NephKids 2025

ELECTROLYTE

-WORKBOOK-

Organized by

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

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NephKids 2025

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PROGRAMME

DAY 1

Saturday, September 13th, 2025
FLUID AND ELECTROLYTE WORKSHOP

TOPIC	MODERATOR	SPEAKERS	09:30-10:35	10:35-11:40	11:40-12:45
Hyponatremia	Dr P Narayanan	Dr Vasanth Kumar S Dr Anita Tarigopula Dr Dhakshayani RV	Group A	Group B	Group C
Fluid in special situations	Dr Poovazhagi V	Dr Sunil Reddy K G Dr Sharadha R C Dr Priyavarthini V	Group B	Group C	Group A
Metabolic Acidosis	Dr Thangavelu S	Dr PremKumar L K Dr Ekambaranath T S Dr Muthiah P	Group C	Group A	Group B
LUNCH	**12:45 – 13:30 hrs**		**13:30-14:25**	**14:25-15:20**	**15:20-16:15**
Hypernatremia	Dr Anitha V P	Dr Venkateshwari R Dr Chidhambharam L Dr Prasanna R	Group A	Group B	Group C
Hypocalcemia/ Hypomagnesemia	Dr Gowrishankar NC	Dr Kalpana S Dr Vidhya P S Dr Karthikeyan M	Group B	Group C	Group A
Hypo/ Hyperkalemia	Dr Shanthi S	Dr Shyamala J Dr Deepika S Dr Naresh Kumar S	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

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HYPONATREMIA

Contributors: Dr. Narayanan P, Dr. Vasanth Kumar S, Dr. Anita Tarigopula, Dr. Dhakshayani R V

Approach to Hyponatremia

Essential facts

- Hyponatremia develop only by two mechanisms
  - Gain of free water (most common) – could be obvious (edema) or clinically occult /subtle
  - Loss of sodium (less common) – mostly iatrogenic; rarely pathological

- Hyponatremia is always associated with low serum osmolarity

- In most instances, the underlying cause is evident from the clinical setting with careful clinical evaluation. Hence not many lab tests are required

Steps in Assessment

Step 1 – Identify true hyponatremia

Confirm hyponatremia (rule out a lab error if the clinical setting is unlikely) - measure serum osmolarity if available. Normal or high serum osmolality suggest pseudohyponatremia

Step 2 – Identify possible mechanism

- 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 osmolarity along with a trial of fluid therapy will help in identifying the predominant mechanism.

Step 3 – 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)

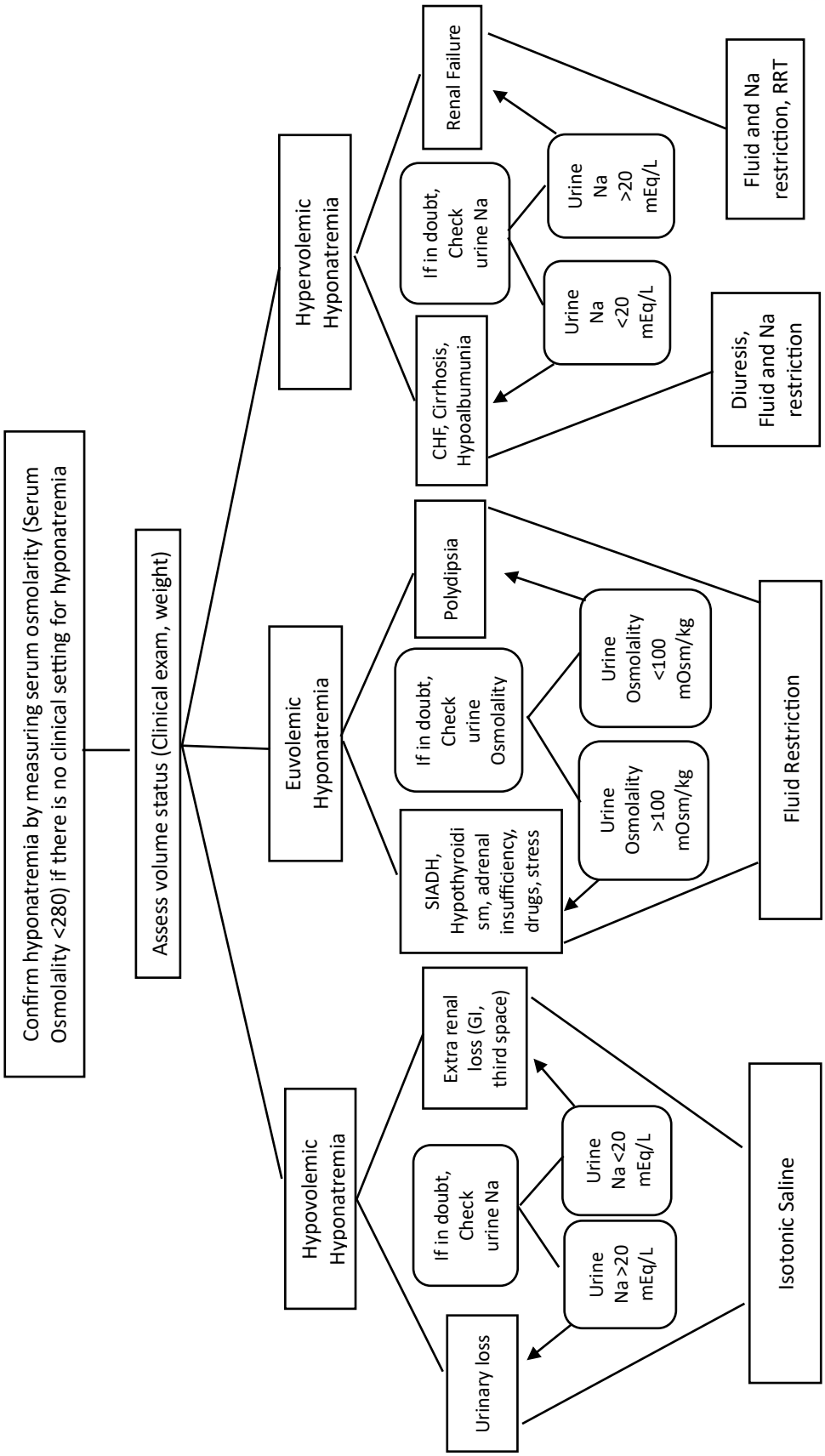
- If euvolemic, think of SIADH / polydipsia /endocrine causes

- If hypovolemic, think of fluid loss (GI/renal/third space), diuretic use /cerebral salt wasting in appropriate settings

Step 4 – 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


Confirm hyponatremia by measuring serum osmolarity (Serum Osmolality <280) if there is no clinical setting for hyponatremia
Assess volume status (Clinical exam, weight)
Hypervolemic Hyponatremia
Renal Failure
If in doubt, Check urine Na
Urine Na >20 mEq/L
Urine Na <20 mEq/L
Fluid and Na restriction, RRT
CHF, Cirrhosis, Hypoalbumunia
Diuresis, Fluid and Na restriction
Euvolemic Hyponatremia
Polydipsia
If in doubt, Check urine Osmolality
Urine Osmolality <100 mOsm/kg
Urine Osmolality >100 mOsm/kg
SIADH, Hypothyroidism, adrenal insufficiency, drugs, stress
Fluid Restriction
Hypovolemic Hyponatremia
Extra renal loss (GI, third space)
If in doubt, Check urine Na
Urine Na <20 mEq/L
Urine Na >20 mEq/L
Urinary loss
Isotonic Saline

Case Scenarios

Case 1:

3yr F, weighing 10kg with evaluation s/o TB meningitis was intubated for refractory seizures. Started on ATT with steroids, neuroprotective care and 3% saline - 1ml/kg/hr. EEG monitoring revealed no seizures. Serum sodium -138meq/L. which was monitored 6hrly and sodium was maintained between 140 to 145 meq/l, so 3% saline tapered and stopped by 72hrs. On Day 5 - serum sodium slowly drifted from 140 to 137 and 130meq/l in 12hours. Child otherwise stable with Vitals HR-100/min, Spo2-99%BP-100/76 mmHg, UO-1.8ml/kg/hr.

Questions:

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 u manage this child?

Case 2:

- 2 years old male child, weighing 10 kgs
- 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 mineral water and fruit juices.
- Brought to Emergency Department with lethargy, refusal of feeds and poor oral intake.
- On examination, child is listless, irritable, temperature- 102deg F, skin turgor is reduced
- Child's HR-160/min, RR- 46/min, liver span-N, peripheral pulses -feeble +++/+, cool below ankles, CRT>3sec, BP- 70/40mmHg.

Questions:

1. What is the physiological status of this child?
2. What is the immediate management in the present status?

- After the first bolus, his vitals were
- HR-140/min, RR-40/min, +++/++, cool below ankles, CRT<3 sec
- Child continued to have loose stools; skin turgor is reduced & eyes are sunken.
- Investigations:
  - CBC – neutrophilic leucocytosis
  - Blood glucose – 90mg/dL
  - Serum electrolytes – Na- 128mEq/L
  - K- 3.5mEq/L
  - HCO3- 18mEq/L

Questions:

1. How will you categorise dehydration based on severity and based on osmolarity?
2. How will you categorise hyponatremia?
3. Is there a possibility of pseudohyponatremia in this child? If so, what are the conditions that you will consider?
4. What is the further management in this child?

Case 3:

A 7-year boy presents to the emergency department with progressive swelling over the face and legs over the past 1 week. There is history of reduced urine output noticed for the past 3 days with the last urine output being 12 hours prior to presentation. Mother also gave past history of 3 - 4 episodes of similar complaints in the last 3 years

On clinical examination he has puffy face with periorbital swelling and edema of the lower limbs. His vital signs are HR – 110/min, RR-28/min, BP: 100/64mmHg, SpO_2: 94% in room air. There is noticeable weight gain of around 2.5Kgs compared to the baseline weight. He is alert but has mild subcostal retractions and diminished air entry bilaterally.

Reports of the initial investigations are as follows:
VBG: pH: 7.36, pCO_2: 30, pO_2: 46, HCO_3: 25, BD: 2, Na^+: 122 mEq/ltr, K^+: 4mEq/ltr, Ca^{2+}: 1.0, Cl^-: 100mEq/ltr, Creat: 0.48mg/dl, Urea:39mg/dl.
CBG :97mg/dl
Sr Albumin: 2gm/dl

Questions:

1. What is the likely diagnosis?
2. What is the pathophysiology causing hyponatremia?
3. Enumerate the steps of management of the child.

FLUID IN SPECIAL SITUATIONS

Contributors: Dr. Poovazhagi V, Dr. Sunil Reddy K G, Dr. Sharadha R C, Dr. Priyavarthini V

Basic principles:

- Fluid therapy is a dynamic process and needs to be tailored to the individual child
- Bedside monitoring for intake output and ongoing loss is mandatory
- History, clinical examination, intake output chart, point of care ultrasound are useful parameters to decide on fluid therapy in sick children
- Fluids to be planned to meet the resuscitation needs, replacement needs, maintenance, and ongoing loss.

Assessment of fluid status:

- Meticulous monitoring of weight
- Clinical assessment for signs of dehydration
- Lab evaluation of electrolytes
- Timed Intake output charts

Fluid Replacement

- Resuscitation fluids as indicated
- Replacement of what is lost- based on clinical features
- Percentage of weight loss
- Can add anticipated losses if deemed necessary

Identify the associated dyselectrolytemia

- Common with sodium and potassium
- Less commonly magnesium and phosphate

Consider co-morbid states

- Severe acute malnutrition (SAM)
- Anaemia
- Cardiac dysfunction
- Renal dysfunction
- Cerebral edema
- SIADH
- Capillary leak states

Final prescription of fluids

Fluids -what? how much? and how long?
Revisit Intake output chart and electrolytes and modify fluids

Summary of steps

Step 1—Assessment --based on weight/clinical features/IO chart
Step 2 – Deficit -percentage of wt. loss/ clinical features
Step 3 –Dyselectrolytemia -look at sodium, potassium, magnesium
Step 4 –Comorbid states - SAM, organ involvement
Step 5 – Final fluid prescription – composition rate and volume

Remember
Fluid therapy ---evaluate status, identify dyselectrolytemia and co-morbid state and intervene
Keep updating based on repeated assessments

Case Scenarios

Case 1:

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) mmHg, 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, pCO2 26 mm Hg, PO2 96 mm Hg, HCO3 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.
3. What are the frequent electrolyte abnormalities encountered in this situation and how do you manage them?
4. What are the common serious complications anticipated and how can they be prevented?

Case 2:

1 year old weighing 10 kgs was brought to emergency department with c/o passing large volume watery stools 8- 10 episodes since yesterday. He had been vomiting 5-6 times earlier and refusing to take oral liquids. He has been listless and has been sleepy for the past 6-8 hours. His last urine output could not be recalled by the parents as the infant had been on diapers. On examination he was febrile, airway stable, had effortless tachypnoea, Tachycardia

Hypotensive, normal liver span with feeble peripheral pulses, cold peripheries, abnormal colour and prolonged CRT. He had features of severe dehydration. His CBG read 58mg%.

1. Prioritise your fluid prescription in the order of severity?
2. Difference between hypovolemia and dehydration?
3. What are the non- invasive measures of dehydration?
4. Will this dehydration be associated with electrolyte imbalances? If so, which electrolyte imbalances will you anticipate?
5. Which is the ideal fluid for dehydration correction?

Case 3:

A 5 years old child with nephrotic syndrome presented to ER with history of fever for 2 days, abdominal pain, progressively increasing anasarca and oliguria. On examination she had mild pallor, severe oedema (genital and sacral areas), cold peripheries with CRT of 3 to 4 sec. She had tachycardia, BP measured was 80/60 mm Hg, systems examination was normal.

