ELECTROLYTE

-WORKBOOK-

Organized by

Department of Pediatrics and Pediatric Nephrology,

Apollo Children's Hospitals, Chennai

Organizing

PATRONS

Dr. Prathap C Reddy, Chairman, Apollo Hospitals Group
Dr. Preetha Reddy, Vice Chairperson, Apollo Hospitals Group
Dr. Suneeta Reddy, Managing Director, Apollo Hospitals Group

CLINICAL ADVISOR

Dr. R K Venkatasalam, Director of Medical Services, Apollo Hospitals, Chennai

Dr. B R Nammalwar
Organising Chairperson

Dr. Sudha Ekambaram
Organising Secretary

Dr. K U Suresh Balan
President, IAP-TNSC

Dr. V Tiroumourougane Serane
Secretary, IAP-TNSC

Dr. R V Dhakshayani
Treasurer, IAP-TNSC

Dr. J Shyamala
President, IAP-CCB

Dr. Sridevi A Naaraayan
Secretary, IAP-CCB

Dr. R Venkateshwari
Treasurer, IAP-CCB

NephKids 2023

www.nephkids.in

PROGRAMME

DAY 1

Saturday, September 9th, 2023
FLUID AND ELECTROLYTE WORKSHOP

Conveners: Dr. S Thangavelu, Dr. R C Sharada

TOPIC	MODERATOR	SPEAKERS	09:15-10:15	10:20-11:20	11:30-12:30
Hyponatremia	Dr. S Thangavelu	Dr. J Shyamala Dr. P S Rajakumar Dr. S Vasanth Kumar	Group A	Group B	Group C
Fluid in special situations	Dr. P Narayanan	Dr. R C Sharada Dr. R V Dhakshayani Dr. V Priyavarthini	Group A	Group B	Group C
Metabolic Acidosis	Dr. Indira Jayakumar	Dr. V P Anitha Dr. Muthiah Periyakaruppan Dr. Anita Tarigopula	Group A	Group B	Group C
12:30 – 13:30	LUNCH				
Hypernatremia	Dr. V Poovazhagi	Dr. Shobana Rajendran Dr. V Vimalraj Dr. L Chidhambharam	Group A	Group B	Group C
Hypo / Hypercalcemia Hypomagnesemia	Dr. NC Gowrishankar	Dr. L K Prem Kumar Dr. T S Ekambaranath Dr. R Venkateshwari	Group A	Group B	Group C
Hypo / Hyperkalemia	Dr. S Shanthi	Dr. Sridevi A Naaraayan Dr. M Karthikeyan Dr. Manasi Garg	Group A	Group B	Group C

Dr. B.R. Nammalwar

Organising Chairperson, NEPHKIDS

INTRODUCTION

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 fluids, 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 organising "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.

Faculty - Workshop

Dr. S Thangavelu, MBBS DCH MD DNB MRCP

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

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

Senior Consultant Neonalogist & Paediatician, Apollo First Med Hospitals and Apollo Children's 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. S Vasanth Kumar, MD (Ped) DNB (Ped) FNB PICU FECMO

Consultant PICU, Apollo Children's Hospital, Chennai

Dr. P Narayanan, MD, DNB

Professor of Paediatrics, JIPMER, Puducherry

Dr. RC Sharada, MBBS DCH DNB FPEM FSTEP

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

Dr. RV Dhakshayani, MD (Paediatrics)

Associate Professor of Paediatrics, Government Vellore Medical College

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

Consultant PICU, Apollo Children's Hospital, Chennai

Dr. Indira Jayakumar,

Sr. Consultant and Lead Pediatric Intensivist, Apollo Speciality Hospital, Chennai

Dr. V P Anitha, MBBS, DCH, Fellowship Paediatric Intensive Care, MRCPH

Sr. Consultant Ped and PICU, Apollo Speciality Hospital Vanagaram, Chennai

Dr. Muthiah Periyakaruppan, MD IDPCCM, FPCC (Ped Critical Care)

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

Dr. Anita Tarigopula, DNB PAEDIATRICS, FPEM, STEP Fellowship (Pead Emergency and Trauma), PGDMLE (NLSIU,Bengaluru)

