



NephKids 2026

ELECTROLYTE

-WORKBOOK-

Organized by

Department of Pediatric Nephrology,
Apollo Children's Hospitals, Chennai

Organizing Team

DR. B R Nammalwar

Organising Chairperson

Dr. Sudha Ekambaram

Organising Secretary

Convenor

Dr. Thangavelu S

Dr. Saradha RC

Chairperson

Dr. Ng Kar Hui

Dr. W.D. Vindhya N

Dr. Selvakumar S

Dr. Syed Saimul Huque





www.nephkids.in

PROGRAMME

DAY 1

Saturday, September 26th, 2026
FLUID AND ELECTROLYTE WORKSHOP

TOPIC	MODERATOR	SPEAKERS	09:30-10:30	10:35-11:35	11:40-12:40
Hyponatremia	Dr Poovazhagi V	Dr Shyamala J Dr Sharada RC Dr Murali T	Group A	Group B	Group C
Fluid in special situations	Dr Thangavelu S	Dr Dhakshayani RV Dr Chidhambharam L Dr Naresh Kumar S	Group B	Group C	Group A
Metabolic Acidosis	Dr Rajakumar PS	Dr Ekambaranath T S Dr Karthikeyan M Dr Anita Tarigopula	Group C	Group A	Group B
LUNCH	12:45 - 13:30 hrs		13:30-14:25	14:30-15:25	15:30-16:25
Hypernatremia	Dr Anitha V P	Dr Venkateswari R Dr Priyavarthini V Dr Sunil Reddy	Group A	Group B	Group C
Hypocalcemia/ Hypomagnesemia	Dr Sridevi A Naaraayan	Dr LK Prem Kumar Dr Pooja G N Dr Pari Palanivelan	Group B	Group C	Group A
Hypo/ Hyperkalemia	Dr Gowrishankar NC	Dr Muthiah P Dr Deepika S Dr Priyadarshini M	Group C	Group A	Group B

FOREWORD

Homeostasis is the fundamental basis for survival for all living creatures. Body water, electrolytes, acid bases and divalent ions have explicit role in maintaining the homeostasis. A solid foundation in understanding the composition of body fluid, fluid requirements, and regulations; assessing and managing dehydration, understanding the physiologic functions of electrolytes, and managing electrolyte derangements is critical. Study of water, electrolytes, acid bases and divalent ions is an important and difficult area of in medicine. Students, Residents (why not teachers) study it intensely, yet too often come away without clarity and confidence they want and need. Why is this? Body water, electrolytes, acid-bases and divalent ions cannot be seen, neither felt, nor palpated or auscultated. It is a mist. The nearer you go, the faster it disappears. In physiology it is taught as a pure science. In clinical medicine it is presented as a group of symptoms with solutions as per guidelines. No life in it. There is diversity in the methodology of teaching of this subject. What is needed is to teach this subject as an applied science. Pediatricians, Pediatric Nephrologists and Pediatric Intensivists with unbridled and non-exhausting enthusiasm for teaching with a team of similar minded colleagues from other teaching Institutions have been organizing “Workshop on fluids, electrolytes, acid-base and divalent ions” for last seven years. Partly it has been fulfilling the lacunae. With this booklet it is much more. To the readers, treasure it as a ‘Rose’ from your beloved. Express your feeling. If you are thankful, contribute your thoughts and knowledge. Knowledge is an ocean and no one person can fathom it. Someday, I know it will be a Monograph on Body water, electrolytes, acid bases and divalent ions. A dream will come true.

- Dr. B.R. Nammalwar

Organising Chairperson, NEPHKIDS

Faculty- Workshop

Dr Poovazhagi V, MD, DCH, PhD

HOD and Professor, Department of PICU, Institute of Child Health and Hospital for Children, Chennai

Dr Shyamala J, MBBS, DCH, DNB (Pediatrics) Diplomate of National Board

Senior Consultant Neonologist & Pediatrician, Apollo First Med Hospital and Apollo Children's Hospital, Chennai

Dr Sharada R C, MBBS, DCH, DNB, FPEM, FSTEP

Senior Consultant, Dr. Mehta's Multispeciality Hospital, Chennai

Dr Murali T

Professor & HOD, Paediatric Emergency Department, Institute of Child Health and Hospital for Children, Chennai

Dr Thangavelu S, MBBS, DCH, MD, DNB, MRCP

Senior Consultant Pediatrician & Director, Department of Pediatrics, Dr. Mehta's Multispeciality Hospital, Chennai

Dr Dhakshayani R V, MD (Pediatrics)

Professor of Pediatrics, Government Tiruvannamalai Medical College

Dr Chidhambharam L, MBBS, MD, FNB Pediatric ICU

PICU Consultant, Apollo Children's Hospital, Chennai

Dr Naresh Kumar S MD (Pediatrics), Fellow (Pediatric Nephrology)

Associate Consultant – Pediatric Nephrology, Rainbow Children Hospital, Chennai

Dr. PS Rajakumar, MD DNB MRCPCH Fellowship in Paediatric Intensive Care (UK)

Professor of Paediatrics, Sri Ramachandra Institute of Higher Education & Research, Chennai

Dr Ekambaranath TS, MD (Ped), PICU

Assistant Professor, PICU, Stanley Medical College, Chennai

Dr Karthikeyan M, MD (Pediatrics)

Senior Assistant Professor, Kilpauk Medical College

Dr Anita Tarigopula, DNB Pediatrics, FPEM, STEP Fellowship (Paed Emergency and Trauma), PGDMLE (NLSIU, Bengaluru)

Consultant, Department of Pediatric Emergency, Apollo Children's Hospital, Chennai

Dr Venkateswari R, MBBS, DCH, DNB

Senior Consultant Pediatrician, Kanchi Kamakoti Childs Trust Hospital, Chennai

Dr Priyavarthini V, MD(Ped), DNB (Ped), FNB (PICU)

Consultant PICU, Apollo Children's Hospital, Chennai

Dr Sunil Reddy K G, FNB (Ped Nephrology)

Assistant Professor – CMC Vellore

Dr. Sridevi A Naaraayan, MD (Paediatrics), PGDEpi

Professor of Pediatrics, Institute of Child Health and Hospital for Children, Chennai

Dr Prem Kumar L K

Sr. Consultant, Deputy Head, Department of Pediatrics, Dr Mehta's Multispeciality Hospital India Pvt Ltd, Chennai

Dr Pooja GN, DNB (Paediatrics), Fellow (Pediatric Nephrology)

Assistant Professor and Consultant, Department of Paediatrics, Shridevi Institute of Medical Sciences and Research Hospital

Dr Pari Palanivelan

Assistant Professor, Sri Ramachandra Medical College, Chennai and Associate Consultant, Medical centre

Dr Muthiah P, MD IDPCCM, FPCC (Ped Critical Care)

Consultant Pediatric Intensivist, Dr. Mehta's Multispeciality Hospital, Chennai

Dr Deepika S MD (Pediatrics)

Consultant, Apollo Children Hospitals, Chennai

Dr Priyadarshini M MBBS, DNB (Paediatrics)

Senior Registrar, Apollo Children Hospitals, Chennai

HYPONATREMIA

Contributors: Dr. Poovazhagi V, Dr Shyamala J, Dr. Sharada RC, Dr. Murali T

Approach to Hyponatremia Essential facts

Hyponatremia is a common electrolyte imbalance in sick children which needs to be recognised early and treated appropriately. Hyponatremia develop only by two mechanisms – Gain of free water (most common) – could be obvious (edema) or clinically occult /subtle and Loss of sodium (less common) – mostly iatrogenic; rarely pathological. Hyponatremia is always associated with low serum osmolality. If associated with normal osmolality or high osmolality it is Pseudohyponatremia or spurious hyponatremia respectively. Clinical conditions like nephrotic syndrome can have pseudohyponatremia and conditions like DKA and mannitol therapy can have spurious hyponatremia. Since hyponatremia is not associated with bedside diagnostic clues one should anticipate hyponatremia based on the clinical setting and confirm with serum values. Sodium balance is always linked to water balance and correction should be appropriately undertaken.

Clinical presentation may include lethargy, Anorexia, nausea, emesis, malaise, lethargy, headache, Confusion, agitation, seizures, coma, decreased reflexes, Brainstem herniation and apnea (raised ICP), Hypothermia, Cheyne-Stokes respiration. In most instances, the underlying cause is evident from the clinical setting with careful clinical evaluation. Hence not many lab tests are required Sequential approach to hyponatremia involves the following steps.

Step 1 – Confirm hyponatremia by serum sodium levels below 135mEq/L.

Step 2 – Identify true hyponatremia. Confirm hyponatremia (rule out a lab error if the clinical setting is unlikely) - measure serum osmolality if available. Normal or high serum osmolality suggest pseudohyponatremia /spurious hyponatremia

Step 3 – Check if it is symptomatic hyponatremia or asymptomatic hyponatremia. Check if it is acute or chronic hyponatremia based on the evolution of hyponatremia in a clinical setting

Step 4 -- Identify possible mechanism and etiology based on the volume status.

Determine the volume status of the patient (clinical signs, serial weights if available). Determine water gain or sodium loss as the predominant mechanism by clinical setting. In rare instances if mechanism is not clear from clinical assessment, serial serum and urine osmolality along with a trial of fluid therapy will help in identifying the predominant mechanism.

Identify possible etiology. If clinical evidence of fluid overload (edema), identify the reason (renal / cardiac / hepatic). Clinical evaluation – History / examination . Relevant lab tests to confirm (urine routine, ECHO, USG, RFT, LFT etc as indicated) . If euvolemic, think of SIADH / polydipsia /endocrine causes. If hypovolemic, think of fluid loss (GI/renal/third space), diuretic use /cerebral salt wasting in appropriate settings

Step 5 – Therapy. Decide to bolus or not to bolus. If acute and symptomatic needs a bolus hypertonic saline 4-6ml/kg. Trial of treatment under monitoring of electrolytes and fluid status Water restriction in hypervolemic and euvolemic state. Replacing water / prevent losses in case of hypovolemic state

Remember to treat slowly, bolus with hypertonic saline only if indicated, follow up the sodium levels with labs over 6-8 hours.

Summary: Five steps for hyponatremia

1. Confirm hyponatremia
2. Identify if it is true or pseudo hyponatremia
3. Check if it is acute and symptomatic or chronic and asymptomatic
4. Assess the volume status to identify the etiology
5. Treat with bolus and maintenance therapy as indicated

Case Scenario 1:

1year 6 months old male child, weighing 10 kg

Presenting with Fever, vomiting & loose stools - 2 days, vomiting 5-6 episodes, loose stools - 8-10 episodes, managed as outpatient with ORS; child had not consumed ORS as advised, had been taking fruit juices and water. Mother noticed lethargy and decreased urine output

At ED lethargy, refusal of feeds and poor oral intake.

O/E child is lethargic, normal work of breathing, looking pale. Primary assessment revealed the following:

Airway stable

Breathing 46/mt, no retractions, no added sounds, chest rise adequate saturation at 94% in room air

Circulation: HR 160/mt, cool below ankle, peripheral pulses -feeble +++/+, CRT>3sec, BP-70/40mmHg.

Disability: Pain responsive

Exposure: febrile at 102F, dehydrated

Blood glucose is 80mg/dl, serum sodium is 129 mEq/l

Questions:

1. What is the physiological status of this child?
2. What is the likely cause of hyponatremia in this scenario?
3. What is the immediate management in the present status?
4. After the first bolus, his vitals were • HR-140/min, RR-40/min, +++/++, cool below ankles, CRT<3sec Bp 80/56mm Hg. How to proceed with treatment and monitoring?

Case Scenario 2:

5yr F, weighing 16kg with evaluation s/o TB meningitis was intubated for super refractory seizures. Started on ATT with steroids, multiple AED's, neuroprotective care and 3% saline - 1ml/kg/hr. Serum sodium -138meq/L. which was monitored and sodium was maintained between 140 to 145 meq/l. Hypertonic saline was stopped by 72hrs.

On Day6 - Serum sodium slowly drifted from 14 to 137 and 130meq/l in 12hours.

Child on sedation under mechanical ventilator, hemodynamic are stable, HR-100/min, Spo2-99%, BP-100/76 mmHg, UO-1.9ml/kg/hr.

