonate or citrate) is the mainstay.
- **Potassium supplementation** as needed.
- **Supportive care:** hydration, nutrition, avoid nephrotoxins.
- **Frequent monitoring**.

New Adjuvant

- October 2025- approval of **ADV7103**,
- Marketed as **Sibnayal**.
- This novel medication combines **potassium citrate** and **potassium bicarbonate** in a **prolonged-release formulation**, allowing for **twice-daily dosing**. It has been authorized by the **European Medicines Agency (EMA)** for the treatment of dRTA in individuals aged **one year and old.**


Thank
You