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

Dr. V Poovazhagi, MD DCH PhD

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

Dr. Shobana Rajendran, DNB Pediatrics, DNB Neonatology
Senior Consultant Neonatologist, Rainbow Childrens hospital Guindy, Chennai

Dr. V Vimalraj, MBBS, DNB, IDPCCM
Assistant Professor, SRM Medical College and hospital, Kattankolathur, Chennai

Dr. L Chidhambharam, MBBS MD FNB Pediatric ICU
PICU Consultant, Apollo Children's Hospital, Chennai

Dr. NC Gowrishankar, MD DCH DNB FIAP
Head – Pediatrics, Dr.Mehta's Multispeciality Hospital India Pvt Ltd, Chennai

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

Dr. TS Ekambaranath, MD (Ped), PICU
Assistant Professor, PICU, Stanley Medical College, Chennai

Dr. R Venkateshwari, MBBS DCH DNB
Senior Consultant Pediatrician, Kanchi Kamakoti CHILDS Trust Hospital, Chennai

Dr. S Shanthi, MD, DCH
Former Professor of Pediatrics, Institute of Child Health and Hospital for Children, Chennai

Dr. Sridevi A Naaraayan, MD (Paediatrics), PGDEpi
Professor of Pediatrics, Institute of Child Health and Hospital for Children, Chennai

Dr. M Karthikeyan, M.D pediatrics
Senior assistant professor, Kilpauk medical college

Dr. Manasi Garg, MD Pediatrics, FPN
Professor, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth, Pondicherry

HYPONATREMIA

Contributors: Dr.Thangavelu S, Dr.Rajakumar PS, Dr.Shyamala J, Dr.Vasanth Kumar S

Physiology of Sodium balance:

- Sodium is the dominant cation of ECF. Maintains ECF osmolarity and thereby cell volume of billion cells in the body, including that of brain.
- ***Sodium intake:*** Infants receive sodium from breast milk (7 mEq/L) and formula (7-13 mEq/L). An average Indian consumes 10.98 grams of salt per day -- 119 % > recommended limit of 5 grams/day by WHO (1 g NaCl = 394 mg, 17 mEq or 17 mmol of Na and Cl).
- ***Excretion***: Occurs through urine, stools and skin.
- ***Control:*** Body sodium content is most intimately coupled with extracellular water content. Water and Na are like olden days couple. Water balance, not sodium balance, usually determines its concentration. When the sodium concentration increases, the resultant higher plasma osmolality causes increased thirst and increased secretion of ADH, which leads to renal conservation of water. During hyponatremia, the decrease in plasma osmolality stops ADH secretion, and consequent renal water excretion leads to an increase in the sodium concentration. Renal sodium regulation plays a major role in sodium homeostasis.
- ***What is the priority in a conflicting situation***? Correction of volume depletion takes priority over osmolarity. Volume depletion stimulates ADH secretion even when there is hyponatremia. E.g. Hyponatremic dehydration in acute diarrhea. Once dehydration is corrected with NS, ADH is switched off, water retention ceases and serum sodium levels raises.

Algorithmic approach to hyponatremia

1. Does the child have neurological symptoms?
2. True or pseudo hyponatremia?

3. Is it acute or chronic?
4. Volume status?
5. Urine sodium levels

Explanatory notes for algorithm

Step 1 – Does the child have neurological symptoms?

The clinical presentation of hyponatremia is usually variable, nonspecific and when severe presents with symptoms of cerebral edema and those of underlying disease as noted below:

<130mEq - Apathy, anorexia, nausea, vomiting

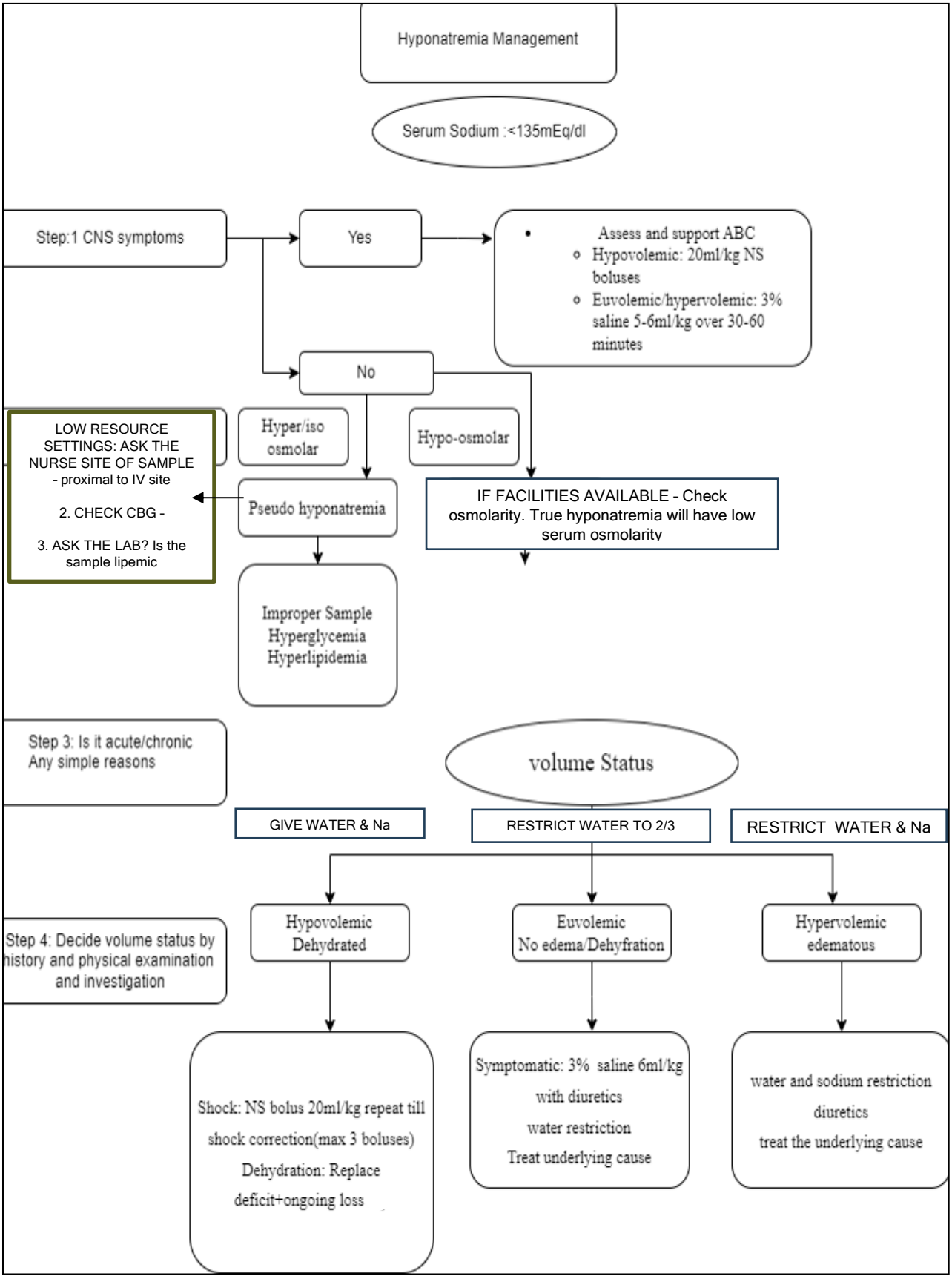
S. Sodium <120 mEq/dl - Muscular twitching, headache, seizures, coma.

Symptoms depend upon the degree and chronicity of hyponatremia. Patients with mild-to-moderate hyponatremia (greater than 120 mEq/L) or gradual decrease in sodium (greater than 48 hours) have minimal symptoms. Patients with severe hyponatremia (less than 120 mEq/L) or rapid decrease in sodium levels have multiple varied symptoms. Symptoms can range from anorexia, nausea and vomiting, fatigue, headache, and muscle cramps to altered mental status, agitation, seizures, and even coma.

Patients with neurological symptoms and signs need to be treated promptly to prevent permanent neurological damage.[18]

Symptomatic hyponatremia

When the child has neurological symptoms, regardless of underlying cause or volume status, or duration of the illness the deficit must be corrected to a safer level within 1-2 hours.