1. What is the possible reason for hyponatremia?
2. How do you approach hyponatremia in this scenario?
3. What are the possible risk factors in this child?
4. How do you manage this child?

Case Scenario 3:

A 5-year-old female child, known case of Steroid dependent nephrotic, syndrome, on mycophenolate mofetil, presented with edema. Parents had stopped medication on their own for last 1 month. No e/o infection.

On examination, child irritable and anxious. Height - 108 cm (25-50 th centile), weight - 27 kg (97th centile), dry weight – 23 kg, BP - 114/84mmHg (95th centile), HR - 74 bpm, CFT < 2 sec, pulses well felt, RR - 33 / min. There is generalised anasarca. Respiratory system exam reveals reduced airtentry on the right side. There is free fluid in the abdomen. Other systems are normal.

Investigations done on admission: Urea 23, Creatinine 0.4, Sodium 128, Potassium 4.1, Chloride 102, Bicarbonate 22, S albumin 2.1, S. cholesterol 343, CBG 85, CBC Normal

Questions:

1. What is the metabolic abnormality in this child?
2. What further investigations will you order?
3. What are the steps in the management?

FLUID IN SPECIAL SITUATIONS

Contributors: Dr. Thangavelu S, Dr. Dhakshayani RV, Dr. Chidhambharam L, Dr. Naresh Kumar S

This chapter traces how pediatric maintenance fluid practice arrived at its present position, sets out the concepts required to prescribe fluid rationally, and then works through an approach to the situations in which standard maintenance and deficit formulae do not apply.

The common clinical uses of intravenous fluid are diarrhoeal dehydration with deficit correction, and maintenance fluid where there is no loss, as in bronchiolitis. The special situations, in which the usual rules must be modified, are diabetic ketoacidosis (hyperosmolar dehydration with an intracellular deficit), oliguric states (risk of fluid overload), polyuric states (risk of electrolyte imbalance), raised intracranial pressure (where electrolyte disturbance is a cause, a complication and a side effect of therapy), and the child with severe acute malnutrition (a failing sodium-potassium membrane pump).

Fluid Calculation

The modern paediatric maintenance prescription dates from the landmark paper of Holliday and Segar in 1957, which derived water requirement from calorie expenditure (Table 1). The fluid that followed from this reasoning was hypotonic, and Isolyte P, introduced in the same year, has an effective tonicity of about 0.15.

Table 1. Holliday-Segar maintenance requirement

Weight	24-hour maintenance fluid	Calories expended
1–10 kg	100 mL/kg	100 cal/kg/day
11–20 kg	1000 mL + 50 mL/kg for each kg above 10 kg	1000 cal + 50 cal/kg for each kg above 10 kg
> 20 kg	1500 mL + 20 mL/kg for each kg above 20 kg	1500 cal + 20 cal/kg for each kg above 20 kg

Water requirement was calculated from calorie requirement, and the calorie figures were those of healthy children.

Why we moved away from hypotonic fluids

Dunn and Butt, reporting on extreme sodium derangement in a paediatric inpatient population, found that among children with sodium disturbances 57% developed the abnormality after hospital admission, and that water overload accounted for 38% of cases of hyponatremia. Moritz and Ayus, in their case for isotonic saline, collected more than 50 reported cases of neurological death and morbidity attributable to the unsafe practice of hypotonic maintenance fluid, ranging from 0.2% to 0.45% saline. The underlying mechanism is increased vasopressin secretion with impaired free water excretion, producing hospitalisation-associated hyponatremia and encephalopathy.

In December 2018 the American Academy of Pediatrics recommended that patients from 28 days to 18 years of age requiring maintenance intravenous fluid should receive isotonic solutions with appropriate potassium chloride and dextrose, because these significantly decrease the risk of hyponatremia.

Key reasons for restricting maintenance fluid in the sick child

- Stress-induced ADH release. Surgery, pain, trauma, nausea, sepsis and infection all trigger non-osmotic release of antidiuretic hormone.
- Overestimation of need. The Holliday-Segar formula was derived from the calorie expenditure of healthy children. Sick or hospitalised children have reduced endogenous metabolism and lower actual calorie and fluid needs.
- Risk of hyponatraemia. Because the kidney retains water when ADH is elevated, free water given as hypotonic fluid produces hospital-acquired hyponatraemia.
- The “less is more” approach. Restricting to a fraction such as 70% of calculated maintenance matches the reduced output state of acute illness.
- Risk of hyponatraemia. Because the kidney retains water when ADH is elevated, free water given as hypotonic fluid produces hospital-acquired hyponatraemia.
- The “less is more” approach. Restricting to a fraction such as 70% of calculated maintenance matches the reduced output state of acute illness.

Concepts: Tonicity and the types of intravenous fluid

Osmolality is the number of osmoles of solute per kilogram of solvent. Osmolarity is the number of osmoles of solute per litre of solution. Tonicity is effective osmolality, and is the sum of the concentrations of those solutes which can exert an osmotic force across the cell membrane — principally sodium. Urea crosses the membrane freely and

therefore, contributes to measured osmolality but not to tonicity. This distinction matters at the bedside: a fluid may have an adequate osmolality on the label and still behave as free water once the dextrose in it has been metabolised.

Hypotonic fluids contain more salt-free water, and effective free water is equivalent to salt-free water in this context. The typical maintenance fluid for children is 5% dextrose in 0.9% saline with potassium 20 mmol/L in each 1000 mL. Table 2 sets out the composition of the commonly used solutions.

Table 2. Composition of commonly used intravenous fluids (per litre)

Fluid	Na ⁺	K ⁺	Cl ⁻	Buffer	Dextrose	Osmolarity
0.9% sodium chloride	154	—	154	—	—	308
Ringer lactate	130	4	109	Lactate 28	—	273
Balanced salt solution (acetate-gluconate type)	140	5	98	Acetate 27, gluconate 23	—	294
5% dextrose in 0.9% saline	154	—	154	—	50 g	560
5% dextrose in 0.45% saline	77	—	77	—	50 g	406
Isolyte P	25	20	22	Acetate 23	50 g	—
5% dextrose in water	—	—	—	—	50 g	252
3% sodium chloride	513	—	513	—	—	1027

Concentrations are in mmol/L unless stated. Values vary slightly between manufacturers; the label should be checked.

Intravenous fluid is a drug

Fluid should be prescribed with the same attention to dose, composition and rate that is given to any other drug. Three questions must be answered before the bag is hung: what fluid, how much, and at what rate of flow. The discipline is conveniently remembered as the four Ds — drug, dose, duration and de-escalation. The choice of drug covers crystalloid versus colloid, saline versus balanced electrolyte solution, the intravenous versus the oral route, and appropriate versus inappropriate indication. The dose must specify the type of fluid and the compartment it is intended to fill, since resuscitation targets the intravascular space while cellular dehydration requires quite different handling.

The ROSE model and the four phases of fluid therapy

The ROSE conceptual model divides fluid therapy into four sequential phases, each with a different goal and a different expected fluid balance (Table 3). Recognising which phase a child is in prevents the common error of continuing resuscitation-phase prescribing into the stabilisation phase, which is how fluid overload accumulates.

Table 3. The ROSE model

Phase	Stage	Objective	Fluid balance
R	Resuscitation	Correct life-threatening shock after trauma, sepsis, burns or pancreatitis. Fluid must be given to save life	Positive
O	Optimisation	Organ rescue; optimise perfusion while avoiding fluid overload	Neutral
S	Stabilisation	Organ support; maintain homeostasis once the patient has stabilised	Neutral to negative
E	Evacuation	Active fluid removal; resolve fluid overload while avoiding hypoperfusion, focusing on organ recovery	Negative

Adapted from Monteiro JN, Goraksha SU. J Neuroanaesthesiol Crit Care 2017; 4:10–16, and Malbrain et al. Ann Intensive Care 2020.

Fluid overload is defined by a cut-off of 10% fluid accumulation, and beyond this threshold it is associated with organ dysfunction and worse outcomes. Percentage fluid overload is calculated as cumulative fluid input minus output, divided by admission weight, multiplied by 100. The distinction that matters clinically is between fluid in the right place and fluid in the wrong place; the same positive balance that restores perfusion in the resuscitation phase becomes interstitial oedema, impaired gas exchange and delayed organ recovery if it persists.

Uses of intravenous fluid

Every intravenous fluid prescription serves one of four purposes, and the purpose determines the fluid chosen. A bolus for shock and a replacement for concurrent loss both require isotonic fluid — normal saline, Ringer lactate or a balanced solution. Deficit correction requires isotonic fluid in the initial phase, with the remainder of the deficit

and the maintenance requirement given together. Maintenance fluid, in a child with no pathological loss, requires an isotonic solution with dextrose and potassium, given at a restricted fraction of the calculated requirement for the reasons set out.

Approach to dehydration correction

The first step is to estimate the deficit from the clinical severity (Table 4), and the second is to decide whether the child is in shock. A child in shock is resuscitated first, and deficit correction begins only after perfusion is restored.

Table 4. Fluid deficit by severity of dehydration

Mild (3%)	Moderate (6%)	Severe (9%)
30–40 mL/kg	50–70 mL/kg	100 mL/kg

Mild dehydration — worked example for weight 10 kg

Oral rehydration solution is preferred. If oral intake is not possible, intravenous fluid may be given by either of two schedules. The first gives normal saline or Ringer lactate 40 mL/kg (400 mL) from 0 to 6 hours, followed by maintenance fluid 1000 mL over the remaining 7 to 24 hours. The second gives a single volume of 1400 mL of 5% dextrose in 0.9% saline with potassium chloride 20 mmol/L over 24 hours, delivered as 700 mL over the first 8 hours and 700 mL over the next 16 hours.

Moderate dehydration — worked example for weight 10 kg

The deficit is 70 mL/kg, giving 700 mL, and maintenance is 1000 mL, so the total requirement is 1700 mL. Correction is in three phases. From 0 to 1 hour, rapid restoration of intravascular volume with 200 mL of normal saline or Ringer lactate over one hour. The remaining 1500 mL is then given over 24 hours as 5% dextrose in 0.9% saline with potassium 20 mmol/L: 750 mL over the first 8 hours, at 94 mL/hour, and 750 mL over the next 16 hours, at 47 mL/hour. Ongoing losses are replaced in addition, volume for volume.

Severe dehydration — worked example for weight 10 kg

Based on WHO criteria, an infant receives 300 mL from 0 to 1 hour and 700 mL from 1 to 6 hours. Beyond one year of age the same volumes are given faster: 300 mL in the first half hour and 700 mL from half an hour to 3 hours. Where pediatric intensive care facilities are available, shock is corrected first with normal saline or Ringer lactate 20

mL/kg repeated every 5 to 20 minutes until perfusion is restored, with reassessment after each bolus; deficit correction and maintenance fluid follow once shock has been corrected.

Beyond clinical assessment: monitoring the fluid prescription

Fluid therapy is titrated, not calculated once. Assessment draws on history, examination, bedside measurement and laboratory data, and increasingly on point-of-care ultrasound.

Assessment and monitoring

- History: fluid losses — vomiting, diarrhoea, polyuria — and urine output.
- Examination: mucosae, skin, anterior fontanelle, alertness, and circulatory assessment including heart rate, pulse volume, blood pressure, effortless tachypnoea and level of consciousness.
- Bedside: weight, which is particularly useful when the pre-illness weight is known, and a strict intake and output chart.
- Laboratory: serum sodium, potassium, bicarbonate and chloride, renal function, and serum and urine osmolality.

Point-of-care ultrasound

Longitudinal imaging of the inferior vena cava in expiration and inspiration allows collapsibility to be assessed as a guide to intravascular filling. Lung ultrasound complements this by detecting the interstitial oedema of fluid overload before it is clinically apparent.

Key ultrasound findings in fluid assessment

- B-lines (comet tails): laser-like vertical hyperechoic artefacts arising from the pleural line. Multiple or confluent B-lines in symmetrical zones across both hemithoraces indicate interstitial pulmonary oedema.
- Pleural effusion: a dark, anechoic fluid collection above the diaphragm, often identifiable at volumes as low as 20 mL.