Questions:

1. What is the diagnosis?
2. Step wise management in treating her condition.
3. Role of diuretics and albumin in treating her.

METABOLIC ACIDOSIS

Contributors: Dr. Thangavelu S, Dr. PremKumar L K, Dr. Ekambaranath TS, Dr. Muthiah P

In addition to the measurement of heart rate, respiratory rate, BP, perfusion, alertness, temperature and glucose, acid-base status is also an important vital function to be measured. But unlike the other parameters, acid-base status needs, arterial sample and interpretation and requires knowledge of the arterial blood gas (ABG) parameters. Hence, ABG analysis is done only in children with critical illness in the ER or PICU. In brief, there are 4 simple disturbances: Metabolic acidosis and metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. Two parameters that decide the pH or the acid-base status are the partial pressure of CO2 and serum bicarbonate. Traditionally, PCO2 reflects the lung function, and HCO3 reflects metabolic or renal function.

Acid-base physiology

Main systems that regulate acid-base status are lungs, kidneys, and buffers (Fig). Acids are normally generated during normal metabolism in the body, during respiration and digestion of food. Fixed acids are excreted by the kidneys, and volatile acids are excreted by the lungs. In addition to excreting acids, kidney also generates bicarbonates. In an unusual situation, when excessive amounts of acids are produced or the lungs and kidneys fail in their function when there is appearance of respiratory failure or renal failure, buffers quickly appear in the scene and reduce the fluctuation in acid-base status

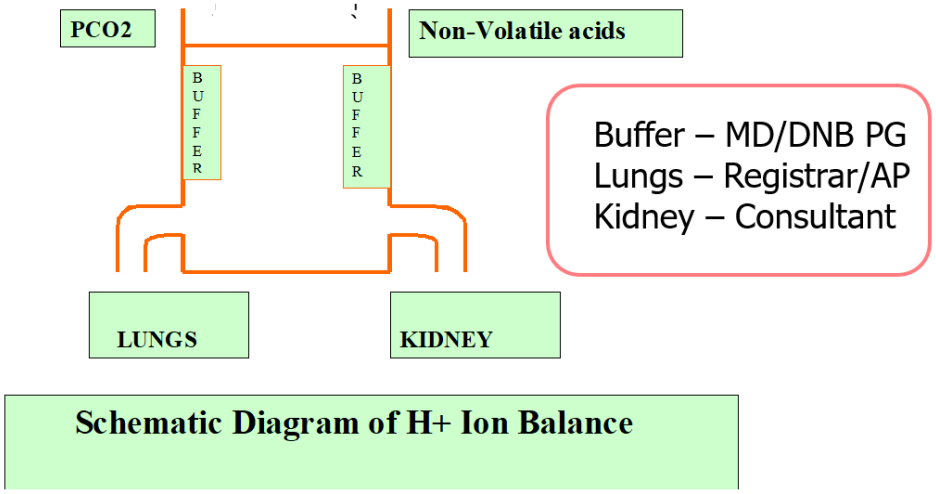
Compensation:

pH does not depend on any individual numbers, but depends on the relative ratio between acid and base, as per the Henderson-Hasselbach equation. Hence compensatory mechanisms help to keep the situation under control in the initial few hours.

$$\left[H^+\right] = 24 \times PCO_2 / \left[HCO_3^-\right]$$

The kidneys in children normally excrete 2-3 mEq/kg/24 hr of H+, which are produced from dietary protein metabolism, incomplete metabolism of carbohydrates, and fat. The lungs excrete CO2 that is produced in the body during respiration. and maintain a normal Pco2 (35-45 mm Hg).

Fig 1. Schematic diagram of acid-base balance and the regulating factors



Each day, approximately 15,000 mmol of CO2 (respiration)
50 -100 meq of non-volatile acids are produced (protein digestion)

Rules for compensation

Once a child develops acid-base disturbances like metabolic acidosis, physiology cannot wait till the disease process, like diarrhea or shock, or respiratory failure, is resolved; hence, it engages in temporary changes in the equation called compensation. For example, a child develops diarrhea and loses bicarbonate, leading to metabolic acidosis, which stimulates the respiratory centre. PCO2 is washed out and reduced, proportionate to the fall of HCO3 and a temporarily crisis is managed

- Lungs compensate for the metabolic alterations, and kidneys compensate for the alteration in PCO2.
- Compensation never crosses the midline. E.g., in compensation for metabolic acidosis, if pH is low, like 7.25, it will move up to 7.35 or beyond, but never 7.40 or beyond
- Lungs start the compensation within minutes, kidneys come for help a little late in hours to days, but finally settle the problem
- There are some formulas that help to assess the degree of compensation.

Table 1. Key for compensation in simple acid-base disorders

DISORDER	EXPECTED COMPENSATION
Metabolic acidosis (Winter's formula is used)	PCO2 = 1.5 x (HCO3) +8+-2
Metabolic alkalosis	PCO2 increases by 7 mmHg for every 10 mEq/mL increase in HCO3
Respiratory acidosis	
Acute	[HCO3] increases by **1** mEq for each 10 mm Hg increase in Pco2
Chronic	[HCO3] increases by **3** mEq for each 10 mm Hg increase in Pco2
Respiratory Alkalosis	
Acute	[HCO3] falls by **2** mEq for each 10 mm Hg decrease in Pco2
Chronic	[HCO3] falls by **4** mEq for each 10 mm Hg decrease in Pco2

Categories of metabolic acidosis based on anion gap (AG)

Metabolic acidosis occurs due to two basic mechanisms:

1. Loss of bicarbonate from the body
2. Impaired ability to excrete acid by the kidney or addition of acid to the body (exogenous or endogenous)
   By calculating the anion gap, we can identify the underlying mechanism. If an anion gap is normal, it is due to loss of bicarbonate, and a high anion gap results from the addition of acids or impaired ability to excrete acid
   Based on this, metabolic acidosis is categorized into 1. Normal anion gap acidosis (NAGMA)
3. High anion gap acidosis (HAGMA)

How anion gap (AG) is calculated?

The anion gap is the difference between the measured cation (Na+) and the sum of measured anions
(Cl− + bicarbonate). In other words, AG is also the difference between the unmeasured cations (K+, magnesium, calcium) and the unmeasured anions (albumin, phosphate, urate, sulfate).
Normal AG is 12 +/- 4. Usually, potassium is not included here.

$$\text{Anion gap} = [Na^+] - ([Cl^-] + [HCO_3^-])$$

The plasma anion gap is useful for evaluating a child with metabolic acidosis and categorizing into two groups, those with NAGMA and HAGMA.

How to do the correction of AG in the presence of hypoalbuminemia?

A normal anion gap is 4-12. A 1 g/dL decrease in the albumin concentration decreases the anion gap by approximately 2.5 mEq/L. Thus, if the albumin is not close to 4 g/dL, the anion gap should be corrected for the albumin concentration:
Anion gap (corrected for albumin) = Na+ − ([Cl −] + [HCO3 −]) + [2.5 x (4−observed albumin)]
In other words
Albumin probably contributes about 75% to the total AG value, which means hypoalbuminaemia should result in a smaller anion gap. For this, there are multiple possible equations, but the easiest to remember is the 4:1 rule: for every 0.4g/L decrease in serum albumin, the normal expected anion gap decreases by 1. Thus, at an albumin of 3.2 (fall of 0.8 g), the expected anion gap is 10 instead of 12; when the albumin level is 2.4 normal AG is 8. So, if the AG of a child is 10 when the serum albumin level is 2.4 is considered normal, but should be considered as HAGMA.

Box 1. Causes of metabolic acidosis

CAUSES OF METABOLIC ACIDOSIS

NAGMA:

1. a) Diarrhea b) Renal tubular acidosis (Both proximal and distal)

HAGMA:

a) Lactic acidosis: Tissue hypoxia – Hypoxemia, shock, anemia
IEM (organic academia, mitochondrial disorder
Medications: Propofol, linezolid, metformin, nucleoside reverse transcriptase inhibitor
Liver failure

b) Ketoacidosis: DKA, starvation ketosis, alcoholic ketoacidosis
c) Kidney failure
d) Poisoning: Ethylene glycol, methanol, salicylate, iron, toluene, carbon monoxide, cyanide

Clinical manifestation of metabolic acidosis before proving by ABG analysis

The etiology of a metabolic acidosis is often apparent from the history and physical examination e.g primary conditions mentioned in Box 1. In addition, respiratory response to metabolic acidosis is compensatory hyperventilation, It is clinically recognizable, though it may be confused with respiratory distress e.g Diabetic ketoacidosis is mistakenly diagnosed as asthma or wheeze associated with lower respiratory infection.

Table 2. Difference between effortless tachypnea and respiratory distress

Effortless tachypnea	Respiratory distress
Rapid and deep respiration without increased work of breathing (WOB)	Rapid respiration with increased WOB (stridor, wheeze or grunt, retractions, flaring of the ala nasi)
Causes: Fever, anxiety, metabolic acidosis, neurogenic hyperventilation	Causes: cardiorespiratory causes: Croup, Pneumonia, asthma, empyema, cardiac failure
Absence of noisy breathing or adventitious sounds or retraction	Above mentioned signs of increased WOB are present
SpO2 may be normal, as it is a non-respiratory pathology	SpO2 is expected to be low

Biochemical manifestations of metabolic acidosis

Low bicarbonate and low pH are the principal observations. PCO2 depends on the stage of compensation and whether it is simple or mixed.

Types of metabolic acidosis based on the degree of compensation

1. Acute uncompensated metabolic acidosis
2. Partially compensated metabolic acidosis
3. Completely compensated metabolic acidosis

Based on whether it is a simple or mixed disturbance

1. Simple metabolic acidosis
2. Metabolic acidosis with respiratory acidosis, under compensation
3. Metabolic acidosis with respiratory alkalosis, overcompensation
4. Double metabolic disturbances: a) Pure HAGMA b) HAGMA + NAGMA c) HAGMA with metabolic acidosis

Box. 2. Simple and mixed disturbances

Child with metabolic acidosis and HCO3 is 14 mEq/L
Apply Winter's formula for the expected PCO2
1.5 X 14 +8 +/- 2 = 21 +8 =29 = Expected PCO2 is 27-31 PCO2
If the measured PCO2 in ABG falls with in 27-31, it is a simple disturbance
If the measured PCO2 is < 27, it indicates overcompensation – Metabolic acidosis + respiratory alkalosis
If the measured PCO2 is >31, it indicates under compensation – Metabolic acidosis +respiratory acidosis.

When will you suspect mixed disturbances?

1. When both HCO3 and PCO2 independently account for the change of pH, or both move in opposite directions
   Eg. pH -7.0 HC03 -15 pCO2 - 50. Here both HCO3 and PCO2 claim they are responsible for acidosis. Usually, during compensation, both variables HCO3 and PCO2 move in the same direction. Here they are moving in opposite directions. HCO3 is falling, but PCO2 is rising. This is a clue for mixed disturbances.

2. A truly normal pH with distinctly abnormal HCO3- and PaCO2.
   Eg. pH 7.40, PaCO2 20, HCO3- 12
   Single acid-base disorders don't lead to a normal pH. Hence, there must be some hidden mixed disturbance

3. If a child with chronic renal failure (Chronic metabolic acidosis) develops vomiting, loss of chloride leads to a rise of HCO3 but AG remains high. This discrepancy - High AG and Normal HCO3 indicates the presence of associated metabolic alkalosis, a mixed disturbance of two metabolic abnormalities (double metabolic disturbances)

Double metabolic disturbances:

These are unusual situations. To quote an example, after initiating treatment for diabetic ketoacidosis, 24 to 48 hours later, NAGMA and HAGMA are seen because of chloride loading.

- Delta/Delta ratio needs to be calculated to identify this problem in every child with HAGMA. It is not routinely done in other abnormalities.
- Eg. If a child with chronic kidney disease (Chronic metabolic acidosis) develops vomiting, loss of chloride leads to a rise in HCO3 and metabolic alkalosis. But AG will remain high, because of the chronic metabolic acidosis.
- This discrepancy of high AG and normal HCO3 indicates associated metabolic alkalosis in a child in this situation

- HAGMA + NAGMA can be seen in CKD with diarrhea
- NAGMA + metabolic alkalosis will be seen in Barter's syndrome with diarrhea

How to check delta/delta ratio?

- Every patient with HAGMA must be evaluated to recognize additional metabolic disturbances

Step 1: First, calculate the anion gap, i.e., serum Na - (serum chloride + Bicarbonate).
E.g, sodium 143 – (Chloride 98 + bicarbonate 10 = 108). 140-108 = 32
Step 2: Delta AG is the difference between measured AG and Normal AG (12)
(Measured AG 32 -12= Delta AG is 20)
Step 3: Delta HCO3= Difference between Normal HCO3 and measured HCO3 (24 -10 =14)
Step 4: Calculating delta–delta ratio (Delta AG / Delta HCO3 is 20/14 = 1.4
What does this delta-delta ratio mean? (Table 2)

Table 2. Interpretation of delta/delta ratio

Delta Ratio	Assessment Guidelines
< 0.4	<0.4 = Pure anion gap acidosis
0.4 - 1	High AG & Normal AG acidosis (NAGMA)
1 to 2	Pure High Anion Gap acidosis (HAGMA) Lactic acidosis: average value 1.6 DKA more likely to have a ratio closer to 1 due to urine ketone loss
> 2	High AG acidosis and a concurrent metabolic alkalosis or a pre-existing compensated respiratory acidosis

Treatment of acidosis

The mainstay of the treatment is the effective treatment of the underlying disease, e.g, Insulin for DKA and IV fluid boluses and or steroids for shock correction. Because treatment of primary illness itself repairs acidosis, without the need for bicarbonate replacement. Bicarbonate is contraindicated in diabetic ketoacidosis, as it can lead to cerebral edema and hypernatremia

1. Management of primary illness

2. Bicarbonate: Oral bicarbonate or citrate therapy in children with chronic metabolic acidosis because the liver generates bicarbonate from citrate. Children with Type I or type II RTA may have hypokalemia and benefit from potassium supplements, but most children with chronic kidney failure cannot tolerate.