Hyponatremia Management
Serum Sodium :<135mEq/dl
Step:1 CNS symptoms
Yes
Assess and support ABC
Hypovolemic: 20ml/kg NS boluses
Euvolemic/hypervolemic: 3% saline 5-6ml/kg over 30-60 minutes
No
Hyper/iso osmolar
Hypo-osmolar
LOW RESOURCE SETTINGS: ASK THE NURSE SITE OF SAMPLE - proximal to IV site
2. CHECK CBG -
3. ASK THE LAB? Is the sample lipemic
Pseudo hyponatremia
IF FACILITIES AVAILABLE - Check osmolarity. True hyponatremia will have low serum osmolarity
Improper Sample
Hyperglycemia
Hyperlipidemia
Step 3: Is it acute/chronic
Any simple reasons
volume Status
GIVE WATER & Na
RESTRICT WATER TO 2/3
RESTRICT WATER & Na
Step 4: Decide volume status by history and physical examination and investigation
Hypovolemic
Dehydrated
Euvolemic
No edema/Dehyfration
Hypervolemic
edematous
Shock: NS bolus 20ml/kg repeat till shock correction(max 3 boluses)
Dehydration: Replace deficit+ongoing loss
Symptomatic: 3% saline 6ml/kg
with diuretics
water restriction
Treat underlying cause
water and sodium restriction
diuretics
treat the underlying cause

Safer levels mean 5 mEq above the current measured level. Eg. If measured Serum Sodium is 115mEq/dL, increasing it to 120mEq/dL is advised and definitely not raised to normal range immediately.

Clinical situation 1: In the presence of hypovolemic hyponatremia (acute diarrheal dehydration) and seizures -NS boluses in 20ml/kg aliquots is given, aiming at simultaneous volume and sodium correction. In the presence of dehydration/shock, both volume and sodium replacement is required. Once hypovolemia is corrected, ADH secretion is switched off and hyponatremia gets corrected.

Clinical situation 2: In normovolemic and hypervolemic hyponatremia with seizures, correction is done by using 3% saline 5mL/kg over 30 – 60 minutes. An increase of 5mEq/L over 2 hours is enough to tide over the crisis

Eg: 1 year old child, weighing 10 kg, serum Sodium is 110mEq/dL. Child is having seizures.

In addition to management of seizures, we should raise the serum Sodium by 5 mEq/L. 60 ml of 3% saline is to be infused over 30-60 min. Close clinical monitoring and frequent (every 2-4 hrs) electrolyte estimation is mandatory. After the initial therapy, replacement is continued as that for an asymptomatic child.

Step 2 -If asymptomatic see whether it is pseudo hyponatremia? Confirm whether hyponatremia is true or false. Usually hyponatremia is associated with hypoosmolality. If facilities are available, check the serum osmolarity. If it is low it is true, if it remains normal or high it is pseudohyponatremia. If serum osmolarity is unavailable, simple history and examination can identify pseudohyponatremia.

Methods to identify pseudohyponatremia

- Improper sample – ask the nurse or resident whether sample was taken proximal to the IV cannula site. Blood obtained from a vein proximal to an infusion of hypotonic saline (1/2 GNS) will have a low sodium.

- Ask the lab persons whether the sample is lipemic. Presence of hyperlipidaemia can be identified as a cause of pseudohyponatremia. Pseudohyponatremia is a laboratory artifact seen in hyperlipidaemia and hyperproteinaemia where the serum osmolarity is normal. Hyperlipidaemia is suspected when serum is lipemic. Hyperproteinaemia is very rare in children.

- Check blood glucose by glucometer, which will identify hyperglycemia as a cause. Each 100mg raise of blood sugar will decrease serum sodium by 1.6 mEq/L. Hyponatremia associated with hyperglycemia generally resolves as hyperglycemia is corrected. In hyperglycemia the serum osmolarity is high.

- Ask for the history of mannitol therapy, which also may be a cause for pseudohyponatremia.