- Normal pleural line and lung sliding: preserved lung sliding with a regular pleural line helps to differentiate fluid-overload pulmonary oedema from primary lung pathology such as ARDS or pneumonia.
- Inferior vena cava collapsibility: assessed longitudinally in expiration and inspiration as a marker of intravascular volume; interpreted alongside the clinical picture rather than in isolation.

Conclusion

Fluid is a drug. It has a dose, a composition, a duration and an exit strategy. Isotonic solutions with dextrose and potassium, given at a restricted fraction of the Holliday-Segar calculation, are the default for maintenance in the sick child. Deficit correction is staged, and shock is corrected before deficit. In each special situation, one assumption behind the standard calculation fails, and the approach consists of identifying which one,

Approach

- Ask the four questions before prescribing: what fluid, how much, at what rate, and for how long.
- Identify the purpose: bolus for shock, deficit correction, replacement of concurrent loss, or maintenance. Each has a different answer.
- Use isotonic fluid for maintenance, with dextrose and potassium, at a restricted fraction of calculated requirement. Hypotonic maintenance fluid causes hospital-acquired hyponatremia.
- Correct shock before correcting deficit.
- Identify the ROSE phase and prescribe for that phase; the resuscitation prescription must not persist into stabilisation.
- Track cumulative balance. Fluid accumulation beyond 10% of body weight is associated with organ dysfunction and worse outcomes.
- In ketoacidosis, clinical signs underestimate the deficit and the deficit is intracellular: replace slowly, add potassium early, avoid bicarbonate.
- In oliguria, restrict to insensible losses plus measured output, and avoid potassium-containing fluid.

- In polyuria, measure the urine volume and its electrolytes and replace what was actually lost.
- In raised intracranial pressure, use isotonic fluid only, keep sodium normal to high, and never allow hyponatremia.
- In severe acute malnutrition, rehydrate orally wherever possible; reserve intravenous fluid for shock with impaired consciousness and stop if the pulse and respiratory rate rise.

Case Scenario 1:

- 2 years old female child presented to the OPD with H/O loose stools of 3 days duration. Mother had fed the child with ajwain water (omam) for symptoms. Child had persistent vomiting since morning. Child had been admitted for 2 diarrhoeal episodes earlier.
- Child had a protuberant abdomen, lustreless hair and visible wasting of the gluteal muscles. The child weighed 8 kgs and measured 84 cms in length Her MUAC was 11 cms.
- Child appeared lethargic & hadn't consumed feeds since morning.
- Rapid cardiopulmonary cerebral assessment revealed the baby in a verbal responsive mental status. Her RR was 48/min, had no intercostal indrawing, HR -130/min with peripheries cool below ankles, prolonged CRT > 3 seconds and +++/+ pulses.

Questions:

1. How will you manage this child at the ED, at this point of first contact?
2. What is the Fluid Management Plan?

Case Scenario 2:

A 4-year-old boy is brought to the hospital due to poor growth, muscle cramps, and excessive thirst. His mother reports that he urinates frequently and often wakes at night to drink water. He also has a craving for salty food.

EXAMINATION:

Dull and dehydrated; Tongue- Dry; Sunken eyes; Skin turgor- reduced; Failure to thrive; Height: 90 cm (Z score -3.18); Weight: 10.2 kg (Z score -3.8); BMI: 12.59 (Z score -2.48)

VITALS:

HR: 138/min, Normal to Low volume pulse; BP: 88/56 mmHg; RR: 18/min; CRT < 3 sec;
Warm peripheries

INVESTIGATIONS:

- **Blood gas & serum:** pH 7.48; HCO_3^- 30 mmol/L; pCO_2 48 mmHg; Na 130 mmol/L; K 2.8 mmol/L; Cl 92 mmol/L; Urea 20 mg/dL; Creatinine 0.3 mg/dL; Calcium: 9.1 mg/dL; Phosphorous: 4.5 mg/dL; Magnesium: 2.2 mg/dL
- **Urine:** Urine pH 5.5; Urine Na 87 mmol/L; Urine Cl 136.2 mmol/L; Urine K 38 mmol/L; Urine Ca/Cr 0.6; Urinalysis: normal
- **Imaging: USG:** Normal sized kidneys with normal echotexture and CMD differentiation, Bilateral medullary nephrocalcinosis.

Questions:

1. What is the most likely diagnosis?
2. What is the treatment approach and fluid plan for this child?

Case Scenario 3:

A 10-year-old girl weighing 30 kg has been brought to the emergency department with a history of frequent micturition, lassitude, excessive thirst for the past week, vomiting, and fast breathing for one day. There was no fever or burning micturition. She has been previously well and developmentally normal with no other significant personal or family history.

On examination, she is conscious and oriented with a respiratory rate of 40/min, no retractions, bilateral equal air entry with no adventitious sounds, and SpO₂ of 98% in room air. Her heart rate is 120/min, BP 102/70 (82) mm Hg, extremities are warm, distal pulses are felt, and capillary refill time is 2 seconds. She has sunken eyes, dry mucous membranes, and reduced skin turgor. Her capillary blood glucose done on arrival is 470 mg%. Urine ketones were 4+. Arterial blood gas values are as follows: pH 7.15, pCO_2 26 mm Hg, PO₂ 96 mm Hg, HCO_3^- 14 meq/L, BE -12, lactate 2.2 mmol/L, Na 132 meq/L, K 3.5 meq/L, Cl 102 meq/L.

Questions

1. What is the clinical diagnosis?
2. Assess the fluid status and draft a fluid prescription for this child.

METABOLIC ACIDOSIS

Contributors: Dr. Rajakumar PS, Dr. Ekambaranath T S, Dr. Karthikeyan M, Dr. Anita Tarigpula

Analysis with ABG (Measured values - pH, paco₂, paO₂: Calculated values – Hco₃, BE, SBE)

1. **History & Physical:** Gives an idea of what acid base disorder might be present
2. **Look at the pH** (pH < 7.35 acidosis; pH > 7.45 alkalosis)
 - pH may be normal in the presence of a mixed acid base disorder, particularly if other parameters of the ABG (PCo₂, HCo₃) are abnormal.
3. **Look at PCO₂, HCO₃** - What is the acid base process (alkalosis vs acidosis) leading to the abnormal pH? Are both values normal or abnormal?
 - Respiratory disorder will alter PaCO₂
High CO₂ = respiratory acidosis; Low CO₂ = respiratory alkalosis
 - Metabolic disorder will alter HCO₃
HCO₃ < 22 = metabolic acidosis; HCO₃ > 26 = metabolic alkalosis
 - One abnormal value will be the initial change (side of the pH change) and the other will be the compensatory response.
 - The direction of compensatory variable is on the same side as the primary variable.
 - Remember compensation never over shoots the pH.
4. If metabolic acidosis, **calculate Anion Gap** (AG = Na – (Cl+HCO₃)).
 - If elevated (> 12), High Anion Gap Metabolic Acidosis (HAGMA) - acidosis is due to addition of (previously unmeasured) acids like Diabetic Ketoacidosis, Uremia, Lactic acidosis, Toxins, organic acids) (MUD PILES)
 - If anion gap is normal, then acidosis likely due to loss of HCO₃ or addition of Chloride (Diarrhea, RTA, Excess Saline/ 3% Saline)
 - In hypoalbuminemia, AG will lower than 12. So in hypoalbuminemia, if AG is 12, it is actually HAGMA

5. If metabolic acidosis, **calculate estimated CO₂** to determine **any additional respiratory acidosis/ alkalosis.**

Expected PaCO₂ = {(1.5 X [HCO₃]) + 8} ± 2 (**Winter's Formula**)

If measured PaCO₂ is higher than expected PaCO₂, a concomitant respiratory acidosis is present.

If measured PaCO₂ is lower than expected PaCO₂, a concomitant respiratory alkalosis.

6. If high anion gap metabolic acidosis, then calculate the **Delta-Ratio (Δ/Δ)** to determine any **additional normal anion metabolic acidosis or metabolic alkalosis.**

- Delta Ratio = Delta Anion Gap/ Delta Bicarbonate Gap (Note – Anion before Bicarbonate)
- Delta Anion Gap = Anion Gap – 12; Delta Bicarbonate Gap = 24 - Bicarbonate
- If Delta ratio between 1 and 2, then only HAGMA
- If < 1, then there is a coexistent NAGMA (NAGMA + HAGMA)
- If > 2, then there is a coexistent Metabolic alkalosis present

Additional Clues:

1. **If respiratory acidosis**, check change in HCO₃ to determine additional metabolic acidosis/ alkalosis

Extent of compensation is given by:

10 mmHg change in PaCO₂ – Bicarbonate changes by 1 (Acute)

10 mmHg change in PaCO₂ – Bicarbonate changes by 4 (Chronic)

If HCO₃ is less than expected, there is additional metabolic acidosis.

2. Clues for mixed disorders:

- a) CO₂ and HCO₃ move in opposite direction (usually in compensation, they move in same direction)
- b) Normal pH, but abnormal CO₂ and HCO₃ (likely metabolic respiratory disorders in opposite direction – one acidotic process and one alkalotic process)
- c) Grossly abnormal pH, but CO₂ and HCO₃ in themselves are only slightly abnormal (Both respiratory and metabolic disorder in same direction eg Both respiratory and metabolic acidosis)

3. If **normal anion gap metabolic acidosis** and cause is unknown, then calculate the **Urine Anion Gap (UAG)**.
 - Urine Anion Gap = Urine (Na + K) - Cl
 - In RTA, UAG is positive. In diarrhea and other causes of metabolic acidosis, the UAG is negative. (neGUTive in diarrhea)
4. If **high anion gap metabolic acidosis and cause is unknown, then calculate Osmolar Gap** (Measured Osmolarity - Calculated Osmolarity) to identify undetected osmotically active acids like acids in Ethyl alcohol poisoning (osmolar gap > 10).
5. **Management of metabolic acidosis** – Simple rule is to treat the underlying cause. Bicarbonate is not the primary treatment. Bicarbonate therapy is reserved in select cases with severe acidosis pH < 6.9 which can cause myocardial depression or select causes like renal tubular acidosis.

Case Scenario 1:

A 11 months baby was brought to the ED with history of loose stools (10-15 episodes of loose watery stools) and vomiting (3-4 episodes/day). There is history of poor feeding for the past 6 hours. On receiving in ED, child has sunken eyes, dry mucosa, increased skin turgor and CRT: 4sec. Vitals recorded were HR: 110/min, RR: 30/min, BP: 74/50mmHg, SpO₂: 96% RA. Peripheries are cold with feeble pulses. VBG done shows pH: 7.29, pCO₂: 38, pO₂: 100, HCO₃: 18, BE: -6, Cl: 88, Na⁺: 121, K⁺: 2.8, Glu 80, Lac: 2.4

Questions:

1. Interpret the VBG.
2. What are the causes of NAGMA?
3. What is the fluid of choice for this child to correct dehydration?
4. What happens to Anion Gap in Hypoalbuminemia?

Case Scenario 2:

A 9-month girl is brought to the ER for lethargy and breathing difficulty since morning. There was no history of fever, or loose stools. She had vomited small quantity 2 times. She was born of non-consanguineous parents, with normal neonatal period and development so far. She had received only the first of the primary series of immunisation as parents were naturopathy practitioners. She was not given any native medicines. She was weaned to semisolids at 6 months and mother continued to breast feed. Child had consumed a lot of chicken for the first time the previous day.

In ER, initial assessment was that of a sick child who was pale and had acidotic respirations. She had a HR 162 / min, pulse volume was low, CFT 3 secs, cool below ankles, BP 84/60 mmHg. RR was 30/min, deep sighing, good chest rise, no added sounds, SpO₂ 98% in room air. She was pain responsive, with pupils equal and reacting to light. CBG 75 mg/dl. AF depressed, dry oral mucosa, normal skin turgor. Mother had noticed urine output the previous night.