3. Sodium bicarbonate may be given as a bolus, usually at a dose of 1 mEq/kg, in an emergency diluted in 5% dextrose solution and can be given over one hour. Sodium bicarbonate can also be added to the patient's maintenance IV fluids. Whenever bicarbonate and IV calcium are given successively, give a normal saline flush to prevent precipitation in the IV infusion set.

4. Trishydroxymethyl aminomethane (tromethamine, THAM) is preferred in a child with a metabolic acidosis and respiratory acidosis, because it neutralizes acids without releasing CO2. THAM also diffuses into cells and therefore provides intracellular buffering. It is not freely available in our country

5. Hemodialysis is another option when there is renal insufficiency

What are the side effects of bicarbonate therapy?

a) Hypernatremia or volume overload b) overcorrection of the metabolic acidosis, because metabolism of lactate or ketoacids generates bicarbonate, c) rapid change from acidemia to alkalemia leads to hypokalemia and hypophosphatemia d) increased generation of CO2 worsens existing respiratory failure, requiring ventilatory support e) CO2 readily diffuses into cells and the intracranial compartment, potentially worsening cell function and cerebral edema.

Bicarbonate is indicated in the following situations

a. Metabolic acidosis caused by RTA or chronic kidney disease requires long-term bicarbonate therapy.
b. Patients with acute kidney injury and metabolic acidosis may need bicarbonate therapy until the kidneys' function normalizes.
c. In salicylate poisoning, bicarbonate administration increases renal clearance of salicylates. In other poisonings, such as ethylene glycol, methanol ingestions, also it is used
d. Along with correction of acidosis, patients with methanol or ethylene glycol ingestion should receive Fomepizole which prevents the breakdown of the toxic substance to its toxic metabolites. It is the preferred choice over ethanol. It works by inhibiting alcohol dehydrogenase, the enzyme that performs the first step in the metabolism of ethylene glycol or methanol.
e. Also useful in inborn errors of metabolism (pyruvate carboxylase deficiency, propionic acidemia)
f. Also used in the treatment of cyanotic spells

Approach to metabolic acidosis

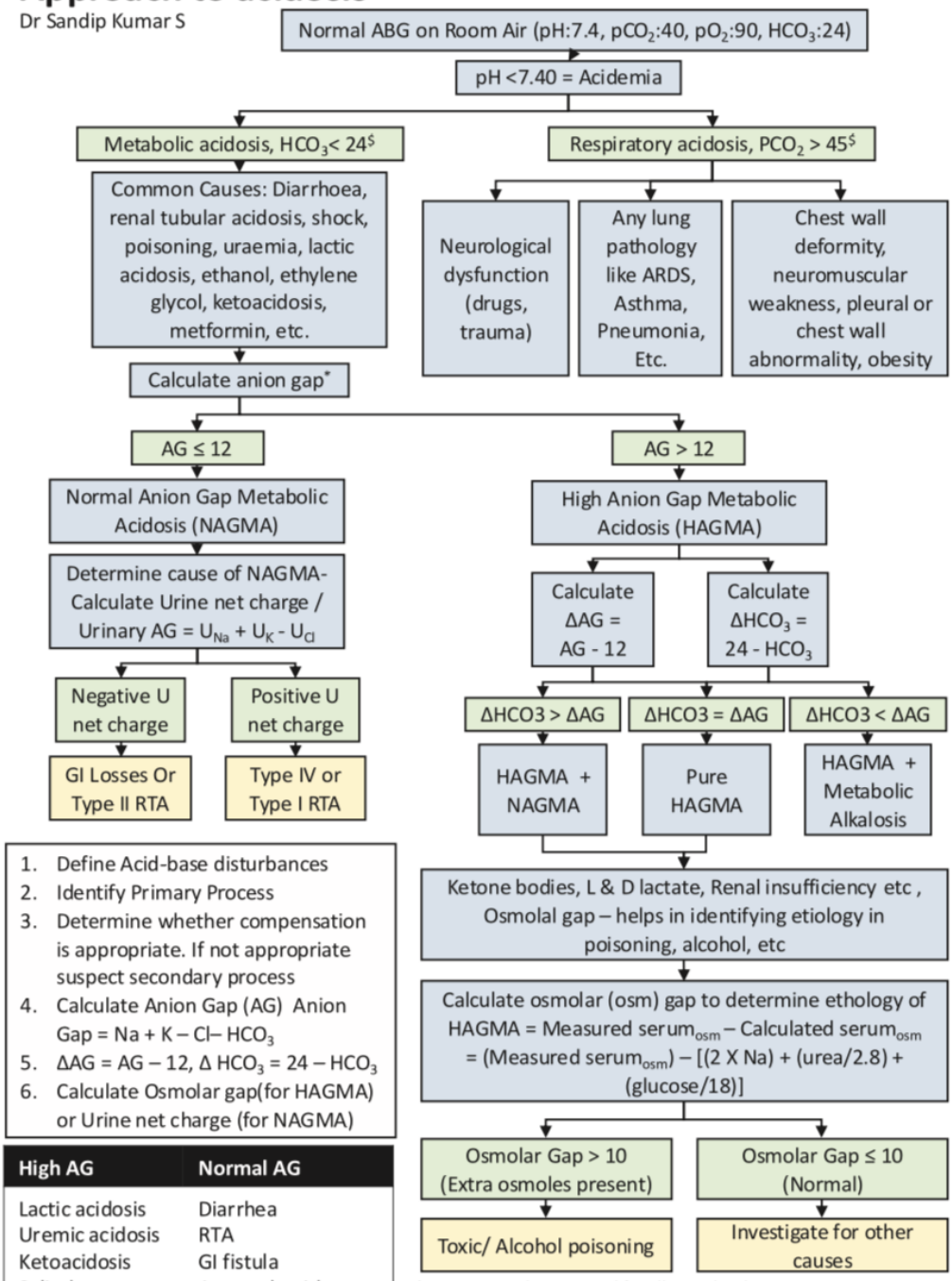
1. Suspect metabolic acidosis based on effortless tachypnea and confirm by Arterial blood gas analysis (ABG)
2. Is it respiratory or metabolic acidosis?
3. Whether it is NAGMA or HAGMA?
4. Is it uncompensated or compensated (older way of categorization)?
5. Is it a simple or mixed disturbance (current way of categorization)?
6. Whether mixed disturbances - metabolic and respiratory?
7. Or it is double metabolic, such as a) Pure HAGMA, b) NAGMA with HAGMA c) HAGMA with metabolic alkalosis
8. By calculating delta/delta gap, the type of double metabolic disturbance is identified
9. Management is treating the primary illness in all causes and in a few replacements of HCO3

For further reading

1. Nelson Text book of Pediatrics. 22nd edition. Chapter on fluid and electrolytes

Approach to acidosis

Dr Sandip Kumar S



1. Define Acid-base disturbances
2. Identify Primary Process
3. Determine whether compensation is appropriate. If not appropriate suspect secondary process
4. Calculate Anion Gap (AG) Anion Gap = Na + K – Cl– HCO_3
5. ΔAG = AG – 12, Δ HCO_3 = 24 – HCO_3
6. Calculate Osmolar gap(for HAGMA) or Urine net charge (for NAGMA)

High AG	Normal AG
Lactic acidosis	Diarrhea
Uremic acidosis	RTA
Ketoacidosis	GI fistula
Salicylate	Acetazolamide
Ethylene glycol	High NS load
Ethanol	Hyperalimentation

* Anion gap to be corrected for albumin levels.
Corrected anion gap = anion gap + 2.5 (4.5- serum albumin)
$ if the compensation is not appropriate, it will be considered acidosis even if PCO2 and HCO3 are normal

Case 1:

2year old boy previously normal, admitted with c/o fever, breathlessness 2 days. No cough/rhinorrhoea. H/o polyuria + for 10 days.

O/e conscious, alert, tachypneic, dehydration +. RS - clear, CVS -tachycardia+, no gallop. Abdomen soft, no organomegaly.
Inv- Blood sugar 540mg/dl, urea 40, creatinine 0.5, Na 142, K 3.7,
Cl-116, urine ketones +ve
ABG Ph 6.9, PO2 90, PCo2 16, Hco3 4,

Questions:

1. Identify the clinical condition
2. Interpret the ABG
   a. What is the primary problem, is it compensated?
   b. What type of metabolic problem is it?
3. Is there any other metabolic abnormality present?
3. What is the management?

Case 2:

One and half year-old child presents with h/o vomiting, abdominal pain, diarrhoea for 6 hours. Child was apparently normal before that. No h/o fever. Suddenly develops these symptoms.

Family h/o: this is the first child. Mother is 5 months pregnant now
O/e child is alert, tachypnea +, tachycardia +. Epigastric tenderness +.
RS, CVS normal.
Inv: Leucocytosis +, blood sugar 240 mg/dl, urine ketones -negative
ABG: Ph 7.1, Po2 98, pco2 36, Hco3 18.
Na 137, K 3.8, Cl 101
Xray abdomen shows small round multiple radio opaque shadows.

Questions:

1. What is this condition?
2. Interpret the ABG
   a. What is the primary problem, is it compensated?
   b. What type of metabolic problem is it?
   c. Is there any other metabolic abnormality present?
3. What is the management?

Case 3:

A 5-year-old child (20Kg) admitted with septic shock to ICU has received 30ml/kg NS bolus and is on inotropic support. His vitals are HR 150/min, RR 40/min, BP 74/34 mmHg. Temp 101 °F, SpO2 98% with room air. Bedside echo suggests moderate LV systolic dysfunction EF 45%, IVC collapsible, lung USG Normal. His blood gas suggests pH 7.25, pCo2 28, Hco3 14. His serum electrolytes are Sodium 142mEq/L, potassium 4 mEq/L, and chloride 112mEq/L. His serum albumin is 2.0 g/dL, Lactate 5 mmol/L. His urine output is 5ml/hr for last 6 hours.

Questions:

1. What is the cause of metabolic acidosis here?
2. Calculate the anion gap. (Albumin and calcium correction)
3. Does soda bicarbonate correct acidosis here?

Case 4:

3-year-old (weight 12 Kg) presented with high fever for 4 days with excessive vomiting, pain abdomen, poor oral intake and increased lethargy for last 2 days, 1 episode of bloody vomiting to ER. On examination HR 140/min, RR 40/min, BP 90/50mmHg, SpO2 96% RA, chest b/l clear, peripheral pulses were felt, with a tender hepatomegaly. History also revealed frequent use of Syp P 250 simultaneously with paracetamol -mefenamic acid combinations. Blood gas pH 7.25/pco2 28/ Hco315/base excess -8/Lactate 10mmol/L CBC Hb 14g/dL, WBC 3000/µL, plt 70,000/µL, Dengue Ns1 positive, Na 128/K 4.5/Cl 102.

Questions:

1. What is the cause of metabolic acidosis here?
2. How do we manage this acidosis?
3. Is there a role of renal replacement therapy here?
4. What are the causes of lactic acidosis?

Case 5:

2-year-old child, H/o fever, cough 7 days, difficulty breathing 3 days, decreased feeding 2 days, decreased UO last 12 hours, 6-year elder brother had URI a week ago. On examination vitals HR 150/min, RR 80/min, BP 80/50(55) mmHg, Severe respiratory distress, increased WOB, SpO2 60% Room air, peripheral pulses felt, irritable and confused look, On NRM SpO2 improved up to 86%. Blood gas showed pH 7.18/ pco2 48/Hco3 18/ Base excess -7/lactate 2.5 mmol/L. Glucose 100mg/dl. Na 135/K 3.8/ Cl 100/CBC 10/8000/3,00,000, CRP 80, SGOT 100/SGPT 40, Urea 20/0.3.

Questions:

1. Interpret the blood gas and probable primary cause.
2. How do we manage this acidosis?

Case 6:

4 months old infant presented with low grade fever, refusal to feed, persistent vomiting, and lethargy developed over 2 to 3 days. His family history is significant for consanguinity and previous one sibling death due to short febrile illness during infancy. O/E HR 160/min, RR 60/min, Systolic BP 70mmHg, CRT 2 sec, SpO2 96% RA, Chest b/l clear, no WOB, no murmur,

peripheral pulses well felt, drowsy sensorium, minimal response to pain. Blood gas pH 7.05/pCo2 12/ HCO3 6/ Base excess -15, Lactate 3 mmol/L. Glucose 70mg/dL. Ammonia-600 mg/dL. Na 126/K 5/ Cl 96/CBC 8/2400/3,00,00 CRP 80, SGOT 30/SGPT 20, S. Alb 3.5, Urea 40/0.7.

Questions:

1. Interpret the blood gas and probable cause?
2. What is osmolar gap
3. How do we further work up this case?

HYPERNATREMIA

Contributors: Dr. Anitha V P, Dr. Venkateshwari R, Dr. Chidhambharam L, Dr. Prasanna R

Hypernatraemia - Ready reckoner

Classified as Mild (146-149 mmol/L)
Moderate (150-169 mmol/L)
Severe (≥170 mmol/L)

Pathophysiology:

- HYPERNATREMIA = INTRAVASCULAR VOLUME DEPLETION (Most common)
- Imbalance in the body's handling of water, with a relative increase in plasma osmolality
- Moderate to severe hypernatraemia can cause acute brain shrinkage with vascular rupture, haemorrhage, demyelination and permanent neurological injury
- Infants are at greater risk due to insensible water loss through a larger surface area and inability to access water
- Chronic hypernatraemia (>48 hours) is better tolerated due to cerebral compensation by idiogenic osmoles which return the cells to normal size and thus if corrected rapidly cerebral oedema
- Whereas with gradual correction, the idiogenic osmoses gradually extrude out of cells retaining the cell shape, with normalisation of serum osmolality.