Step 3: Further evaluation when the child is asymptomatic and it is true hyponatremia

If it is true hyponatremia, volume status needs to be evaluated based on history and physical examination

History: The following should be looked for: Diarrhea, vomiting, polyuria, oliguria, edema, breathlessness, altered level of consciousness or convulsions, any surgical procedure done, drugs and IV fluids administered.

Physical Examination: One has to look for signs of dehydration, features of shock, S3 gallop, respiratory distress, ascites, edema, pigmentation, stigmata of liver or renal disease and bony deformity suggestive of rickets. Genital examination is mandatory to look for signs of congenital adrenal hyperplasia.

Investigations needed: Serum electrolytes, glucose, urea, creatinine, chloride, x-ray chest, serum osmolarity, Urine osmolarity and urine sodium are the most useful investigations

Urine Na <20 mEq / L indicates extra renal loss

Urine Na > 20 mEq / L indicates renal loss

HYPOVOLEMIC HYPONATREMIA

In hypovolemic hyponatremia, one should elicit history to identify cause of fluid loss.

If child has vomiting/diarrhea/significant nasogastric tube aspirate - GI loss

If child has polyuria or voiding urine despite dehydration-, consider renal loss.

If the history does not point toward GI or renal loss – consider cutaneous loss, ask for excessive sweating.

a) GI LOSS:

If there is dehydration, correction needs replacement of water and sodium. So in a child with symptomatic hyponatremia and dehydration, correct with Normal saline bolus 20 mL/kg every 20-60 mins depending on the hydration status (20ml/kg aliquots will provide 5 mEq/kg).

- How? Eg – 10 kg child will receive 200 ml (20 mL/kg). Each 100 ml contains 15 meq and 200 mL will provide 30 mEq.
- How much sodium is needed to raise the serum level by 1 mEq?

  10 x 0.6 x 1 = 6 mEq. Hence when we provide 200ml of NS, it will raise the serum sodium level by 5 mEq/L. For example, serum level increases from 115mEq/L to 120 mEq/L.
- In short, correct the dehydration with NS, hyponatremia will get corrected automatically

b) CUTANEOUS LOSS:

Because of excessive loss of sodium and chloride through the skin, from sweating, hyponatremia can occur and it can be worsened in those who consume plain water without electrolytes. This is more commonly seen in hot climates. In cutaneous loss as in cystic fibrosis and in marathon running, degree of dehydration will be mild and child will be mostly asymptomatic. In cystic fibrosis, metabolic alkalosis and hypokalemia are associated findings. Hence this can be corrected by replacing sodium by oral route either as salt containing oral fluid, dietary preparations such as butter milk, coconut water, vegetable soup, rasam (spicy soup containing salt, used with rice) or electrolyte

solution such as ORS. Otherwise, it can be corrected by replacing maintenance fluid as isotonic fluid like normal saline in addition to oral supplements, periodically monitoring serum sodium levels. Drinking plain water should be replaced by electrolyte solution to prevent hyponatremia. Rapid correction may not be necessary in this situation

c) URINARY LOSS:

Rapidity of correction depends on the severity of dehydration. If the child is hospitalised, child can be managed with normal saline depending on the severity. For example: mild dehydration 30-50 ml/kg, moderate dehydration 50-70ml/kg This deficit is to be combined with administration of maintenance fluid simultaneously and given over 24-48 hrs.

EUVOLEMIC/HYPERVOLEMIC DEHYDRATION

In the presence of symptomatic hyponatremia, hypertonic saline (3% sodium chloride) 5- 6 mL/kg is preferred.

Example: A 10 kg child will require 60 mL 3%saline infusion over 30 - 60 mins (depending on the urgency). This will not cause volume overload.

- How much sodium this will provide? 60 mL of 3% saline will provide 30 mEq (1 mL of 3% saline gives 0.5 mEq of sodium)

  How much sodium is needed to raise the serum level by 1 mEq? Eg. Baby weight 10 kg. 10 x 0.6 x1 = 6 mEq. Hence when we provide 30 mEq, it will raise the serum level by 5 mEq.