ABG

pH 7.08, pCO₂ 13, pO₂ 67, HCO₃ 6, BE -26

Na 137, K 4.1, Cl 108, Ca 1.37 Lactate 1.8, Glucose 66

Hb 12.5, COHb 1.9, MetHb 0.9

Questions:

1. Interpret the ABG
2. Calculate the anion gap and delta gap
3. How will you manage?

Case Scenario 3:

An 8-year-old girl(20kg) was brought the ED with history of fever & vomiting of 2 days duration. She has been reported by the mother to be tired during this period. On initial assessment she is noticed to have RR: 40/min, HR : 130/min, BP: 90/60mmHg, SpO₂: 98%RA. Her peripheral pulses are feeble with a CRT of 3 seconds. Past medical history reveals a weight loss of 3kgs over 4weeks duration.

VBG done reveals pH 7.244, pCO₂: 24, HCO₃: 10.4, BE: -18.9, Na⁺128, K⁺: 3.3, Cl:103, Glu: 470.

Questions:

1. Interpret the VBG
2. What do you think is the reason for acidosis?
3. Comment on the sodium level in relation to glucose level
4. What is your first step here towards correction of acidosis?

Case Scenario 4:

2-year-old toddler presents with fever, tachycardia and tachypnea 4 hours after being found playing with Grandmother's medicine cabinet.

pH 7.4	PaCO ₂ 25 mmHg	PaO ₂ 105 mmHg	Bicarbonate 15.3 mmol/l	BE - 6
Na 135 mmol/L	K 4.1 mmol/L	Cl 95 mmol/L	Glucose 6.3 mmol/L	FiO ₂ 0.21

Questions:

1. Interpret the ABG
2. Calculate the anion gap and delta gap
3. What is expected CO₂? Is there additional respiratory component?
4. How will you manage?

Case Scenario 5:

4-year-old post resuscitation after cardiac arrest from drowning episode.

pH 7.10	PaCO ₂ 50 mmHg	PaO ₂ 105 mmHg	Bicarbonate 10.3 mmol/L	FiO ₂ 0.5
---------	---------------------------	---------------------------	-------------------------	----------------------

Questions:

1. Interpret the ABG
2. Why is the pH so bad despite mild rise in CO₂?
3. How will you manage?

Case Scenario 6:

A 2-year-old boy is found lethargic with bluish discoloration of lips. Examination reveals tachycardia and tachypnea, HR 145/min, RR 44/min, pulses well felt, CFT normal, BP normal, no lung signs, SpO₂ 86% in room air, does not improve with O₂ administration through NRM.

No h/o cardiac disease; no murmurs heard. Mother is on Dapsone for a skin condition.

ABG sample was drawn; there was a doubt if it was a venous sample.

pH 7.19, pCO₂ 26, pO₂ 162, HCO₃ 14, BE -9.1, Na 142, K 5.2, Ca 1.19, Cl 109, Hb 12.5, Hct 40, sO₂

99.7%, FO₂HB 63.2 %, FCOHB 0.8%, FMetHB 35.5%, Lactate 4, Glucose 78

Questions

1. What is the cause of acidosis in this child?
2. Calculate delta gap
3. How do we treat the acidosis?

Case Scenario 7:

An infant with IEM with repeated vomiting & dehydration. Drowsy.

pH	7.30
pCO ₂	40 mmHg
pO ₂	86 mmHg
Na	145
Cl	100

Questions

1. What is the cause of acidosis in this child?
2. Calculate delta gap, delta ratio and what does that indicate?
3. How do we treat the acidosis?

Case Scenario 8:

An infant with IEM develops diarrhea & vomiting

pH	7.44
pCO ₂	12 mmHg
pO ₂	88 mmHg
Na	136
Cl	106
HCO ₃	8 mmol/L

Questions:

1. What is the cause of acidosis in this child?
2. Calculate delta gap, delta ratio and what does that indicate?
3. How do we treat the acidosis?

Case Scenario 9:

5-year-old child with acute exacerbation of asthma.

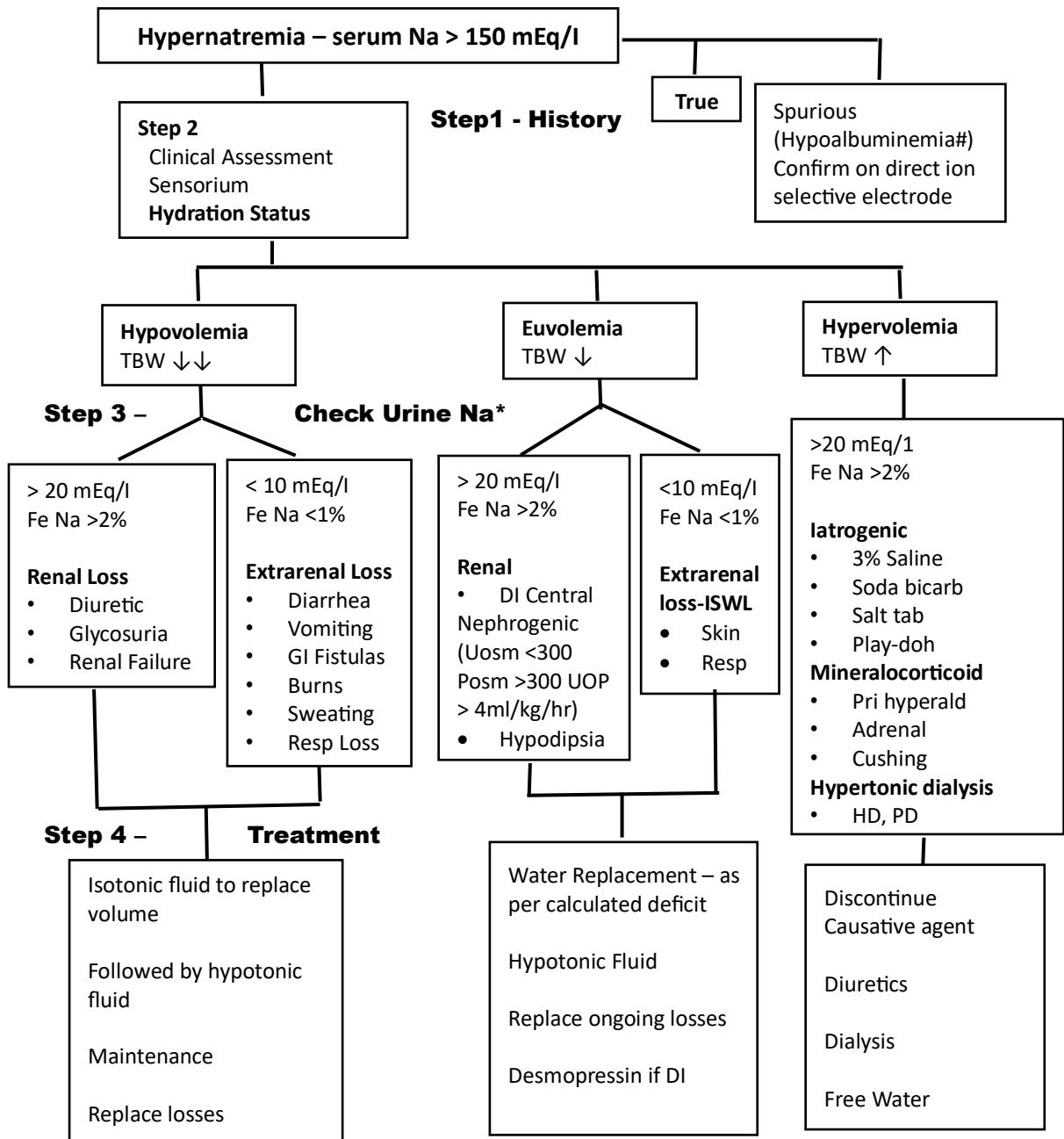
pH 7.22	PaCO ₂ 56 mmHg	PaO ₂ 63 mmHg	Bicarbonate 18 mmol/l	BE -6
Na 138 mmol/L	K 4.6 mmol/L	Cl 102 mmol/L	Lactate 4.6 mmol/L	FiO ₂ 0.21

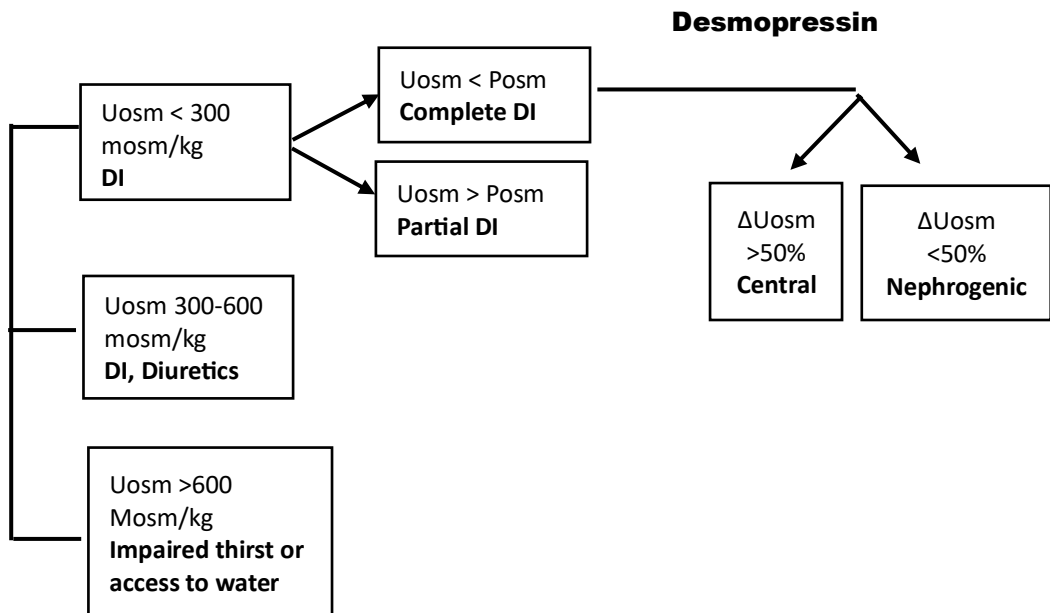
Questions

1. What is the cause of acidosis in this child?
2. Calculate expected change in HCO₃?
3. Calculate delta gap, delta ratio and what does that indicate?
4. How do we treat the acidosis?

HYPERNATREMIA

Contributors: Dr. Anitha VP, Dr. Venkateswari R, Dr. Priyavarthini V, Dr. Sunil Reddy



Step 5 – Check Urine Osmolality

* Useful to do UEC, calcium, magnesium, phosphate and glucose. These may need concurrent management

In the presence of hypoalbuminaemia (albumin <30 g/L), a blood gas sodium level is more reliable

* Initial paired serum and urine sodium, creatinine and osmolality is ideal, but if results will be delayed, a urine dipstick for specific gravity will give an indication of urinary concentration and treatment should not be delayed

Acute hypernatremia, of < 48 hours duration and symptomatic, is managed to achieve a rise in Na of 3-5 mEq/l immediately with a rise of 10-12 mEq/l in 24 hours

Chronic hypernatremia, of > 48 hours duration is corrected more gradually, with a rate of rise of not more than 0.5 per hour or 8-10 mEq/l per day.

Free water deficit in milliliters = Current total body water x [(current plasma Na/140) - 1)

Free water deficit in milliliters = (4 mL/kg) x (weight in kg) x (desired change in plasma Na)

Adroque Madias formula predicts the change in Na achieved with 1 litre of chosen fluid

$$\text{Change in serum (Na}^+) = \frac{\text{infusate (Na}^+) + \text{infusate (K}^+) - \text{serum (Na}^+)}{\text{total body water} + 1}$$

Case Scenario 1:

5 months old (birth weight: 2.8kg), formula fed infant, was admitted with complaints of passage of watery stools with recurrent non bilious vomiting for 2 days, decreased activity, poor feeding and decreased urine output for 1 day.

On examination, infant was lethargic with sunken eyes. His perfusion and blood pressure were normal. His weight was 5.5 kg.

Labs showed the following:

Urea: 77mg/dl, Creatinine: 0.78mg/dl, Na: 158 mEq/dl, K: 3.5 mEq/dl, Cl: 110 mEq/dl, HCO₃: 8 mEq/dl.

Questions:

1. What is the most likely cause of hypernatremia in this infant?
2. Mention one important clinical parameter in the assessment of this infant.
3. Mention the diagnostic lab parameter useful in the management of this infant.
4. Steps in the management of this infant?