Causes:

- Water loss that is not replaced
- Excess salt intake, relative to that of water

- Water deficit
  - Common:
    - Gastrointestinal loss e.g., diarrhoea, stomal losses
    - Skin loss (excess sweating/burns)
    - Renal losses e.g., osmotic diuretics, diabetes mellitus, polyuria of acute tubular necrosis
  - Less Common:
    - Diabetes insipidus (central, nephrogenic, systemic disease, drugs)
    - Impaired thirst mechanism secondary to underlying neurological abnormalities or hypothalamic dysfunction

- Sodium excess
  - Ingestion of high sodium (inappropriate formula concentration, high osmolality rehydration solutions, salt poisoning)
  - Iatrogenic (hypertonic saline, sodium bicarbonate, Antibiotics with a High sodium Content-Cephalosporins, Penicillins)
  - Hyperaldosteronism - Primary (Conn's), Secondary (CCF, nephrotic syndrome, steroids)

Assessment:

History will elicit the most likely cause of Hypernatremia

Clinical examination includes assessing the degree of dehydration and the effects of hypernatremia.

Hydration assessment is unreliable due to the movement of water from intracellular to extracellular compartment in an attempt to restore the osmolality.

Skin has a typical doughy feel.

Look for sacral edema, or periorbital puffiness, or peripheral edema, which points to sodium excess as the cause.

Initial signs of hypernatremia are nonspecific and include irritability, restlessness, weakness. This is followed by muscle twitching, fever, vomiting, high pitched cry in infants. Severe signs include seizures, hyperreflexia and coma.

Investigations:

Include other electrolytes, blood glucose
Paired serum and urine sodium, creatinine and osmolality, as far as possible, which helps in identifying the source of loss.

- Renal losses / Sodium gain - Urine Na > 20 mEq/L and FeNa > 2%
- Extrarenal losses - Urine Na < 20 mEq/L and FeNa < 1%
- Urine osmolality < serum osm indicates urine concentrating defect as in DI, osmotic diuresis, renal disease
- Urine osm > se. Osm indicates intact urine concentrating defect, extra renal losses or excess sodium gain.

Treatment:
Shock should be addressed as a priority and corrected with isotonic fluids
Management of hypernatremia includes management of sodium and management of water deficit
Mild hypernatremia - treat the cause and repeat electrolytes after 24 hours
Moderate hypernatremia -Due to water loss
Total fluid requirement = maintenance + replacement of deficit + replacement of ongoing losses
Acute symptomatic hypernatremia (<48 hours)
Sodium corrected to achieve a reduction by 3-5 mEq/l over 6 hours with a maximum reduction of 10-12 mEq/l in 24 hours
Chronic hypernatremia, of > 48 hours duration is corrected more gradually, with a rate of drop of not more than 0.5 per hour or 8-10 mEq/l per day.
Severe hypernatremia is corrected over many days.

Deficit replacement is corrected with isotonic fluids (which is hypotonic relative to the hypernatremic hyperosmolar serum). Rehydration itself will result in a drop in Se. Na.

Free water deficit is calculated as per the formula and corrected over 48-72 hours.
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)
Adrogue Madias formula predicts the change in Na achieved with 1 litre of chosen fluid

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

Infusate Na refers to the sodium content of the fluid chosen (75 mEq if 0.45% saline is chosen)
Total body water is 0.6 x body water

Replacement of ongoing losses is done ml for ml with a fluid tonicity which matches the losses.

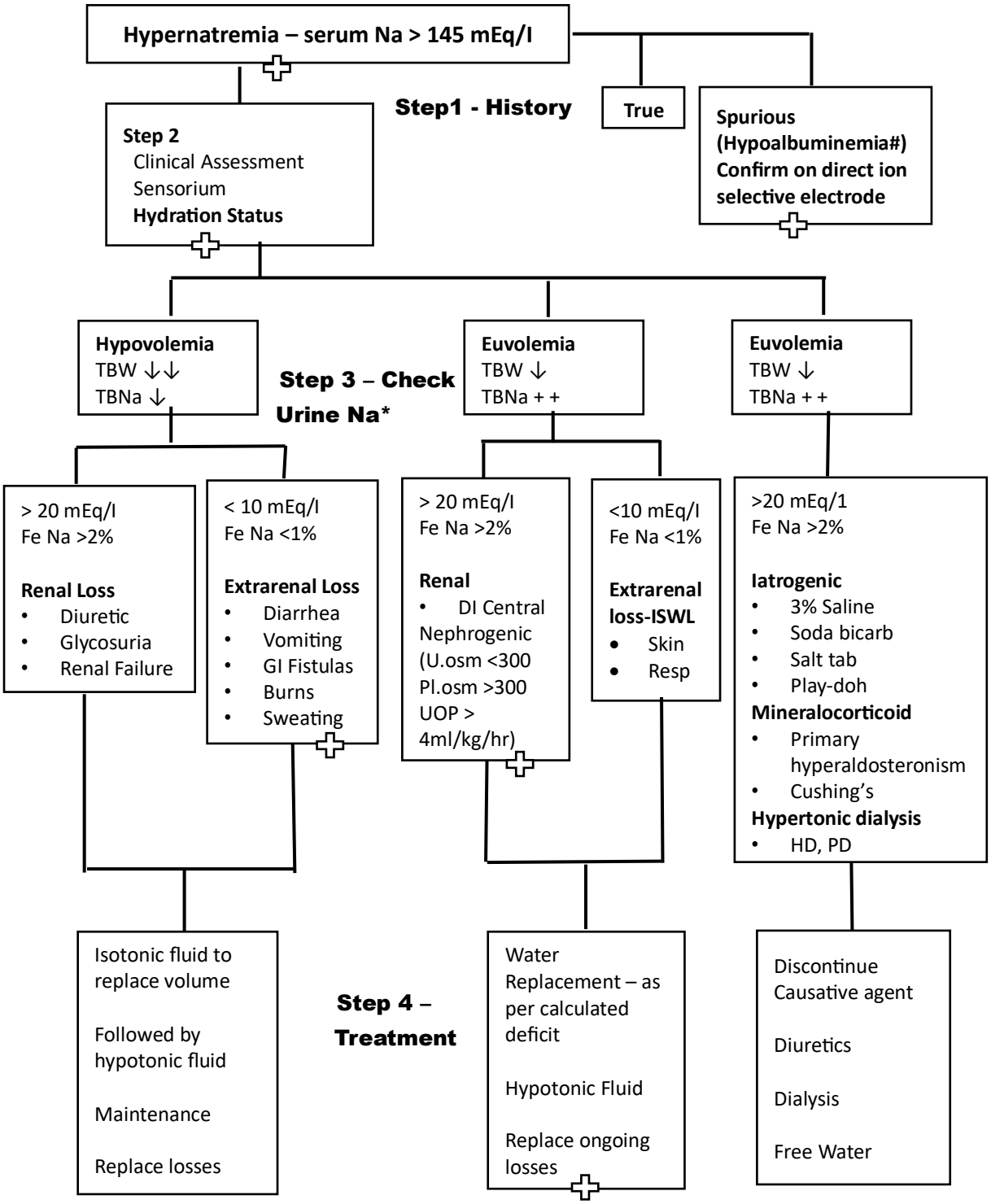
If seizures occur, get a CT brain with contrast, give 3% saline 3-5 ml/kg to raise the Se. Na by 5 mEq/l and aim for a slower drop in Se. Na

If Se. Na is dropping rapidly, slow the fluid rate or increase the tonicity.

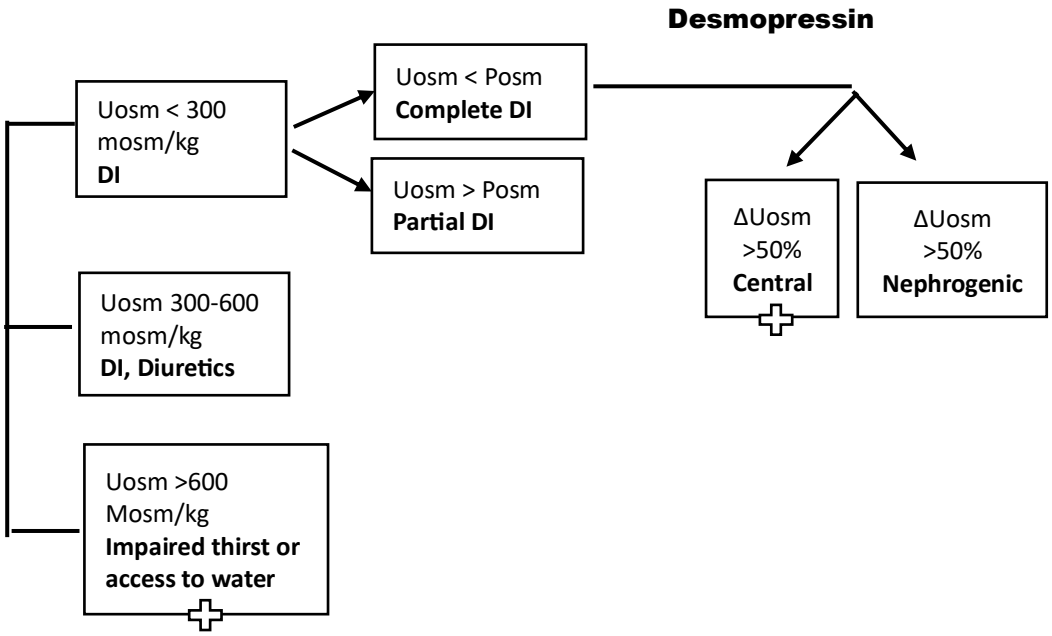
If Se. Na is not dropping, increase fluid rate or decrease the tonicity.
Manage DI and other endocrine causes with expert inputs.

Due to sodium excess - Decrease the sodium intake.

Refractory cases may require Renal replacement therapy


Hypernatremia – serum Na > 145 mEq/l
Step1 - History
True
Spurious (Hypoalbuminemia#) Confirm on direct ion selective electrode
Step 2
Clinical Assessment
Sensorium
Hydration Status
Hypovolemia
TBW ↓↓
TBNa ↓
Step 3 – Check Urine Na*
Euvolemia
TBW ↓
TBNa + +
Euvolemia
TBW ↓
TBNa + +
> 20 mEq/l
Fe Na >2%
Renal Loss
• Diuretic
• Glycosuria
• Renal Failure
< 10 mEq/l
Fe Na <1%
Extrarenal Loss
• Diarrhea
• Vomiting
• GI Fistulas
• Burns
• Sweating
> 20 mEq/l
Fe Na >2%
Renal
• DI Central Nephrogenic (U.osm <300 Pl.osm >300 UOP > 4ml/kg/hr)
<10 mEq/l
Fe Na <1%
Extrarenal loss-ISWL
• Skin
• Resp
>20 mEq/1
Fe Na >2%
Iatrogenic
• 3% Saline
• Soda bicarb
• Salt tab
• Play-doh
Mineralocorticoid
• Primary hyperaldosteronism
• Cushing's
Hypertonic dialysis
• HD, PD
Isotonic fluid to replace volume
Followed by hypotonic fluid
Maintenance
Replace losses
Step 4 – Treatment
Water Replacement – as per calculated deficit
Hypotonic Fluid
Replace ongoing losses
Discontinue Causative agent
Diuretics
Dialysis
Free Water

Step 5 – Check Urine Osmolality



Case Scenarios:

Case 1:

5 years old child had recently joined football coaching during his summer vacation. His mother was concerned about him getting dehydrated and asked him to drink 5 packets of ORS diluted in one litre of water. The next day, mother noticed that child was lethargic and sleeping excessively. Hence, she brought the child to ER.
On examination, child was drowsy. His perfusion and blood pressure were normal. Mild periorbital puffiness and tachypnea was present. His weight was 15kg.

Labs showed the following:
Urea: 28mg/dl, Creatinine: 0.6mg/dl, Na: 170 mEq/dl, K: 3.5 mEq/dl, Cl: 110 mEq/dl, HCO3: 20 mEq/dl.

Questions:

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

Case 2:

A 6-year-old child with craniopharyngioma underwent surgery and was shifted to the PICU postoperatively on ventilatory support.
12 hours later:
- Heart rate: 155/min
- Respiratory rate: 34/min
- BP: 80/46 mmHg
- On sedation due to ventilatory support
- Urine output: 10 ml/kg/hr over the last 3 hours

Investigations:
- CBG: 80 mg/dl
- Serum sodium: 159 mEq/L
- Serum potassium: 3.8 mEq/L
- Urea: 55 mg/dl
- Creatinine: 1 mg/dl
- Serum osmolality: 346 mOsm/kg

Questions:

1. What is the inference from the above case scenario?
2. How do we differentiate central from nephrogenic diabetes insipidus?
3. What are the main causes of hypernatremia?
4. What are the clinical manifestations of hypernatremia in children?
5. How is hypernatremia managed?
6. What is the role of Adrogue–Madias equation in hypernatremia management?