Hypovolemic Hyponatremia	Euvolemic Hyponatremia	Hypervolemic Hyponatremia
Dehydration present Causes 1.Extra renal loss – GI -Vomiting, diarrhea - significant nasogastric aspirate 2. Renal Loss -Renal Tubular Acidosis (RTA), osmotic diuresis (Diabetic ketoacidosis), diuretic therapy, adrenal insufficiency If history does not point toward GI or renal loss – consider cutaneous loss ask for excessive sweating/ manifesting in summer. 3. Cerebral salt wasting syndrome **Investigation** Urine Na >20mEq/L- Renal cause Urine Na <20 mEq / L – Non Renal ↓Na ↓K ↑cl – RTA ↓Na ↑K ↓ glucose - Adrenal insufficiency	**No Dehydration no edema** Causes 1. Water intoxication (Use of 5% Dextrose in post operative Period). 2.Psychogenic water drinking 3.SIADH **Investigation** Urine Na > 20 mEq/L – SIADH Cerebral salt wasting Urine Na< 20 mEq/L – water intoxication Psychogenic water drinking	**Edema Present** Causes 1. Renal failure 2. Nephrotic syndrome 3. Congestive heart failure 4. Protein energy malnutrition 5. Cirrhosis liver **Investigation** Urine Na > 20 mEq/L – Renal failure Urine Na < 20 mEq – all others

Most of the time hyponatremia is managed with clinical decisions supported by basic investigations.

Case Scenario 1

A 1-year-old developmentally normal, previously healthy baby boy is admitted with Lobar Pneumonia with fever, cough and tachypnea. CxR confirms Pneumonia. Baby is started on IV Ceftriaxone and IV maintenance fluids. On day 2 of admission, baby has decreased urine output (0.4 ml/kg/hr) for last 24 hours.

Clinically perfusion is normal with no signs of dehydration and no h/o of vomiting/ diarrhoea. There is no edema/ hypertension. Urinary bladder is not distended. Rest of the examination is unremarkable apart from respiratory signs of Pneumonia. He has normal sensorium with no neurological deficit.

Lab Results:

Serum studies

Sodium 126 mEq/L	BUN 4 mg/dL
Chloride 98 mEq/L	Creatinine 0.4 mg/dL
Potassium 3.7 mEq/L	Glucose 129 mg/dL
Bicarbonate 25 mEq/L	

Urine studies

Specific gravity 1.035

Ultrasound Abdomen (KUB)

Normal

1. What is the likely cause of oliguria and hyponatremia?
2. What test will you do to confirm the diagnosis?
3. How will you manage the child further?

In spite of starting the correct management, baby develops generalised tonic clonic seizures refractory to IV Lorazepam and IV Phenytoin. ABG done shows Sodium of 116.

1. What will be your immediate management?
2. How will you manage the baby further after seizure stops?
3. What will you monitor (clinical and lab)?

What are the clinical features of the dreaded complication that can happen if you correct very fast?

Case scenario 2

3 year old female, weighing 10kg was brought to with complaints of fever for 3 weeks associated with bifrontal headache and GTCS refractory to lorazepam and levetiracetam and fosphenytoin, hence she was intubated and mechanically ventilated. CSF study and MRI was suggestive of Tubercular

Meningitis. Antituberculosis treatment (ATT) along with intravenous steroids were started. Ventriculoperitoneal (VP) shunt was placed in view of obstructive hydrocephalus. Her admission serum sodium -132. On day 2 serum sodium -121meq/l). vitals HR-100/min, Spo2-99%, ABP-100/76 mmHg, UO-2.8ml/kg/hr.

Q: How will you approach this low sodium? What is your immediate management?

Case scenario progresses:

Fluid restriction was done to 2/3rd maintenance and 3% saline was continued at 1ml/kg/hr to maintain normal serum sodium levels as a part of neuroprotective care. EEG monitoring revealed no seizures. Serum sodium was monitored 6hrly and Na transiently improved to 125 meq/l, but subsequently by day 4, the serum sodium gradually reduced to 123, 121 and 119 meq/l?

Q: What is the reason for hyponatremia despite 3% saline? What additional test will you do to confirm your diagnosis?

Q: Based on reports, How will you calculate fluid prescription for this child weighing 10kg to correct hyponatremia?