Case Scenario 2:

3yr old male child diagnosed in antenatal period with posterior urethral valve and was fulgurated. He is constant follow up and was started on CIC and night drain. His last follow up showed Wt 7kg Ht 86cm, creatinine 2.6 mg/dl, urea 85 mg/dl. Currently he has acute diarrheal disease and he was advised to give ORS after every loose stool. On day 2 of illness, he developed fever with chills and oliguria and few hours later had generalized seizures lasting for 10 mins and he was brought to casualty. Clinically he had no dehydration, febrile to touch and had tachycardia with high BP (above 95th+12) mm of Hg. He was immediately given antiseizure medications, intubated and other necessary

conservative measures. His initial blood investigations showed Hb of 5, TC 22000, N89, platelets of 1.5L, creatinine 7mg/dl, urea 180mg/dl, Na 178meq/l, k 4.8meq/l, Hco3 12 meq/l.

Questions:

1. What are the possibilities that can be considered here?
2. What are the initial steps of management?
3. When do you consider peritoneal dialysis?

Case Scenario 3:

A 10-year-old boy weighing 30 kg is undergoing treatment for severe traumatic brain injury after a road traffic accident in the PICU (day 5 of PICU stay). He had severe cerebral edema and underwent decompressive craniectomy on day 2 of admission. There is ongoing discussion regarding poor neuroprognosis given the CT Brain and clinical features such as non-reactive pupils (4 mm each). He has no response to pain, absent deep tendon reflexes. He is invasively ventilated and on ketamine and midazolam infusions. His vitals are as follows:

HR 150/min, BP 86/44 (55), SpO2 98% (FiO2 30%.

His peripheral perfusion is poor and CVP is 2 mmHg.

He is on 3% saline infusion at 30 ml/hr. A routine review of charts shows increasing sodium trends over the past 24 hours as follows:

Na (in mmol/L) every 6 hours: 145 – 150 – 153 – 164

Other labs: K 4 mmol/L, HCO3 – 24 mmol/L, Cl- 110 mmol/L, Urea 30 mg/dl, Creatinine 0.6 mg/dl

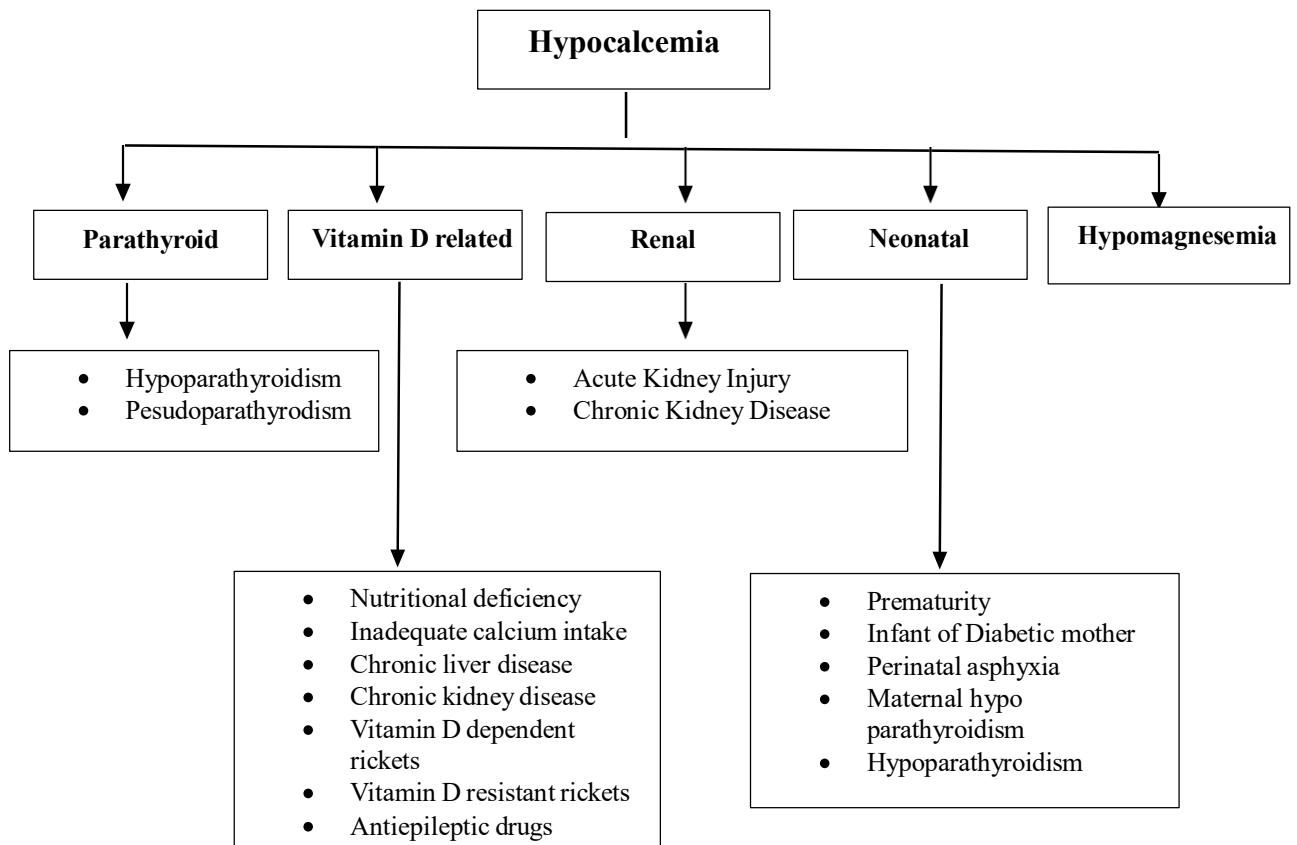
Questions:

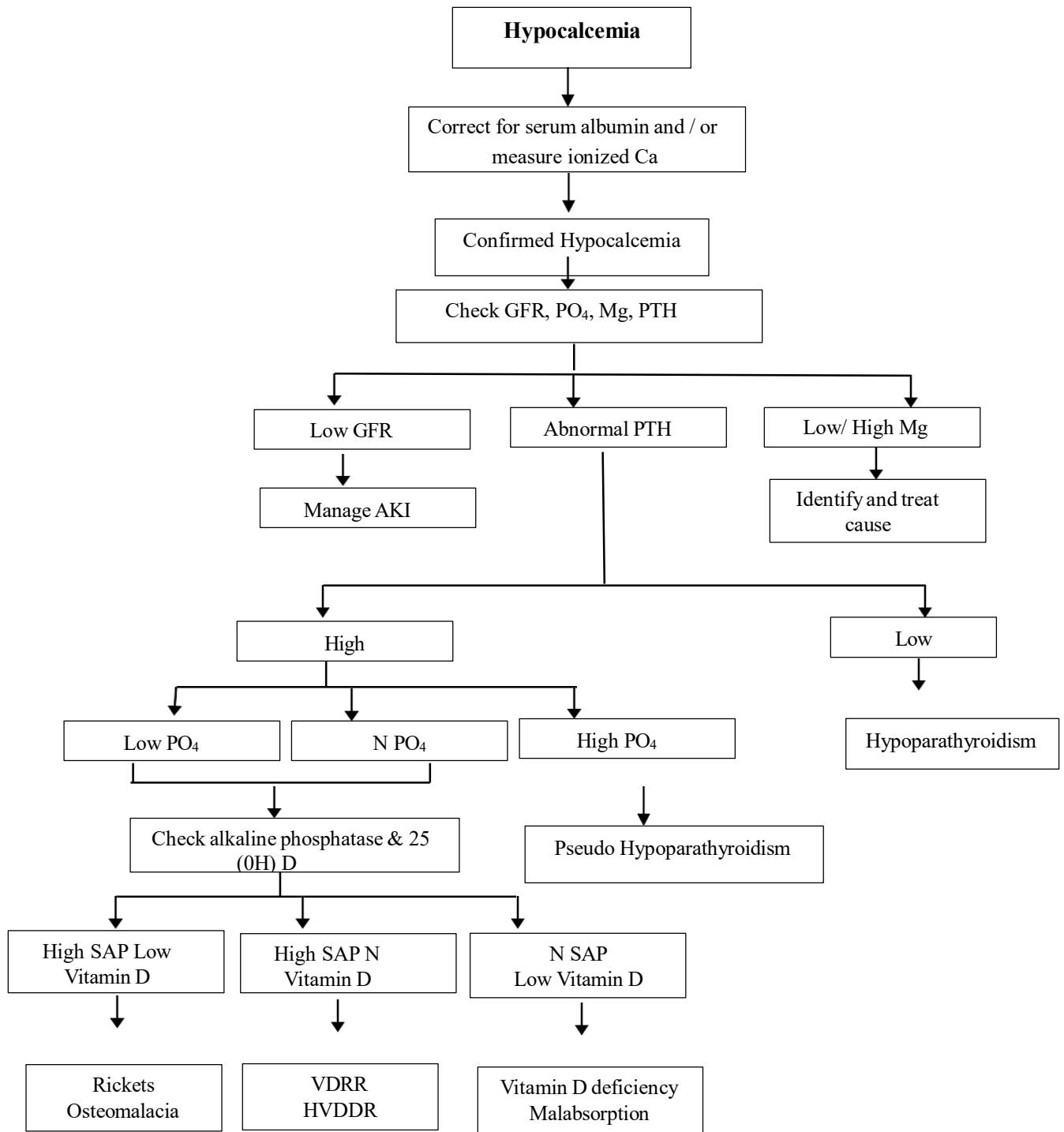
1. Are you concerned about the electrolyte trends?
2. Could 3% saline be the cause of this rising sodium trends?
3. What is the one clinical parameter that will guide you to the possible diagnosis?
4. How will you approach this situation?

HYPOCALCEMIA

Contributors: Dr. Sridevi A Naaraayan, Dr. LK Prem Kumar, Dr. Pooja, Dr. Pari Palanivelan

- Calcium is essential for normal neuromuscular function, cardiac conduction, bone mineralization, blood coagulation and intracellular signalling.
- The total body calcium content is 1000 to 1200 grams 99% of which is stored in bones as calcium phosphate complexes/hydroxyapatite.
- The rest 1% is present in intra and extracellular fluid. In ECF, 40% is bound to albumin (0.8mg/dl of Ca per 1 gm/dl of albumin), 15% is bound to organic and inorganic anions including phosphate, bicarbonate and lactate and 45% is in physiologically active ionized (free) form.
- Serum calcium is tightly regulated by the interaction of parathyroid hormone (PTH), calcitonin and vitamin D; ECF calcium level is maintained by coordinated action of the kidneys, gastrointestinal tract and bone.
- Normal serum calcium: 8.8-10.4mg/dl; Ionized calcium 4.4-5.4 mg/dl.
- Definition of hypocalcemia
 - Preterm newborns <7 mg/dl
 - Term newborns <8mg/dl
 - Children < 8.8 mg/dl; Ionised calcium < 4.4mg/dl or 1.1 mmol/L
- Clinical manifestations vary from asymptomatic biochemical abnormalities to tetany, seizures, laryngospasm, cardiac dysfunction (prolonged QT interval) and life-threatening arrhythmias.
- Corrected calcium (mg/dl) = total calcium (mg/dl) + 0.8 [4-albumin (g/dl)]

CAUSES OF HYPOCALCEMIA

APPROACH TO HYPOCALCEMIA

Treatment of hypocalcemia:

- Intravenous calcium
 - Indications – symptomatic and ionized calcium <0.8 mmol/L
 - Preparations – calcium gluconate and calcium chloride
 - Calcium gluconate – 10% - contains 9mg/ml or 0.46mEq
 - Calcium chloride – 10% - contains 27 mg/ml or 1.36mEq
 - Dose – 5-20 mg/kg of elemental calcium (0.2-0.7 ml/kg of Cacl or 0.5-2.0ml/kg of Ca gluconate) over 10 to 20 minutes.
 - Caution – Hyperosmolar; hence extravasation results in tissue necrosis Risk of precipitation if given along with phosphate and bicarbonate.
- Oral calcium in the dose of 50-100mg/kg/day in 4-5 divided doses along with vitamin D in deficient children. Daily regimen of vitamin D is preferred in the dose of 2000 to 3000 IU per day.