Case 3:

8 Months old male infant has (Wt: 10 kg)
history of fever, loose stools for 3 days with weight loss weight loss. Baby is irritable and with reduced urine output since one day and there is history of posturing today lasting for 3 minutes before being rushed to the ER. On examination Baby has bulging anterior fontanelle, intermittent posturing present, Skin is doughy. Vitals: baby HR: 180, BP: 50/35 With a temperature of 101 f. Mother revealed improper mixing of 21gm ORS packet in 200 ml water

- **Possible Diagnosis and reason for bulging AF?**
  Investigations: Sr.Na : 169 Meq/L , Urea : 70 mg/ dl , Creatinine : 0.9 mg/dl

↓

- **Next plan of action? What fluid and what Rate?**
  200 ml NS bolus has been given

↓

- **What is the Adrogue – Madias formula and formula to calculate Free water deficit. For hypernatremia correction. What fluid and at what rate u want to give**

↓

Baby was started on ½ NS – 44, NS- 25 ml/hr and kept NPO as sensorium was irritable. Repeat Serum Na -4 hrs later – 169.

- **What is the next treatment plan?**

↓

12 hrs into admission – serum Na: 165 Meq/L. Baby sensorium improved – baby was allowed top up feeds – 40 ml per hr and was on ½ NS – 30 ml per hr. Baby did not pass urine and was tachypneic.

↓

- **What is happening. What is the further treatment plan considering child is oligoanuric?**

HYPOCALCEMIA

Contributors: Dr. Gowrishankar NC, Dr. Kalpana S, Dr. Vidhya P S, Dr. Karthikeyan M

Calcium

- Hyper- and hypocalcemia - physiologically important electrolyte derangements resulting in short and long-term sequela for children

- Calcium is a crucial component of many physiologic processes like nerve conduction, blood coagulation, hormone secretion

- Total body calcium -1000 -1200 g
  - 99% store: skeletal calcium phosphate complexes/hydroxyapatite (serves - dynamic rapidly exchangeable pool to maintain serum calcium levels)
  - 1%: intra& extracellular fluid (half in active ionized form; remaining inactive(bound): to proteins (mostly albumin) /complexed with anions including phosphate, bicarbonate, lactate

- Normal S. Calcium: 8.8 - 10.4 mg/dL; ionized: 4.4 - 5.4 mg/dL

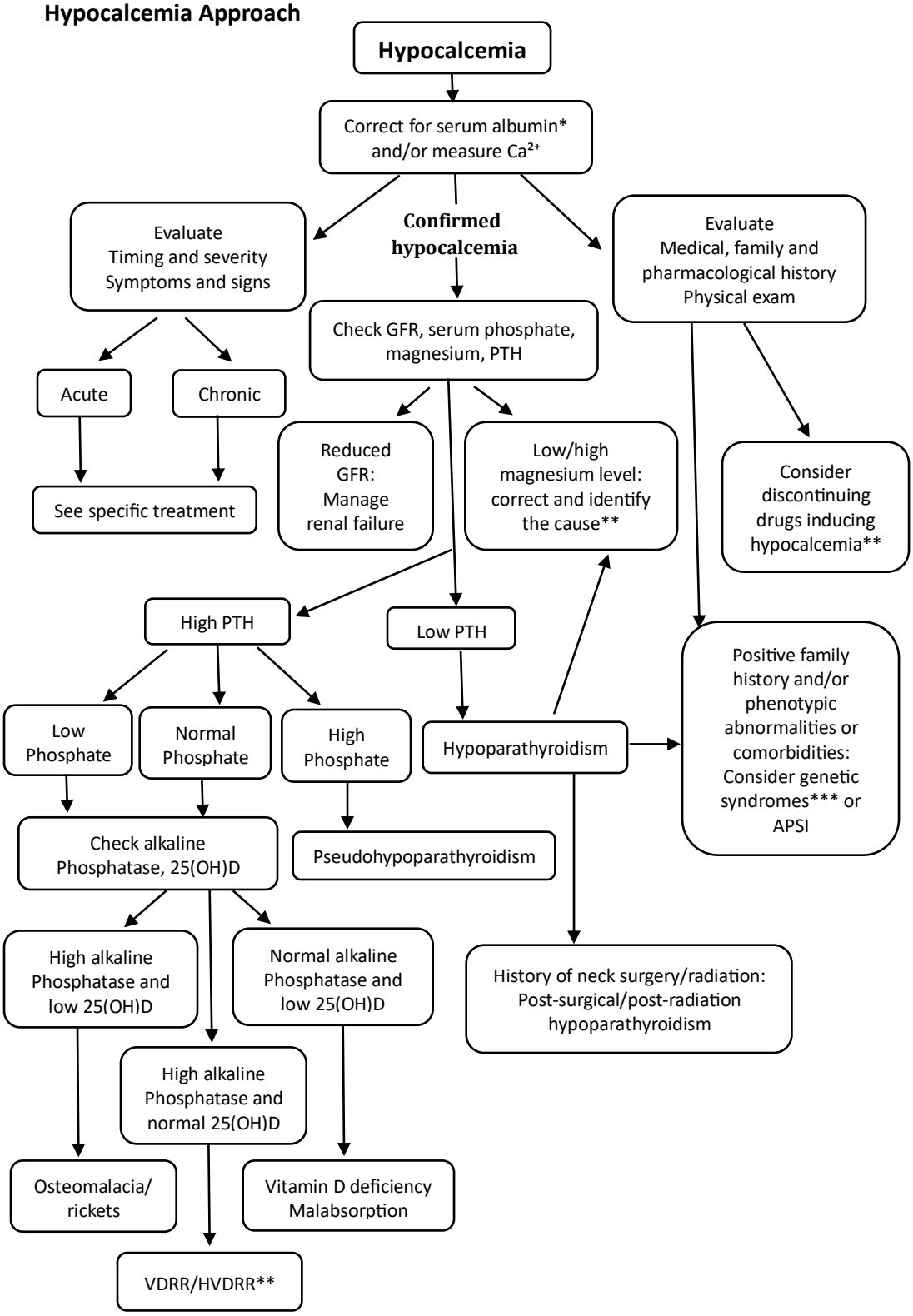
- Calcium homeostasis by hormonal & physiological factors - coordination with intestine, kidney, bone

- Tightly regulated processes- to serum ionized calcium -monitored by
  - parathyroid gland's calcium-sensing receptor (CasR)
  - actions - calciotropic hormones parathyroid hormone (PTH), active vitamin D (1,25(OH)2D) or calcitriol

HYPOCALCEMIA

- low total serum calcium levels below age specific normative values
  - Pre-term new-borns < 7.0 mg/dl
  - Term new-borns <8.0mg/dl
  - Children <8.8 mg/dl
- Hypocalcemia - often nonspecific symptoms- severity of symptomatic hypocalcemia can vary widely
  - in young: poor feeding, lethargy
  - in older patients: irritability, muscle twitching, jitteriness, tremors
- More severe symptoms: tetany, seizure, cardiac arrhythmia
- Chronically low serum calcium levels: short stature, rickets, brittle nails, and dry skin & hair

Hypocalcemia Approach



Step 1: Correct for serum albumin +/- ionic calcium – confirm hypocalcemia

Step 2: Timing and severity of symptoms + signs of hypocalcemia: treatment and further treatment based on acute / chronic

Step 3: Treat: raise serum calcium to normal level – minimize symptoms and investigate

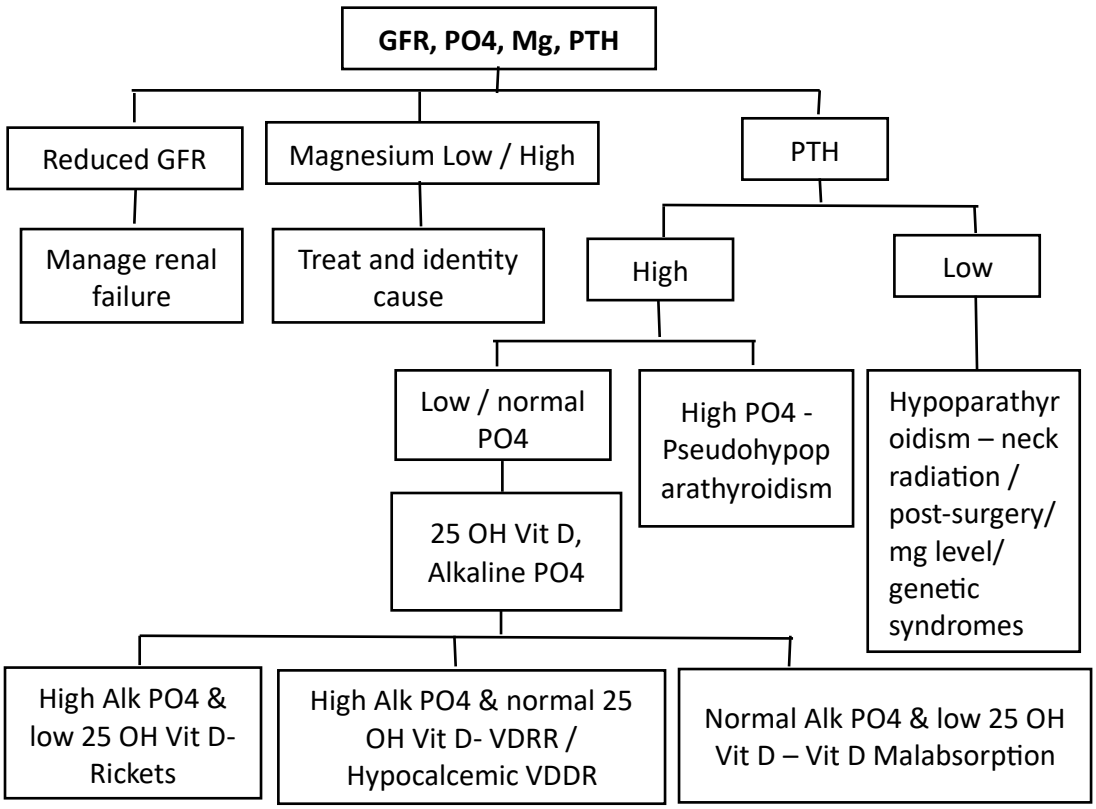
Step 4: IV calcium gluconate (1g= 93 mg elemental Ca) / IV calcium chloride (1g= 100-300 mg elemental calcium) - infusion

Hypomagnesemia associated hypocalcemia –

Mild: oral

Severe: IV MgSO4: 1 g MgSO4(8mEq) raises Mg level by 0.15mEq/L in 18-30 hrs as bolus and followed by infusion for 24 hrs in adults

- Good history including medication and family history and a good clinical exam
- Family history and phenotypic abnormalities: genetic syndromes
- Discontinue drugs causing hypocalcemia
- Next evaluation
- Check GFR, serum phosphate, serum magnesium, parathyroid hormone



Management

- Immediate management depends on the severity of clinical symptoms
- Severe clinical manifestations -seizures, broncho- or laryngospasm, tetany, mental status changes, impaired cardiac contractility, and/or prolongation of the QT interval
- Initial treatment - IV calcium administration until symptoms resolve & oral replacement can be initiated
- Typical infusions - 20 mg/kg elemental Ca over 10 to 20 min (equivalent approximately 0.7 mL/kg of 10%Ca chloride or 2mL/kg of 10% Ca gluconate)
- Other dosing regimens: calcium chloride:
    - for cardiac arrest: 20 mg/kg/dose (maximum dose: 2000 mg)
    - for tetany, give 10 mg/kg/dose over 5–10 min
- IV calcium gluconate –for symptomatic children
- 100 to 200 mg/kg/dose over 5–10 min followed by continuous infusion of 200 to 800 mg/kg/day
- Rapid infusion rates of IV calcium may precipitate bradycardia and cardiac arrest
- Calcium gluconate IV rates - not exceed 100mg/min
- Close monitoring for cardiac arrhythmia any IV calcium administration
- Calcium gluconate preferred over calcium chloride - especially in emergent situations - peripheral lines preferred as there is less tissue necrosis if extravasation occurs
- Avoid fluids - with phosphorous/ bicarbonate - to avoid precipitation of parenteral calcium
- Oral calcium and active vitamin D therapy - initiated as soon as possible

Oral management

- Elemental calcium 50–100 mg/kg of in 3–4 divided doses
- Vitamin D replacement – vitamin D3 at 150,000–600,000 units by IM once -severe vitamin D–deficient rickets
- Multiple oral vitamin D replacement regimens
    - 2000–10,000 units per day
    - 50,000 units per week
    - 50,000 units per month
- rechecking serum 25(OH) vitamin D levels after 3 months of therapy - if normal - conversion to maintenance doses (400– 1200 units/day depending on age)
- In addition to treating vitamin D deficiency -magnesium deficiency should be corrected - success of treatment of hypocalcemia - limited in cases of coexisting hypomagnesemia

Kusumi K, Narla D, Mahan JD. Evaluation and Treatment of Pediatric Calcium Disorders. Current Treatment Options in Pediatrics. 2021 Jun;7(2):60-81.

Case Scenarios

Case 1:

A 14-day-old male neonate, born at 30+2 weeks gestation, birth weight 1.1 kg, currently in NICU, presents with new-onset jitteriness and intermittent apnea. He was on CPAP for 48 hours, now on room air. Enteral feeds were started on day 3 and gradually advanced. He is currently on fortified expressed breast milk (EBM) and gaining weight slowly.

On examination:

- HR: 158/min, RR: 52/min, Temp: 36.7°C
- Jittery movements noted during handling
- Mild abdominal distension, active bowel sounds
- CNS: Alert, no focal deficits
- No dysmorphism, no hepatosplenomegaly
  Serum calcium is 7.9mg/dL

Questions:

1. What clinical features suggest hypocalcemia?
2. What are the risk factors for late-onset hypocalcemia in preterm neonates?
3. List essential biochemical tests.
4. Outline acute and maintenance therapy.
5. What role does vitamin D play here?
6. How would you adjust feeding and fortification?

Case 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 are the additional history you would like to elicit?
2. What are the investigations you would like to send as first line?

3. What is the management of nutritional rickets?
4. When do you suspect non nutritional rickets?
5. How do you approach hypocalcemia and rickets?