Q: What will you need to monitor?

Case scenario progression: She continued to have persistent hyponatremia with polyuria despite sodium replacement

Q: How will you manage refractory hyponatremia in this specific scenario?

Case progression:

After 10 days of hospitalisation, polyuria improved though serum sodium levels showed fluctuations requiring further fluid and medication adjustment. The child finally improved after 24days of treatment and got discharged.

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 with strangers. 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 air entry on the right side. There is free fluid in the abdomen. Other systems are normal.

Investigations done on admission:

S.Creatinine	0.4
B urea	23
S. Sodium	128
S. Potassium	4.1
S. Chloride	102
S. Bicarbonate	22
S albumin	2.1
S. cholesterol	343
CBG	85
CBC	Normal

Q1. What is the metabolic abnormality in this child?

Q2. What further investigations will you order?

Q3. What are the steps in the management?

FLUIDS IN SPECIAL SITUATIONS

Contributors: Dr. Narayanan P, Dr. R.C.Sharada, Dr. R.V.Dhakshayani, Dr. V.Priyavarthini

Essential facts

- Fluid therapy is a dynamic process on constant flux
- Monitoring at the bedside and constant adjustments are necessary especiallyin a sick child
- Resuscitation is based on intravascular fluid status
- Restoration to baseline is based on ECF status and finally total body water

Step 1 – Assessment of fluid status

- Change in weight if available is the best
- Clinical assessment
- Input / output charts
- Measurement of ongoing losses (Urine, GI, drains)

Step 2 – Calculation of deficit – Determines Volume of replacement

- Expressed as percentage of body weight
- Based on all parameters in step 1
- Can add anticipated losses if deemed necessary

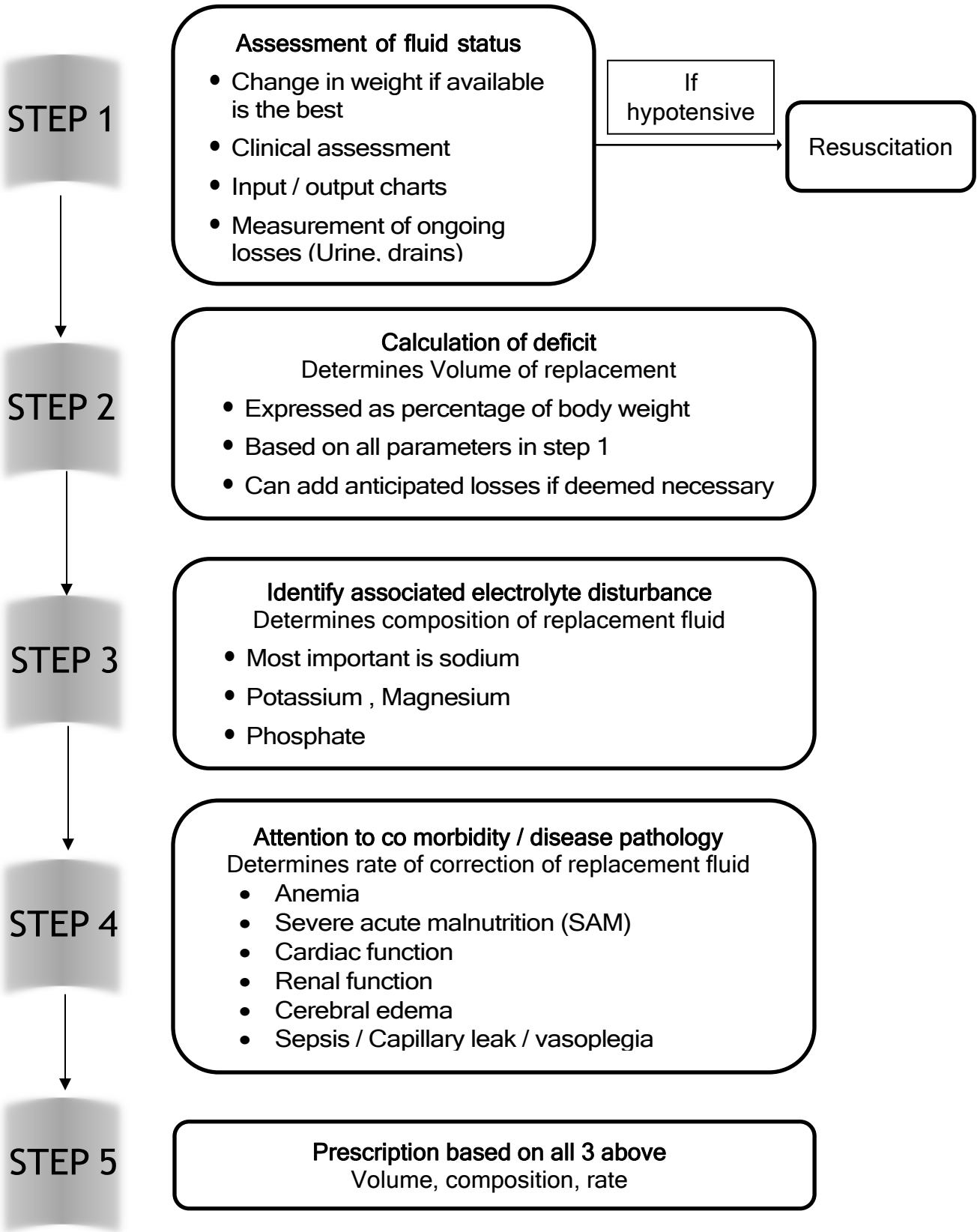
Step 3 – Identify associated electrolyte disturbance – Determinescomposition of replacement fluid