Case Scenario 1:

A male baby is born at 30+3 weeks, birth weight 1.3kg, following spontaneous preterm labour with Apgar of 6 & 8 at 1 & 5 minutes. Baby was admitted in NICU for respiratory distress & started on CPAP.

On day 2 of life, the baby develops increasing jitteriness, irritability, poor tolerance to feeds, intermittent episodes of limb stiffening, no documented hypoglycemia.

Vitals: HR 168/min, RR 58/min, SpO2 95% on CPAP, Temp 36.7° C.

Questions:

1. What are the differentials for jitteriness/ stiffening in this baby?
2. What investigations will you send immediately?
3. S. Calcium 6.0 mg/dl & I. Ca 0.72mmol/L, what is the diagnosis?

4. Why is this baby particularly predisposed to hypocalcemia?
5. How will you treat this baby?
6. 6 hours later, baby is clinically better, no further spasms but I. Ca is 0.76 mmol/L. What are the reasons for persistent hypocalcemia?
7. After stabilization, baby continues to require calcium. Further workup reveals: Ca low, Phosphate high, Mg normal, ALP normal, 25 OH vitamin D normal, PTH low.
8. What is your diagnosis? Enumerate the syndromic association with neonatal hypoparathyroidism.

Case Scenario 2:

2 years old male child, hailing from Chennai, born to 3rd degree consanguineous couple, presented with complaints of progressive Bowing of legs and not able to walk. He weighs 10.5 kgs (3rd to 15th centile), and height is 80cms (<3rd centile). He is an active child and examination shows frontal bossing, widened wrists, rachitic rosary and genu varum (bowing of legs).

Questions:

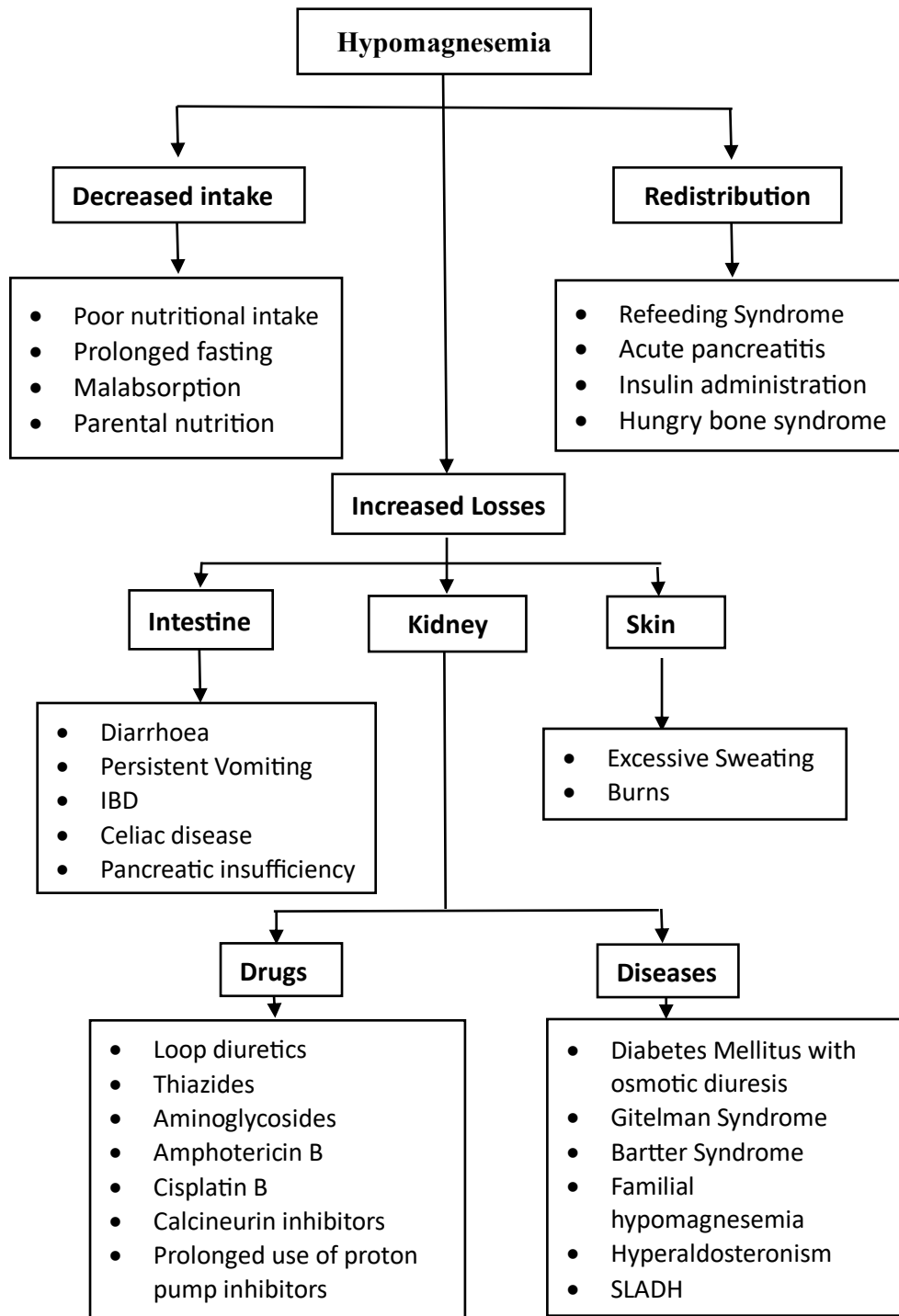
1. What is the additional history you would like to elicit?
2. What is the 1st line of investigation you would like to send?
3. What is the management of nutritional rickets?
4. When do you suspect non nutritional rickets?
5. How do you approach hypocalcemia and rickets?

HYPOMAGNESEMIA

Contributors: Dr. Sridevi A Naaraayan, Dr. LK Prem Kumar, Dr. Pooja, Dr. Pari Palanivelan

Magnesium:

- Magnesium is the fourth most abundant cation in the body and an essential intracellular mineral involved in more than 300 enzymatic reactions.
- Magnesium plays an important role in energy metabolism, neuromuscular transmission, cardiac conduction, bone health, DNA and protein synthesis, and regulation of potassium and calcium homeostasis.
- Largest store of magnesium is present in musculoskeletal system (bones 53%, muscles 27% and soft tissue 19%); blood contains less than 1% of total body magnesium.
- Hypomagnesemia is relatively common in hospitalized and critically ill children. It may be asymptomatic or present with significant neuromuscular and cardiac manifestations.
- Normal concentration of magnesium in the serum is 1.7 to 2.4mg/dl.
- Hypomagnesemia is defined as a serum magnesium concentration below 1.7 mg/dl
- An important clinical point is that magnesium deficiency can produce refractory hypokalemia and hypocalcemia; therefore, magnesium should be measured whenever these abnormalities are unexplained or difficult to correct.
- Neuromuscular manifestations include weakness, tremor, muscle cramps, fasciculations, tetany, hyperreflexia, ataxia, seizures, nystagmus and irritability.
- Cardiovascular manifestations include prolonged QT interval, ventricular arrhythmias, atrial arrhythmias, torsades de pointes and increased susceptibility to digoxin toxicity.

CAUSES:

APPROACH:

Step 1: Confirm Hypomagnesemia (Serum Mg < 1.5 mg/dl)

Step 2: Focused history taking to identify obvious causes

Step 3: Fractional excretion of magnesium

$\text{FeMg} = \frac{\text{Urine Mg} \times \text{Plasma creatinine}}{[(0.7 \times \text{plasma Mg}) \times \text{U creatinine}] \times 100}$

FeMg > 5% signifies urinary loss of Mg while <5% signifies other causes.

TREATMENT:

➤ Intravenous-

Preparation – Magnesium sulphate 25% and 50%

Dose - 25-50 mg/kg/dose over 30-60 minutes (maximum ceiling dose 2g) Can be repeated every 6 hourly for a total of 3 doses.

➤ Oral

Indicated – In asymptomatic children with mild hypomagnesemia without significant GI intolerance (diarrhea)

Dose – 2.5 to 5 mg/kg 3 times a day; can go upto 10-20mg/kg, tolerance better for lower doses.

Case Scenario:

15-year-old boy, second child of a third-degree consanguineous marriage, referred with intermittent right loin pain for 3 months and one episode of passing gravel per urethra. On further questioning — he has longstanding polyuria/polydipsia and nocturia twice nightly. Occasionally he has muscle cramps. Two culture-proven urinary tract infections, at 6 and 11 years. No seizures, carpopedal spasm or paraesthesiae. Term birth, birth weight 2.6 kg, normal development.

Height 152 cm (SDS -2.3), weight 41 kg, BSA 1.32 m². BP 108/70 mmHg. No clinical rickets. Other systems normal.

Poor distance vision since at least 8 years of age, never evaluated. Ophthalmology opinion now revealed: best-corrected visual acuity 6/36 in either eye, refractive error -12.00 DS bilaterally, horizontal nystagmus, bilateral macular colobomata.

Investigations

Investigation	Result
Blood	
Sodium / potassium / chloride	138 / 3.7 / 108 mmol/L
Bicarbonate	20 mmol/L (anion gap 10)
Urea / creatinine	42 / 1.0 mg/dL — eGFR 63 mL/min/1.73 m ²
Calcium (albumin-corrected) / ionised calcium	8.6 mg/dL / 1.11 mmol/L
Phosphate / alkaline phosphatase	4.4 mg/dL / 348 U/L
Magnesium	1.1 mg/dL (0.45 mmol/L) [normal 1.7–2.4 mg/dL]
Intact PTH / 25-hydroxyvitamin D	152 pg/mL / 21 ng/mL
Urine	
Calcium (24 h) / spot calcium: creatinine	324 mg/day (7.9 mg/kg/day) / 0.47 mg/mg
Magnesium and creatinine (spot, paired with plasma)	Urine Mg 4.8 mg/dL, urine creatinine 23 mg/dL (plasma Mg 1.1 mg/dL, plasma creatinine 1.0 mg/dL)
Imaging and other studies	
Ultrasonography of urinary tract	Normal-sized kidneys with bilateral medullary nephrocalcinosis; 4 mm calculus in right lower calyx

Questions:

1. What is the most likely diagnosis?
2. What are the symptoms and complications of hypomagnesaemia?
3. Which investigation distinguishes renal from extrarenal magnesium loss?
4. How will you correct the hypomagnesaemia?
5. Why is the urinary calcium high in this disorder? How to manage?

HYPO/ HYPERKALEMIA

Contributors: Dr. Gowrishankar NC, Dr. Muthiah P, Dr. Deepika S, Dr. Priyadarshini M

Potassium is the principal intracellular cation and its intracellular concentration is ~150 mEq/L. Extracellular concentration of Plasma K⁺ is lower (<1% of total body K⁺). Primary reservoir of most body K⁺ is muscle tissue. Body K⁺ increases during puberty significantly in males as muscle mass increases. But plasma concentration does not always reflect total body K⁺ content. The normal K⁺ values are given in Fig 1.

Age	Range (mEq/L)
Preterm	4 to 6.5
Newborn	3.7 to 5.9
Infant	4.1 to 5.3
>1 year	3.5 to 5

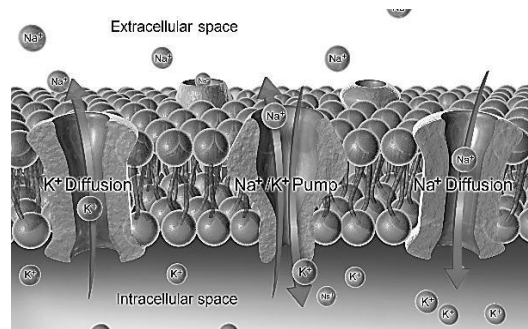


Fig 1. Normal K⁺ values

Fig 2. Na⁺ K⁺ pump

The high intracellular K⁺ is maintained by Na⁺, K⁺ -ATPase which pumps Na⁺ out of cell and K⁺ into cell (Fig 2). This balances normal leak of K⁺ out of cells via potassium channels and is driven by favourable chemical gradient. The resulting chemical gradient produces resting membrane potential of cells. K⁺ controls the electrical responsiveness of nerve and muscle cells, contractility of cardiac, skeletal, smooth muscle. Any condition which alters the intracellular and extracellular compartments can alter distribution of K⁺ (Box 1).