Case 3:

9 years old female child presented to ER with GTCS, preceded by history of muscle cramps and tingling around the mouth for past 1 week. Mother gives a history of mild motor and speech delay. There is no past medical illness, on normal diet and not on any medications.

On examination, she has a round face, short stubby fingers and weighs 38kgs (at 90th centile), height is 117cms (<3rd centile). Her mother also appears short.

Her initial labs show ionized calcium of 0.5 mmol/L with normal CBC and renal function tests.

Questions:

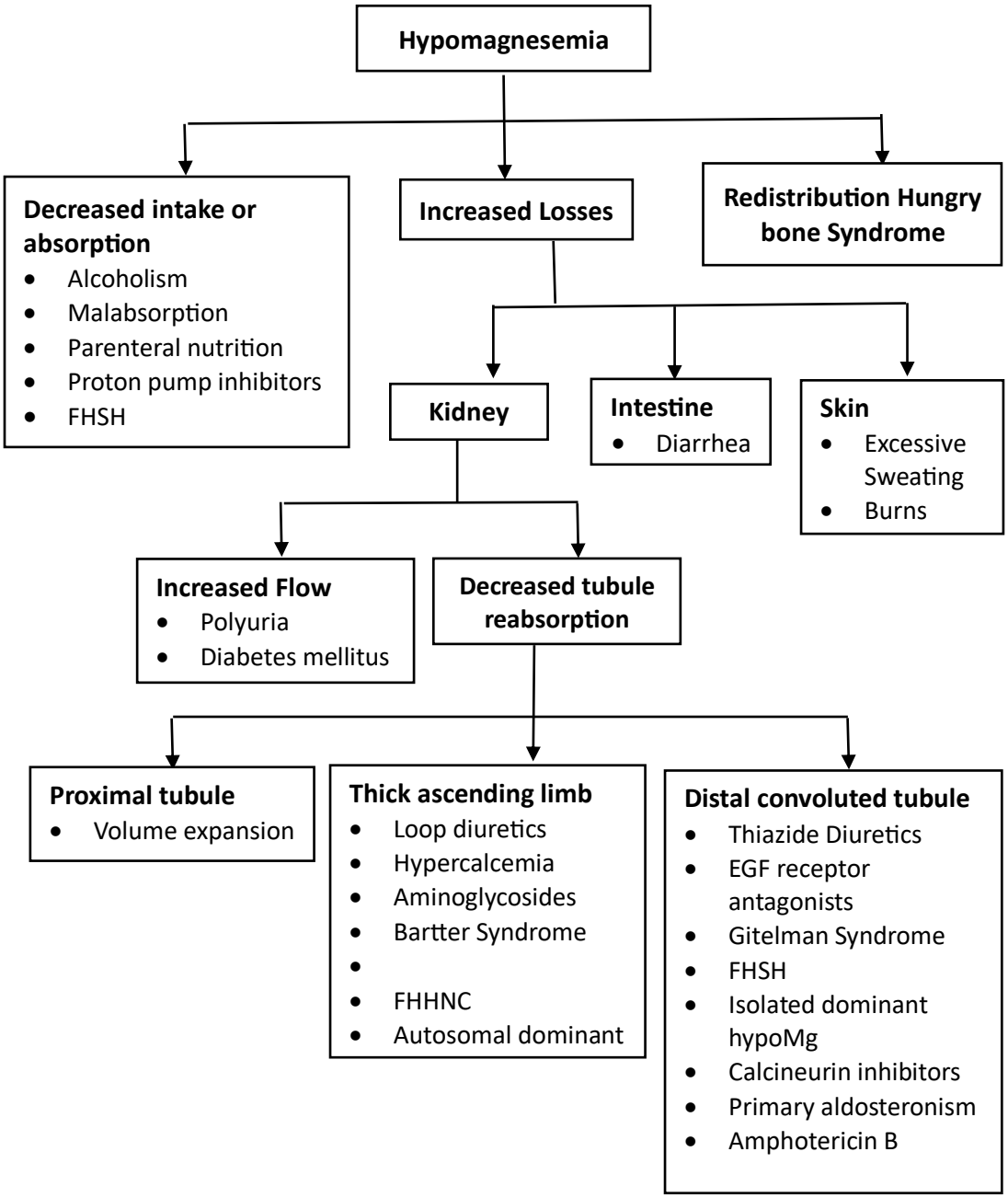
1. What are the signs and symptoms of hypocalcaemia in children?
2. What are the ECG manifestations of Hypocalcaemia?
3. How will you manage acute symptomatic hypocalcaemia and chronic hypocalcaemia?
4. How will you evaluate this child?

HYPOMAGNESEMIA

Contributors: Dr. Gowrishankar NC, Dr. Kalpana S, Dr. Vidya K, Dr. Karthikeyan M

Magnesium

- Measurement of serum magnesium concentration - not included in routine chemistry laboratory testing
- Abnormalities from low serum levels and body stores of magnesium -potentially serious and life-threatening consequences
- Some forms of hypomagnesemia -preventable and may require immediate-, short- or long-term management
- Hypomagnesemia has garnered less attention than other forms of electrolyte disturbances - appearance likely to be overlooked, potentially resulting in delayed management.
- Magnesium -important role in many biochemical processes
    - Synthesis of proteins and nucleic acids
    - binding of ATP to ATP-dependent enzymes



FHHNC familial hypomagnesemia with hypercalciuria and nephrocalcinosis,

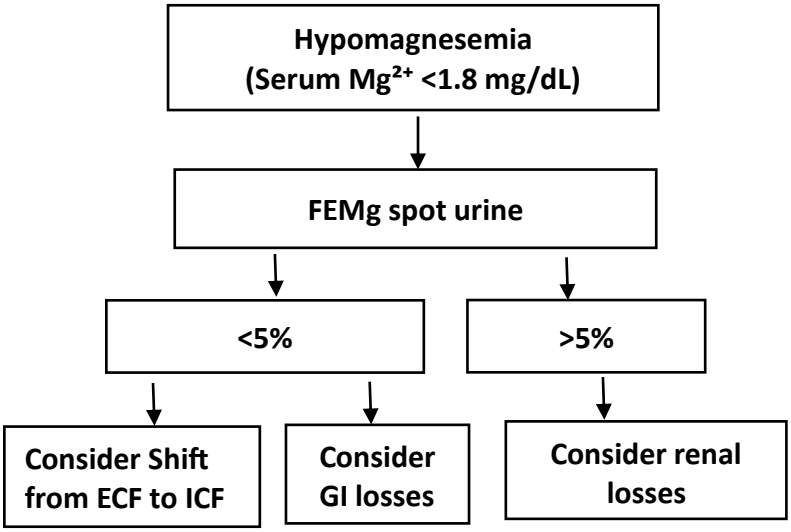
IDH isolated dominant hypomagnesemia

FHSH - Familial hypomagnesemia with secondary hypocalcemia

Treatment

- Oral/enteral dosing: oral magnesium replacement in asymptomatic children with mild hypomagnesaemia, unless significant gastrointestinal intolerance (eg diarrhoea) which oral magnesium will exacerbate
  - Dose: 2.5 - 5 mg/kg (0.1 - 0.2 mmol/kg) 3 times daily orally
  - Increase to 10 - 20 mg/kg (0.4 - 0.8 mmol/kg) up to 4 times daily orally if required
  - Tolerance is better with smaller, more frequent dosing

- **Intravenous dosing:** Children with severe symptoms (eg tetany, arrythmia, seizures)
  - Dose: IV magnesium 0.1 - 0.2 mmol/kg up to 0.4 mmol/kg (max dose 8 mmol)- administer over 2-4 hours, (reduces risk of adverse effects, improves cellular uptake of administered dose)
  - In children with severe symptoms, can be given over shorter period of time



Case Scenario:

Case 1:

2-year-old girl, 2nd born to 2nd degree consanguineous marriage was evaluated elsewhere for complaints of increased frequency of micturition and frequent episodes of vomiting for past 2 months.

- History of death of elder sibling due to renal failure at the age of 8 years present, but other details were not available.
- Usg abdomen done revealed nephrocalcinosis and hence child was referred to tertiary care centre for further evaluation.
- Child had a weight of 9.5 kg and length of 85 cm.
- Basic evaluation revealed the following.
- Uro-sepsis work up was negative.
- Bl urea 24 mg/dl
- Sr creatinine 0.6 mg/dl
- Sr sodium 141 meq/l
- Sr potassium 4.1 meq/l
- Sr calcium 9.8 mg/dl
- Sr vitamin D 24 ng/ml
- Sr PTH was 570 pg/ml
- Sr Magnesium was 0.8 mg dl
- Sr uric acid 4.5 mg/dl

Questions:

1. What is the step wise approach to evaluate for the cause of Hypomagnesemia?
2. What additional investigations are needed at present?
3. What will be the treatment for acute symptomatic and chronic hypomagnesemia?

HYPO/ HYPERKALEMIA

Contributors: Dr. Shanthi S, Dr. Shyamala J, Dr. Deepika S, Dr. Naresh Kumar S

Potassium

- Total body K -50mEq/Kg. Predominantly an intracellular cation. 98% is in the intracellular compartment and majority in the skeletal muscle.
- The normal serum level is 3.5-5.5mEq/L. Higher levels may be seen in newborns and young infants.
- Potassium is essential for growth, to maintain the resting membrane potential of skeletal, smooth, cardiac muscle and nerves. It also helps to regulate cellular volume as well as intracellular calcium content.
- 90% excreted in urine and 10% GIT, sweat. Most of the filtered K is absorbed. In the distal convoluted tubule and cortical collecting duct K is secreted into the tubular lumen in exchange with Na and H ions.

HYPOKALEMIA

Definition

- Severe hypokalemia – Potassium level less than 2.5 mEq/L
- Moderate hypokalemia – Potassium level between 2.5 and 3 mEq/L
- Mild hypokalemia – Potassium level between 3 and 3.5 mEq/L

Etiology of hypokalemia

Decreased intake	Increased intracellular uptake	Increased loss-extra renal	Increased loss-renal	Endocrine
Severe acute malnutrition Anorexia	Metabolic alkalosis Insulin Beta adrenergic agents Heavy metals(barium) Anti -psychotic drugs Hypokalemic periodic paralysis	Diarrhea Emesis Cystic fibrosis	Diuretics DKA Tubulo interstitial disease Bartter syndrome Gitelman syndrome Renal tubular acidosis Amphotericin Liddle syndrome Hypomagnesemia	Aldosterone-secreting adenoma Glucocorticoid remediable aldosteronism Apparent mineralocorticoid excess (AME) 11-beta-hydroxylase deficiency 17-alpha-hydroxylase deficiency Thyrotoxicosis

Clinical features

Many patients are asymptomatic. If severe hypokalemia, can present with muscle weakness (headlag, hypotonia, paralysis, respiratory failure, death) cramps, fasciculation and arrhythmias

ECG changes

PR prolongation, flattening of T waves, ST depression, U waves can emerge after the T waves (best seen in the precordial leads).

Management

Emergent treatment is needed in symptomatic patients, or those with ECG changes or severe hypokalemia

- Potassium chloride IV 0.5 to 1 mEq/kg of body weight per hour. The goal is to raise the potassium level by 0.3 to 0.5 mEq/L. May be associated with pain and phlebitis when administered through a peripheral vein. Choose a large vein. External jugular vein is a good option. Maximum adult dose is 40 mEq.
- Do NOT administer undiluted or by IV push. It must always be **diluted in infusion fluid** (RL or 0.9% sodium chloride).
- **Rapid intravenous administration or overdose may cause cardiac arrest. Administer via an infusion pump.**
- An infusion with a potassium concentration of no more than 40 mEq/L is given in most situations. Occasionally a higher concentration of 60mEq/L may be needed.
- When adding potassium chloride to an IV fluid bag, mix well by inverting the bag at least 10 times
- Clearly label all bags, syringes, pumps and lines that contain potassium to avoid inadvertent flushing
- Continuous ECG monitoring is needed.
- Serum concentrations should be evaluated 1 to 2 hours after completion of infusion
- May repeat dose as needed based on lab values
- Watch for rebound hyperkalemia
- IV fluids should not contain dextrose as it can stimulate insulin secretion.

Note: 1 ml of KCl contains 2 mEq.

Asymptomatic patients

Stop diuretics/laxatives and drugs which result in hypokalemia. Use potassium-sparing diuretics if diuretic therapy is required.

Treat underlying cause - Diarrhea or vomiting.

Replace ongoing excessive losses.

Moderate hypokalemia: Oral replacement. IV only for those who are unable to take oral medications.

Mild hypokalemia

Increase dietary potassium. Oral K supplements
For those who are unable to take enteral potassium, the addition of a maintenance amount of potassium to IV fluids 20mEq/L is sufficient

In asymptomatic patients with chronic hypokalemia (RTA) potassium supplementation may be needed

Oral potassium is preferred over IV potassium in asymptomatic patients. Potassium chloride, phosphate, potassium acetate, potassium citrate-citric acid, and potassium bicarbonate are the various salts available. Potassium chloride is commonly used. Patients with acidosis can be given potassium acetate or citrate.

Dose:

Initial: 1-2 mEq/kg/day in divided doses. Titrate to desired clinical response. Usual range: 1 to 5 mEq/kg/day. Not to exceed 1 to 2 mEq/kg as a single dose up to 40 mEq/dose

Note:

- The strength of K in most commonly available potassium chloride syrup is 20 mEq in 15 ml
- Oral and parenteral potassium can safely be used simultaneously.
- Best taken with or soon after food to reduce gastrointestinal irritation.