S.K⁺ is normally 0.4 mEq/L higher than plasma value secondary to K⁺ release from cells during clot formation. It is exaggerated with thrombocytosis due to K⁺ release from platelets. For every 100,000/m³ increase, S. K⁺ rise - 0.15 mEq/L. Elevated

Box 1. Factors affecting k⁺

- Factors driving K⁺ intracellularly: Insulin, β -agonists, Alkalosis.
- Factors driving K⁺ extracellularly: Acidosis, α -agonists, Exercise, Hyperosmolality.
- Factors increasing renal excretion: Aldosterone, High filtrate flow rate, High distal Na⁺ delivery, Alkalosis.
- Factors decreasing renal excretion: Acidosis, Insulin, Catecholamines

WBC counts, typically $>200,000/m^3$ there is a dramatic elevation of S.K⁺. Analysis of plasma sample always provides an accurate result.

Potassium and intestines: Potassium is plentiful in food. 1-2 mEq/kg is the recommended intake. 90% of ingested K⁺ is absorbed in small intestine. Colon exchanges body K⁺ for luminal Na⁺. There is increased colonic secretion of K⁺ in Renal failure, aldosterone, glucocorticoids. Intestinal losses have minimal role in maintaining potassium homeostasis but increases in renal failure and hyperkalemia.

Potassium and kidneys:

More than 90% filtered K⁺ reabsorbed in proximal tubule (PT) & thick ascending limb (TAL) of loop of Henle. In early proximal tubule: passive K⁺ reabsorption paracellularly through "solvent drag," secondary to Na & H₂O reabsorption (Fig 3). In late proximal tubule, the luminal voltage shifts from negative to positive and provides additional driving force for paracellular reabsorption of K⁺.

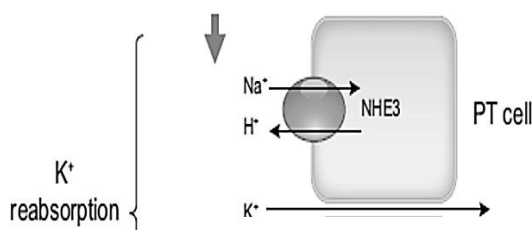


Fig 3: K⁺ in proximal convoluted tubule

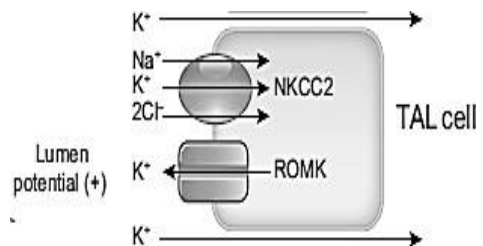


Fig 4. K⁺ in Thick ascending limb of loop of Henle

Renal K⁺ handling- thick ascending limb (TAL) of loop of Henle re-absorption: Reabsorption of K⁺ by proximal tubule and thick ascending limb by paracellular & transcellular reabsorption is relatively constant and on average (~10%) of filtered K load reaches distal nephron (Fig 4). Major apical transporter-Na-K-2Cl cotransporter (NKCC2) reabsorbs Na, K, Cl. The driving force is low intracellular Na concentration generated by basolateral Na-K-ATPase. This activity depends on K recycling from cell to tubular lumen by renal outer medullary K channel (ROMK, also called Kir1.1) : K⁺ recycling through ROMK leads to positive luminal charge which provides additional driving force for paracellular K⁺ reabsorption.

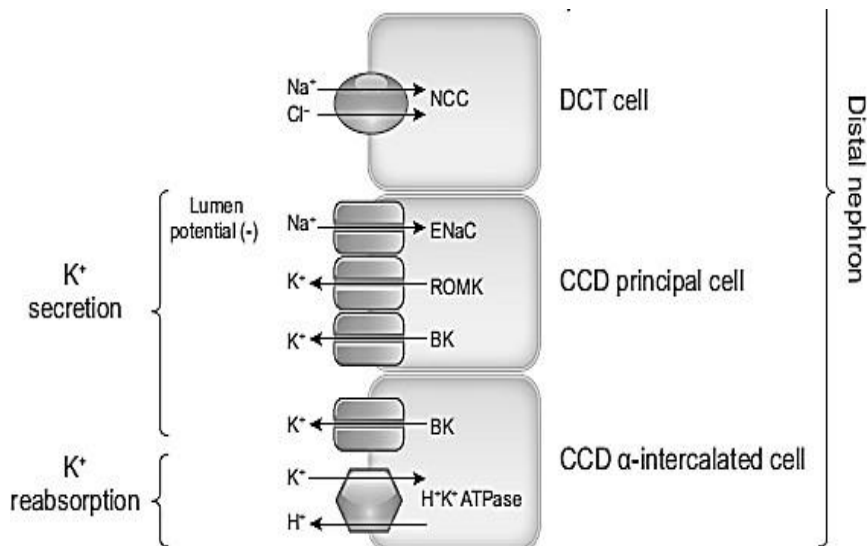


Fig 5: K⁺ in Distal convoluted tubule

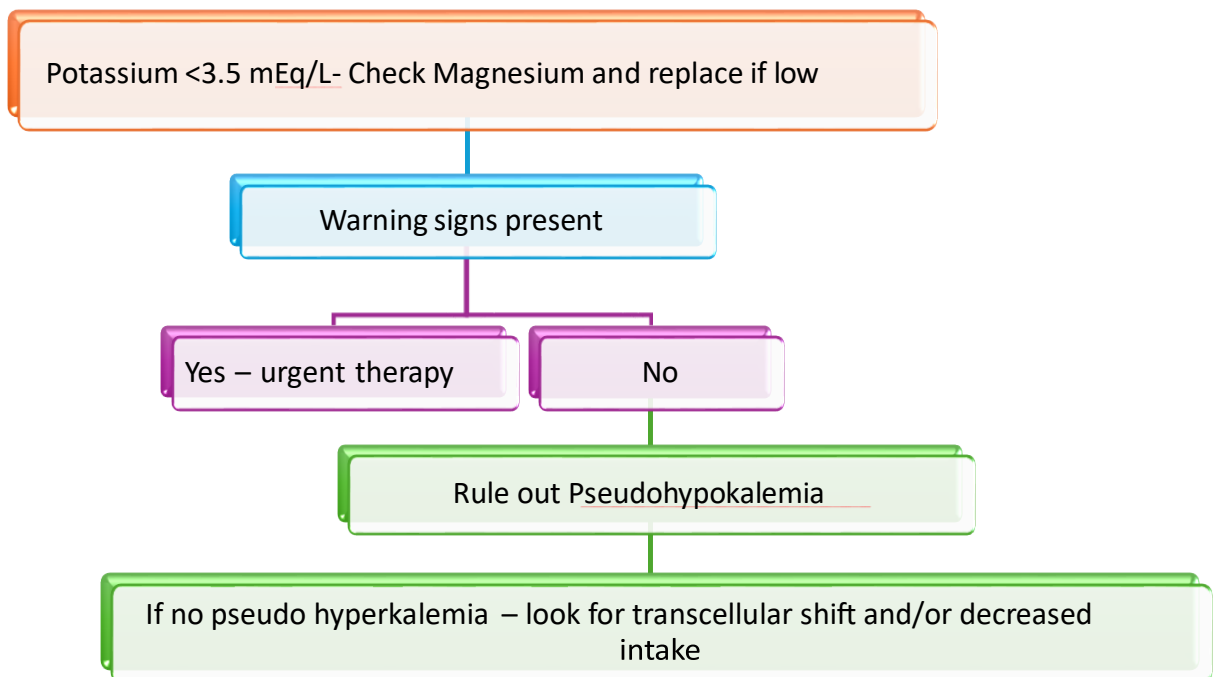
K⁺ in distal convoluted tubule and collecting duct: K⁺ secretion is determined by intracellular & luminal K⁺ concentrations and voltage difference across apical plasma membrane & its K⁺ permeability. K⁺ secretion is mediated by K⁺ chloride cotransporters (KCC), Renal outer medullary K⁺ channel (ROMK) and "big" K⁺ (BK) channels. K⁺ secretion increases in cells that express ENaC (epithelial sodium channels) & renal outer medullary K⁺ channel (ROMK) - especially principal cells in collecting duct. It occurs as a result of electrochemical coupling of Na⁺ reabsorption by ENaC to K⁺ secretion by ROMK (electrogenic secretion). But in circumstances where K⁺ depletion occurs, H⁺-K⁺-ATPase in intercalated cells switch distal nephron function from K⁺ secretion to K⁺ reabsorption.

Hypokalemia

It can be classified as mild (potassium level from 3 to less than 3.5 mEq/L), moderate (potassium level 2.5 to less than 3 mEq/L) and severe (potassium level less than 2.5 mEq/L). Hypokalemia can be classified based on the causes of Potassium Imbalance (Table 1). Approach to hypokalemia is given in Fig 6 and 7.

Transcellular shift	Primary hyperaldosteronism Diuretics Familial / periodic paralysis
Renal loss of potassium	Bartter syndrome, Gitelman syndrome Renal tubular acidosis Nephrotic syndrome, ATN Celiac disease, villous adenoma Hypomagnesemia
Extra renal loss of potassium	Diarrhoea Vomiting Laxative abuse Excessive sweating
Decreased intake - dietary	Lower potassium intake
Spurious hypokalemia	Leukemia & elevated WBC counts (if plasma for analysis left at room temperature - permit WBCs to take up K ⁺ from plasma)

Fig 6. Approach to hypokalemia



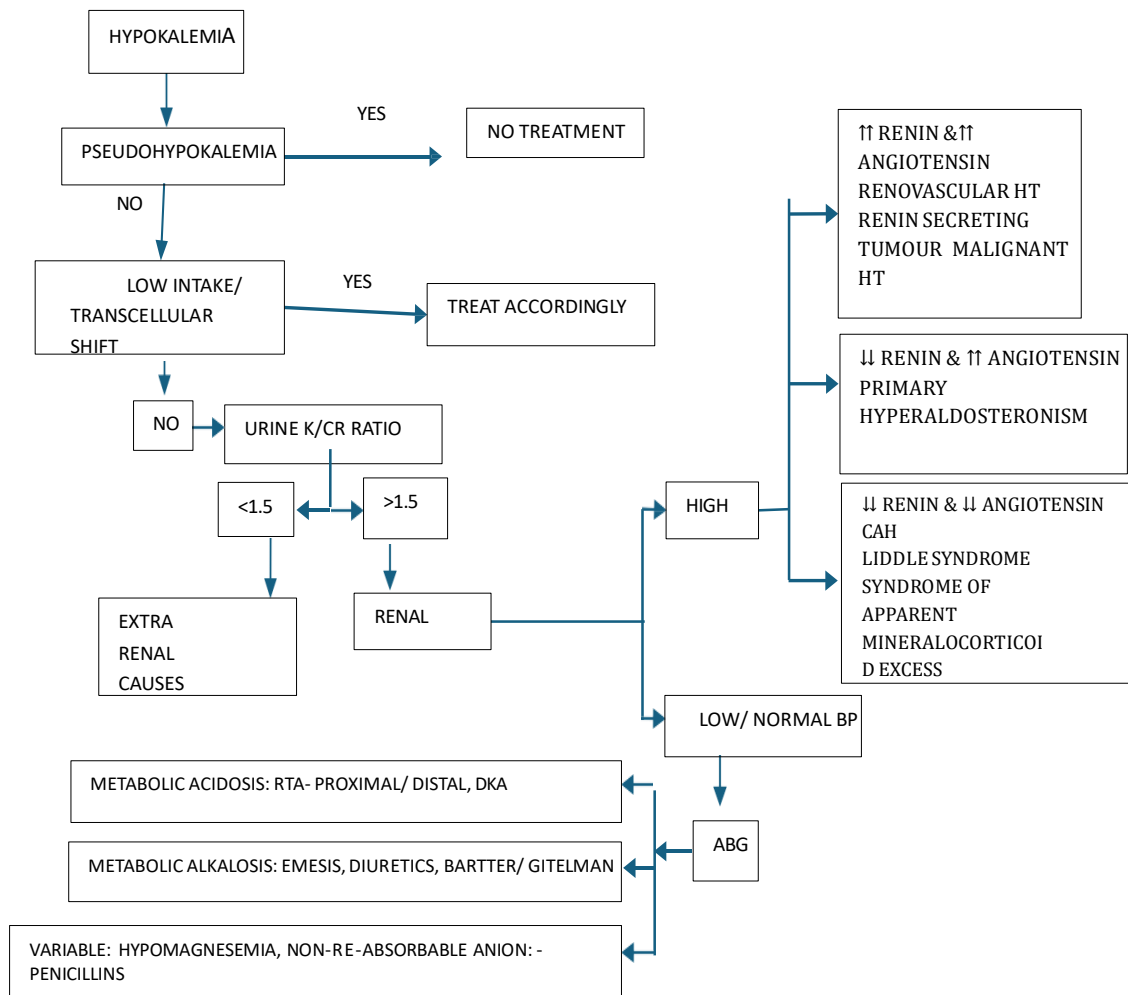


Fig 7: Approach to hypokalemia

Case Scenario 1:

A 12-year-old girl weighing 40 kg presents to you with abdominal pain, vomiting and rapid breathing for 1 day. Parents say she has lost weight over the past few weeks and has been very thirsty and passing urine frequently.

On examination she is drowsy but arousable, dehydrated and tachypneic with deep labored breathing.

Vitals: HR 130/min, BP 100/70 mmHg, RR 32/min. Peripheral pulses feeble. Central pulses well felt.

Questions:

1. What is the physiological status?
2. Immediate management?
3. Probable etiology?
4. What investigations would you like to order?

Case Scenario 2:

A 7-year-old boy weighing 22 kg, known to have recurrent wheezing, presents with severe respiratory distress for 6 hours.

He has received multiple salbutamol nebulisations at a peripheral hospital before referral.

On arrival, he is anxious, tachypneic and using accessory muscles of respiration.

After oxygen, systemic steroid and repeated nebulised salbutamol, his respiratory distress begins to improve.

The mother now reports that the child is complaining of severe generalized weakness and is finding it difficult to stand.

EXAMINATION

Conscious and oriented • Mild residual wheeze • Generalized hypotonia • Power 3/5 in lower limbs • Deep tendon reflexes reduced • No sensory deficit

VITALS

HR 138/min • BP 104/68 mmHg • RR 30/min • SpO₂ 96% with oxygen • CRT <2 sec

Questions:

1. What is the immediate concern?
2. What is the likely cause of the weakness?
3. Is the potassium being lost from the body—or shifted intracellularly?
4. What investigations would you order?
5. Does the child need potassium replacement?

Case Scenario 3:

A 3-year-old girl weighing 11 kg is brought with poor weight gain for the last year. Parents report excessive thirst, frequent urination, poor appetite and recurrent episodes of generalized weakness.

There is no history of chronic diarrhea or recurrent vomiting.

No significant drug history.

The child has been seen several times for 'poor weight gain' without a clear diagnosis.

EXAMINATION

Alert but thin • Mild dehydration • Mildly reduced muscle tone • No edema • No focal neurological deficit

VITALS

HR 118/min • BP 92/58 mmHg • RR 28/min • SpO₂ 99% room air

Questions:

1. First define the acid–base disorder.
2. Then decide whether the potassium loss is renal or extrarenal.
3. Check serum Na⁺, K⁺, Cl⁻ and HCO₃⁻, together with blood gas and renal function.
4. Assess urinary potassium excretion.
5. Urine pH becomes particularly important if the child has metabolic acidosis.

Hyperkalemia

Serum or plasma potassium that is higher than upper limit of normal potassium – typically considered to be 5.5 mEq/L. The common symptoms are nonspecific - predominantly related to muscular or cardiac function especially cardiac arrest. Weakness and fatigue are the most common complaints. It is a common complication - very low birth weight infants especially in infants with low urinary flow rates during the first few hours after birth. Pathophysiological correlation of hyperkalemia is given in Table 2. ECG changes with hyperkalemia is given in Box 2.

Table 2. Hyperkalemia and its etiology

Mechanism	Pathophysiological correlation
Increased potassium intake (rare cause -with exception - children with chronic kidney disease)	Exposure to high potassium loads in intravenous fluids or medications Exposure to potassium-containing medications Massive transfusions of stored blood
Transcellular potassium movement	Structural cellular damage due to: Hemolysis /Rhabdomyolysis / Tumor lysis No structural cellular injury: Metabolic acidosis / Diabetic ketoacidosis / Hyperkalemic periodic paralysis
Abnormal renal potassium excretion	Decreased effective circulating volume Decreased RAAS activity: Cong adrenal hyperplasia/ Adrenal insufficiency Drug effect (ACE inhibitor/ARB, eplerenone, spironolactone or aliskiren) Significant renal impairment + either acute or chronic loss of GFR Impaired tubular potassium secretion: Reflux nephropathy /Obstructive uropathy / Sickle cell nephropathy / Drug effect (amiloride, triamterene) / Hypoaldosteronism (type IV renal tubular acidosis)
Pseudohyperkalemia	Hemolyzed specimen Leukocytosis or thrombocytosis

Box 2 : ECG changes and hyperkalemia

K = 5.5 to 6.5 mEq/L: Tall, peaked T waves

K = 6.5 to 7.5 mEq/L: Flattening or loss of P waves

K = 7 to 8 mEq/L: Widening of the QRS complex

K = 8 to 10 mEq/L: Severe arrhythmias, sine-wave pattern, and potential progression to asystole

Approach to hyperkalemia

Look for S K+: if it is more than 5 mEq / L, look for warning signs. If absent, look for pseudohyperkalemia. To avoid pseudohyperkalemia, analyze sample promptly to avoid K+ release from cells as it occurs if sample is stored in cold. Transport by pneumatic tube if cell membranes are fragile as in leukemia can lead to pseudohyperkalemia. Heparin causes lysis of leukemic cells leading to false elevation of plasma sample. If blood gas syringe has less heparin, it can give more accurate reading than standard tube. Rare genetic disorders cause in vitro leakage of K+ from red blood cells (RBCs) as in familial pseudohyperkalemia, an autosomal dominant condition with ABCB6 gene.

If pseudohyperkalemia is not present, look for evidence of increased intake or transcellular shifts. If absent, do Urine sodium. If sodium < 25 mEq/L, it could be due to decreased distal renal flow (e.g., AKI/ CKD/ CCF/ cirrhosis). If > 25 mEq/L, measure serum aldosterone. If normal aldosterone level present, it could be medication use, pseudo hypo aldosteronism, amyloidosis, SLE, sickle cell, primary renal tubular defects. If low aldosterone, measure serum renin. If normal Renin, it could be primary adrenal insufficiency, medication use (e.g., ACEI, ARB, heparin). If low Renin, it could be hyperglycemia, primary renal tubular defects, medication use-NSAID, beta blockers).

Fig 8 gives approach to hyperkalemia.

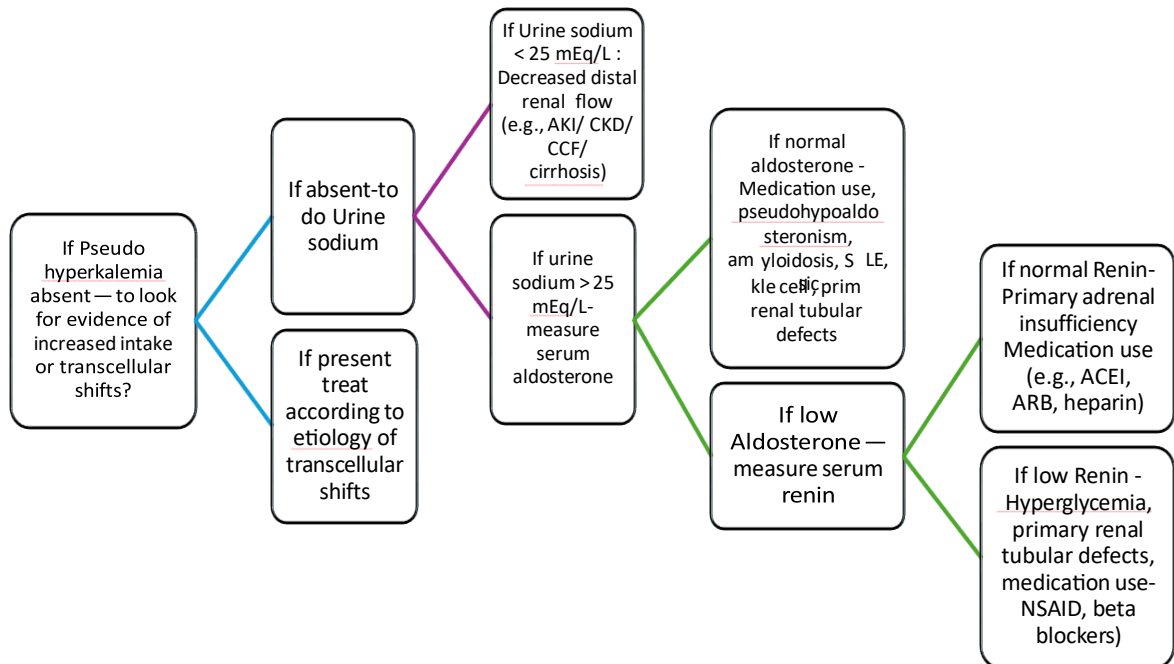


Fig 8: Approach to hyperkalemia

Case Scenario 1:

- 1 week old term male neonate with 2 days of irritability and inconsolable cry.
- Child very hungry, receiving supplement formula feeds, but post feeds having non projectile vomiting. Birth weigh 3.3 kg, now 2.7 Kg. O/E dry mucus membrane, irritable, tears+. No dysmorphism, organomegaly, rash.
- What are we thinking here?
- Investigations: Na⁺ 123, K⁺ 7.8 mEq/L, and there is metabolic acidosis with a bicarbonate level of 13 mEq/L.

Questions:

1. How do we go about? Diagnosis??
2. What are the other investigations that have to be done?
3. Management options available to treat?
4. Quickest way to bring down potassium ?
5. Mechanisms and strategies in treatment of hyperkalemia?

Case Scenario 2:

- 10-year-old male child 30 Kg newly diagnosed with T-cell ALL with
- TLC 1,00,000, Plt-30,000, Hb 7. electrolytes- Na⁺ 130, K⁺ 7, Cl 100, Ca 6, Phosphate 9, Hco₃ 22
- He is planned for chemotherapy initiation.

Questions:

1. What are the concerns in treating this patient?
2. What are the other relevant investigations we want to know?
3. Quickest way of bringing potassium down?
4. He is also observed to have tall T waves after admission. What do we do?
5. What are the other supportive measures and management strategies for this patient?
6. What are the goals of treatment?

Case Scenario 3:

- 8 years old male child k/c/o VUR with reflux uropathy presenting to hospital with loose stools and lethargy with tiredness.
- Height < 2 SD for age, weight < 2 SD for age. BP 140/70mmHg. Urine output is normal.
- Investigations: Urea 50, Creat 2.2, K 6, Na 134, Cl 100, HCo₃ 14. He is on oral calcium, oral Sodabicarb.

Questions:

1. What is the diagnosis? What are the symptoms of hyperkalemia?
2. How do we manage his hyperkalemia?
3. How to prevent hyperkalemia in this patient?
4. What if medical management fails to control electrolyte disturbance?
5. What mode of dialysis is suitable for this patient?

Case Scenario 4:

- 3 years 12 Kg child who is admitted to PICU with MRSA septic shock on inotropic support adrenaline 0.2 $\mu\text{g/kg/min}$, noradrenaline 0.1 $\mu\text{g/kg/min}$ with oliguria 0.6 ml/kg/hr for last 6 hours.
- Electrolytes K 6.5, Na 137, Cl 100, Hco3 12, blood gas pH 7.2, pCo2 20, Hco3 13. Urea 25 mg/dL, creatinine 0.7 mg/dL

Questions:

1. How to treat hyperkalemia immediately?
2. What is the main stay of treatment for this patient?