Other treatment:

Magnesium sulphate if hypomagnesemia is the cause. (25-50mg/kg IV over 30 minutes)
Potassium-sparing diuretic such as amiloride in Bartter, Gitelman
Spironolactone or eplerenone in hyperaldosteronism

Approach

Step 1 - **Is it true or spurious hypokalemia?**

Spurious hypokalemia can occur due to sampling errors - Recent line flush, IV fluids near sampling site. It can also occur if the WBC count is very high as in leukemia, if plasma for analysis is left at room temperature where the K is taken up by the cells.

Step 2 - **If true hypokalemia, is hypokalemia severe or symptomatic?**

If yes take a 12 lead ECG and start on continuous cardiac (ECG) monitoring. Initiate treatment.

- Treatment- Care of ABC, fluid boluses may be needed if volume depleted, IV potassium. Once K levels normalize evaluate.

Step 3 - History and clinical examination to identify the etiology

History

- Polyuria, polydipsia (impaired concentrating capacity due to hypokalemia or DKA)
- Loose stools, emesis
- Drug intake- beta adrenergic agonists, insulin, diuretics
- Renal disease
- Dark urine (rhabdomyolysis, myoglobinuria)
- Symptoms of thyrotoxicosis
- Family history (Bartter syndrome, Gitelman syndrome, familial hypokalemic periodic paralysis)

Clinical examination

HR (bradycardia) and rhythm, blood pressure, muscle tone, reflexes, nutritional status and volume status

Step 4 - **Relevant investigations based on provisional diagnosis**

- First line: sodium, potassium, chloride, bicarbonate, calcium, magnesium, urea, creatinine, venous blood gas
- Second line: Urine K (24 hours urinary potassium levels, Spot potassium-to-creatinine ratios, TTKG) urinary chloride, USG kidney
- Endocrine: Renin, aldosterone, 17 alpha hydroxylase, 11 beta hydroxylase
- Genetic testing

Step 5- Diagnosis

Diagnosis can often be made based on the history, clinical examination and first line investigations

a. If renal loss of K is suspected do urinary K , transtubular potassium gradient(TTKG) > 4 or urinary K >15mEq/L indicates renal loss.

b. Do a VBG.

Hypokalemia with metabolic acidosis- consider RTA, DKA

c. Do urinary chloride if metabolic alkalosis

Is it < 10-15mEq/L?

Consider gastro intestinal chloride loss (emesis) , congenital chloride losing diarrhea, cystic fibrosis

Is it > 20 mEq/L? If yes check BP

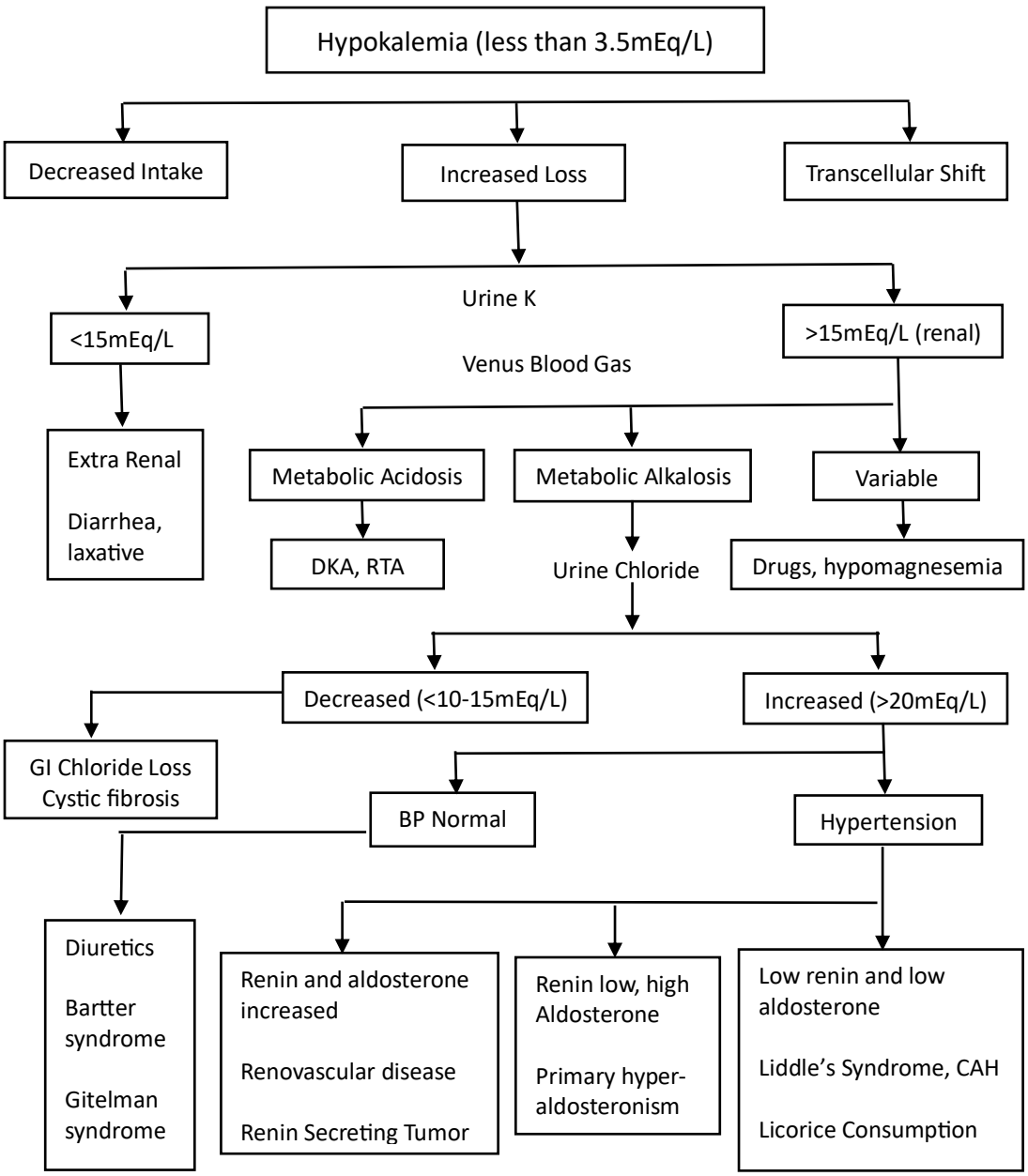
If BP -normal

Consider Bartter(calciuria), Gitelman (hypomagnesemia, hypocalciuric), EAST syndrome (normal calcium excretion)

If BP high

Consider hyperaldosteronism, renovascular disease, Cushing's syndrome

Algorithm for Hypokalemia



Case Scenarios

Case 1:

A 12-year-old girl with newly diagnosed type 1 diabetes mellitus presents to the ED 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 BP 85/50 RR 32. Her capillary glucose is >400 mg/dL and blood gas show pH 7.05 HCO_3^- 8 mEq/L anion gap 22. Serum sodium is 130 potassium is 3.1 mEq/L and urine is positive for ketones and glucose.

Questions:

1. Why is her serum potassium low?
2. What are the risks of insulin therapy on potassium balance in this patient?
3. How should potassium be managed during the treatment of DKA in this child
4. What monitoring is necessary during DKA therapy to prevent complications related to potassium
5. What are the ECG changes in hypokalemia?

Case 2:

A 10-month-old male infant is brought with a 3-day history of profuse watery diarrhea, decreased feeding and lethargy. Parents say he has passed more than 8–10 stools per day no blood and has vomited 2–3 times. On examination he is irritable lethargic when undisturbed has sunken eyes dry tongue and sluggish skin pinch. Vitals: HR 160 BP low; RR 28. Weight loss is about 12% of baseline. Capillary refill time is 4 seconds. Lab tests show Na^+ 132 Cl^- 90 bicarbonate 12 K^+ 2.7 m Eq/L and mild metabolic acidosis. ECG shows flattened T waves.

Questions:

1. How does acute diarrhea lead to hypokalemia and metabolic acidosis?
2. How should potassium replacement be incorporated into rehydration therapy for this infant

Case 3:

A 17-year male competitive wrestler is brought to the ER from a tournament after he collapsed with muscle weakness and palpitations. He had been trying to lose weight rapidly for an upcoming match. On evaluation, he is alert but dizzy and very thirsty. He

admits to taking some water pills obtained from a friend to lose weight quickly over the past week along with restricting fluids. On examination he has BP of 90/50 mmHg, pulse 120/min. Mucous membranes are dry. Cardiac examination shows irregular beats. Labs reveal K^+ 2.2 mEq/L, Cl^- 88 mEq/L, HCO_3^- 34 mEq/L, BUN 35, Cr 1.2.

Questions:

1. What is the likely cause of this patient's hypokalemia?
2. Interpret the lab findings – why is there metabolic alkalosis and what does an elevated BUN indicate?

Case 4:

A 6-year-old boy presents with poor growth, muscle weakness, and frequent urination. His parents report delayed walking and bowing of the legs. His investigations are as follows.
Arterial blood gas: pH 7.30, HCO_3^- 14, pCO_2 30, **K**: 3.0 mmol/L, **Na**: 138 mmol/L, **cl**: 112 mmol/L; creatinine: 0.3 mg/dl, Urea: 20, Calcium: 9.1, Phosphorous: 2.5, Magnesium: 2.2**;** Urine pH: 5.5; Urinalysis: Protein 1+, Glucose 2+

Questions:

1. What further investigations are needed?
2. What is the most likely diagnosis?
3. How to estimate TMP/GFR for Phosphaturia?
4. What is the treatment approach for this condition?

Case 5:

A 4-year-old boy is brought to the clinic 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. On exam, he has low blood pressure and signs of dehydration, but no edema. His investigations are as follows
Arterial blood gas**:** pH 7.48, HCO_3^- 30, pCO_2 48**,** K: 2.8 mmol/L, Na: 130 mmol/L**,** Cl: 92 mmol/L; Serum creatinine: 0.3 mg/dl, Urea: 20, Calcium: 9.1, Phosphorous: 4.5, Magnesium: 2.2
Urine pH**:** 5.5, Urinary electrolytes: Na: 87 mmol/l, Cl:136.2 mmol/l, K: 38 mmol/l, Urine ca/cr: 0.6. Urinalysis**:** Normal
USG: Bilateral medullary nephrocalcinosis

Questions:

1. What is the most likely diagnosis?
2. What is the treatment approach for this condition?

3. What is the differential diagnosis?

HYPERKALEMIA

Definition: Hyperkalemia is defined as serum or plasma concentration of K >5.5mEq/L; in neonates > 6mEq/L
Mild hyperkalemia - <6 mEq/L.
Moderate hyperkalemia -6-7 mEq/L.
Severe hyperkalemia ->7 mEq/L.

Causes of hyperkalemia

Transcellular shifts	Decreased K excretion	Increased production followed by transcellular shift*	Increased K intake
Metabolic acidosis	AKI, CRF	Tumor lysis syndrome	IV/oral
DKA	Renal tubular disease (Pseudohypoaldosteronism type I and II, type 4 renal tubular acidosis, obstructive uropathy, sickle cell disease)	Excessive trauma	Blood transfusions
Lactic acidosis	Primary adrenal disease: CAH, hypoaldosteronism and Pseudohypoaldosteronism	Rhabdomyolysis (Crush injuries, convulsion, infection)	High dose penicillin G
Drugs-succinyl choline, beta blockers	Drugs- ACE inhibitors, ARB, K sparing diuretics, trimethoprim, NSAID	Hemolysis	Parenteral nutrition
Hyperosmolality (mannitol)	Hypovolemia	Malignant hyperthermia	
Hyperkalemic periodic paralysis			

* Acute kidney injury may co-exist with these conditions and can worsen the Hyperkalemia

Symptoms of hyperkalemia

Patients with mild and moderate hyperkalemia may be asymptomatic. Symptoms range from muscle weakness to ascending flaccid paralysis. Cardiac symptoms include palpitations, syncope, arrhythmia and cardiac arrest. Respiratory depression, Ileus and paresthesia can also occur.

ECG changes

May be present in mild to moderate hyperkalemia also. A normal ECG does not rule out the risk for arrhythmia.

Tall, peaked T waves, prolonged PR interval, absent P wave, wide QRS, sine wave (fusion of QRS and T wave) VT, VF, asystole

Approach

Step -1 Find out if the patient is symptomatic. Connect to cardiac monitor and take a 12 lead ECG. Look for ECG changes. If present- **start treatment**.

- **Care of ABC**. Correct shock if present with isotonic fluids as this will improve renal excretion of K. Arrhythmias to be managed as per PALS protocol though antiarrhythmic drugs may not be very useful unless emergent measures are taken to reduce potassium levels.

- **Hyperkalemia management**

a. Stabilise the cardiac cell membrane - calcium gluconate 0.5ml/kg IV (max 20 ml) with equal amount of 5% dextrose given over 5 min with cardiac monitoring. Discontinue if HR drops significantly.

b. Promote shift of K to intracellular compartment
   - Nebulised salbutamol - 2.5mg for <25 kg in 2ml NS and 5 mg in 2ml NS between 25-50Kg
   - Insulin dextrose infusion- 0.1unit/kg of plain insulin(max 10 units) with 0.5g/kg of dextrose over 30minutes.(5ml/kg of 10% dextrose or 2ml/kg of 25% dextrose).
   - NaHCO3 - 1mEq/kg with equal amount of 5% dextrose given over 10-15 minutes in children with co-existing metabolic acidosis. (<7.2)

c. Increase potassium excretion (cation exchange resin, diuretics, dialysis)

d. Stop all K containing fluids and drugs that cause hyperkalemia. Avoid blood transfusion in a child with hyperkalemia unless absolutely necessary.

e. Replacement with a mineralocorticoid(fludrocortisone) and corticosteroid in children with salt wasting congenital adrenal hyperplasia, hypoaldosteronism. Inj hydrocortisone 2mg/kg stat can be given when there is suspected adrenal insufficiency.

Monitoring- Continuous cardiac monitoring and serial ECGs till K is normal, hourly K initially, blood glucose every 30 min if on insulin dextrose infusion, I/O chart

f. Treatment of the underlying disease

Moderate hyperkalemia without ECG changes:

Salbutamol nebulisation, insulin dextrose ±kayexalate + bicarbonate if metabolic acidosis

Mild hyperkalemia without ECG changes

Salbutamol nebulisation, iv furosemide, kayexalate

Step-2. If patient is asymptomatic and does not have setting of hyperkalemia rule out pseudohyperkalemia which is very common in infants and young children.

a. It can be secondary to heel prick, prolonged tourniquet application, fist clenching, using a small-bore needle and syringe to sample blood, all of which can cause hemolysis of the sample resulting in falsely elevated levels of K.
b. Restraining the limb in a crying agitated child can result in repetitive limb movement and muscle contraction with release of significant amount of potassium into the blood.
c. Blood sampling proximal to an intravenous line with potassium containing fluid can falsely increase the potassium level.
d. Leucocytosis and thrombocytosis also can increase serum K levels but plasma levels are normal. Check plasma K rather than serum K.
e. Repeat potassium levels from a non-hemolysed sample when pseudohyperkalemia is suspected.

Step 3: Once pseudohyperkalemia is ruled out take a detailed history and do a clinical examination to find out the etiology.

History

a. H/o palpitations, fasciculations, syncope and parasthesias
b. H/o burns, trauma leading to crush injury, drug intake by family members (accidental ingestion) or the child, diabetes, excess potassium intake
c. H/o renal disease- oliguria, anuria, edema, hypertension
d. H/o ambiguous genitalia in a female child, recurrent shock, failure to thrive in a boy may suggest primary adrenal disease like congenital adrenal hyperplasia
e. H/o blood transfusion
f. H/o drug intake
g. History suggestive of urinary tract infection
h. H/o episodes of paralysis with family history (familial hyperkalemic periodic paralysis)
i. H/o surgery, anaesthesia (malignant hyperthermia)
j. H/o chemotherapy in a child with a large tumor load
k. Color of the urine (cola colored in AGN, hemoglobinuria, rhabdomyolysis)
l. Bloody stools may occur in HUS
m. Family history of similar disorder (Single gene disorders causing hyperkalemia, malignant hyperthermia, neuromuscular disorders)

Clinical examination:

- Edema, acidotic breathing, short stature, pallor, rickets, hypertension (CKD)
- Ambiguous genitalia, hyperpigmentation (addisons disease, adrenoleukodystrophy)
- Hepatosplenomegaly, lymphadenopathy (leukemia, lymphoma)
- Muscle tenderness(rhabdomyolysis)
- Dehydration (adrenal disorders).

Step 4: Investigations based on provisional diagnosis

- **First line**: CBC, smear, urea, creatinine, blood glucose, electrolytes (Na, K, HCO3, Ca, P), urine analysis for proteinuria, casts and myoglobin, venous blood gas

- **Second line:** CPK, uric acid, USG abdomen, urine potassium and sodium

- **Endocrine workup as needed** – 17-OH progesterone, aldosterone, renin, angiotensin, cortisol, 21-hydroxylase, and 11-beta-hydroxylase.

Plasma renin activity and aldosterone concentration should be interpreted as per the table below:

Plasma renin activity	Aldosterone concentration	Interpretation
Low	Low	Intrinsic renal disease
High	Low	CAH, hypoaldosteronism
High	High	Pseudohypoaldosteronism

Clues to diagnosis based on biochemical parameters:

Diagnosis	Na	K	HCO3	Ca	P	CPK	Glucose	Urea, Cr	Uric acid
CAH	↓	↑	↓				↓		
Hypoaldosteronism	↓	↑	↓				Normal		
TLS	N	↑	N or ↑	↓	↑	N	N	N or ↑	↑
AKI/CRF	N/↑/↓	↑	↓	N or ↓	↑	N	N	↑	
Rhabdomyolysis	N	↑	↓	↓	↑	Inc	N	N or ↑	↑
Renal tubular disease	↓	↑	↓			N	N	N or ↑	

Prevention of recurrence

- Treat the underlying etiology to prevent recurrence.
- In patients with renal dysfunction reducing dietary K intake (bananas, potatoes, beans, grains), use of a loop diuretic, avoidance of drugs causing hyperkalemia and correction of metabolic acidosis with carbonate are recommended.
- A mineralocorticoid may be useful in patients with hyporeninemic hypoaldosteronism. Oral kayexalate daily in these patients help reduce hyperkalemia.

Case Scenarios:

Case 1:

7-day old female baby born at term by LSCS presented with complaints of poor sucking and excessive sleepiness. Unremarkable immediate neonatal period. This baby is second born baby of III-degree consanguineous parents. First baby expired at 11 days of age with similar symptoms at 1 week of life and had a diagnosis of suspected sepsis. Weight loss 16.5%

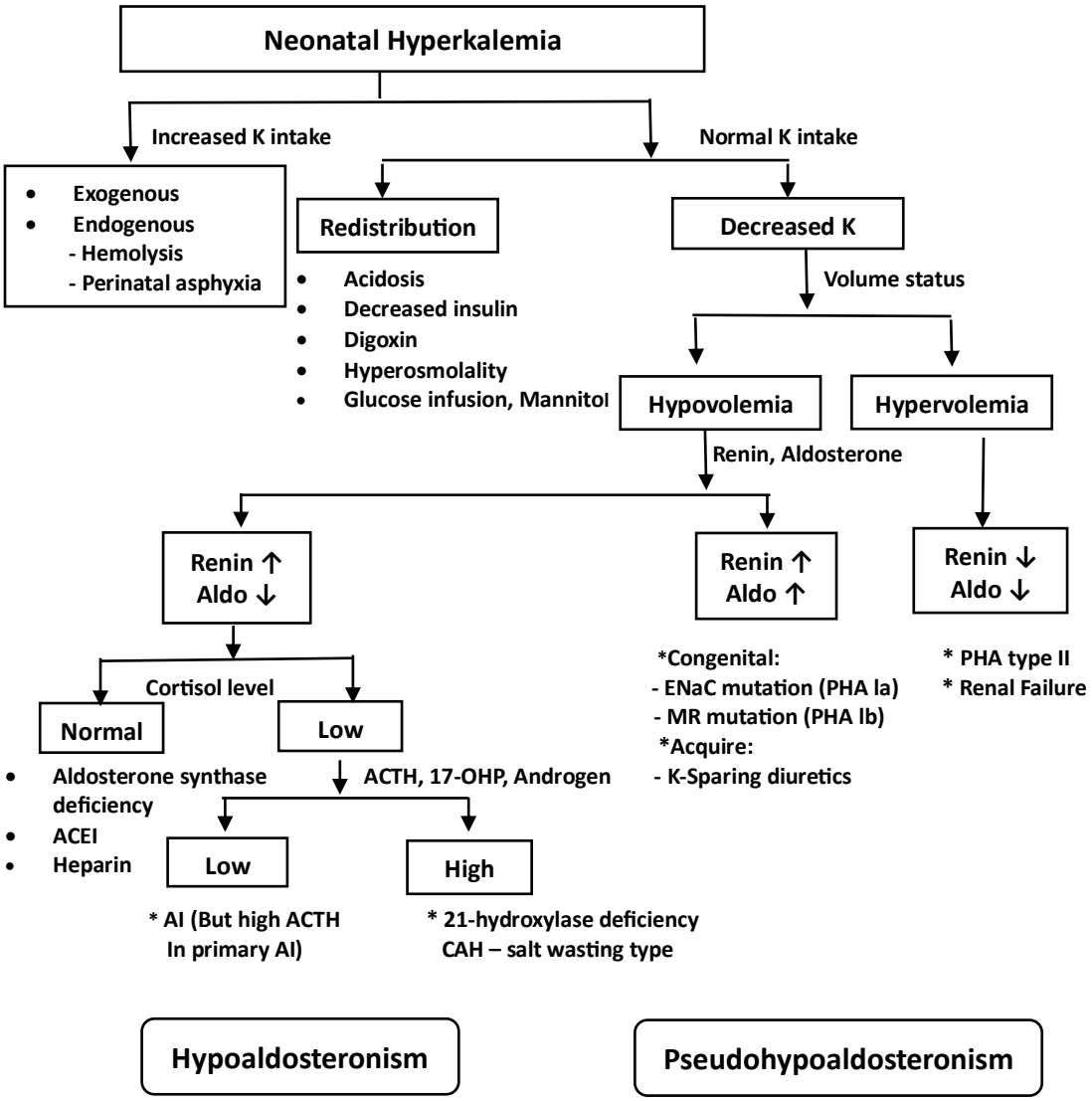
Sepsis workup and renal function tests sent. After starting feeds baby had repeated episodes of vomiting. Blood culture sent and baby was empirically started on anibiotics. CBC showed elevated total counts. CRP was negative. RFT showed elevated blood urea and normal serum creatinine. Electrolytes showed hyponatremia (124 meq/ lit) and hyperkalemia (9.6 meq/lit). Sepsis workup and renal function tests sent. After starting feeds baby had repeated episodes of vomiting. Blood culture sent and baby was empirically started on anibiotics. USG abdomen was normal. Echo done showed normal cardiac function.

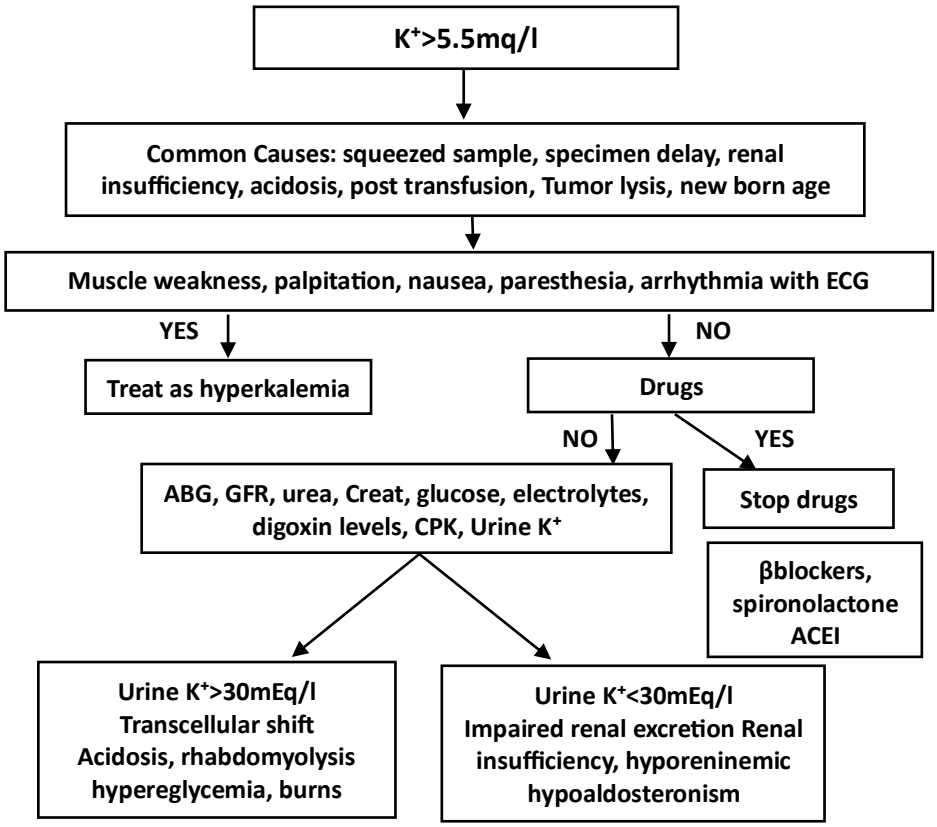
S. Creatinine	0.39
B urea	25
S. Sodium	122
S. Potassium	10.5
S. Chloride	105
S. Bicarbonate	11.7
BE	-16.6
S Lactate	6.75
CBG	85
CBC	12000 P65L35

BP – 73/30 (MAP-45)
No hyperpigmentation, no genital ambiguity, gonads not palpable

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?


Neonatal Hyperkalemia
Increased K intake
Normal K intake
Exogenous
Endogenous
- Hemolysis
- Perinatal asphyxia
Redistribution
Acidosis
Decreased insulin
Digoxin
Hyperosmolality
Glucose infusion, Mannitol
Decreased K
Volume status
Hypovolemia
Hypervolemia
Renin, Aldosterone
Renin ↑ Aldo ↓
Renin ↑ Aldo ↑
Renin ↓ Aldo ↓
*Congenital:
- ENaC mutation (PHA Ia)
- MR mutation (PHA Ib)
*Acquire:
- K-Sparing diuretics
* PHA type II
* Renal Failure
Cortisol level
Normal
Low
Aldosterone synthase deficiency
ACEI
Heparin
ACTH, 17-OHP, Androgen
Low
High
* AI (But high ACTH In primary AI)
* 21-hydroxylase deficiency CAH – salt wasting type
Hypoaldosteronism
Pseudohypoaldosteronism


K+>5.5mq/l
Common Causes: squeezed sample, specimen delay, renal insufficiency, acidosis, post transfusion, Tumor lysis, new born age
Muscle weakness, palpitation, nausea, paresthesia, arrhythmia with ECG
YES
NO
Treat as hyperkalemia
Drugs
NO
YES
ABG, GFR, urea, Creat, glucose, electrolytes, digoxin levels, CPK, Urine K+
Stop drugs
βblockers, spironolactone ACEI
Urine K+>30mEq/l Transcellular shift Acidosis, rhabdomyolysis hypereglycemia, burns
Urine K+<30mEq/l Impaired renal excretion Renal insufficiency, hyporeninemic hypoaldosteronism