- Most important is sodium
- Potassium , Magnesium
- Phosphate

Step 4 – Attention to co morbidity / disease pathology – Determinesrate of correction of replacement fluid

- Anemia
- Severe acute malnutrition (SAM)
- Cardiac function
- Renal function
- Cerebral edema
- Sepsis / Capillary leak / vasoplegia

Step 5 – Prescription based on all 3 above - volume, composition, rate


STEP 1
Assessment of fluid status
• Change in weight if available is the best
• Clinical assessment
• Input / output charts
• Measurement of ongoing losses (Urine. drains)
If hypotensive
Resuscitation
STEP 2
Calculation of deficit
Determines Volume of replacement
• Expressed as percentage of body weight
• Based on all parameters in step 1
• Can add anticipated losses if deemed necessary
STEP 3
Identify associated electrolyte disturbance
Determines composition of replacement fluid
• Most important is sodium
• Potassium , Magnesium
• Phosphate
STEP 4
Attention to co morbidity / disease pathology
Determines rate of correction of replacement fluid
• Anemia
• Severe acute malnutrition (SAM)
• Cardiac function
• Renal function
• Cerebral edema
• Sepsis / Capillary leak / vasoplegia
STEP 5
Prescription based on all 3 above
Volume, composition, rate

Case scenario 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) 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, 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.

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

Case scenario 2

A 10 month old infant presents to the emergency department with a 4-day history of frequency watery stools. He is now refusing to drink. He is listless in his mother's arms. On physical examination, his mucous membranes are dry and the skin on his abdomen is tenting. His heart rate is 160 beats/min and blood pressure is 80/40 mm Hg. His current weights is 9 kg. One week ago, when he was seen in clinic for a routine examination, he weighed 10 kg. His serum sodium measures 138 mEq/dL (138 mmol/L). After failing a trial of oral therapy, intravenous access is obtained and he is given 20 m/L/kg (200 m) of normal saline. Following the infusion, his heart rate, perfusion and mental status improve.

How will you prescribe fluids for this child?"

Case scenario 3

18 months old female child presented to the OPD with H/O loose stools of 2 days duration. Child had persistent vomiting since morning. Child had been admitted for a diarrhoeal episode, the previous month. Child was looking undernourished with a protuberant abdomen, lustreless hair and visible wasting of the gluteal muscles. The child weighed 7.5 kgs and measured 82 cms in length. Her MUAC

was 11 cms. The child appeared lethargic and hadn't consumed any feed since morning. Rapid cardiopulmonary cerebral assessment was done at ED and the baby was in a verbal responsive mental status with peripheries cool below ankles, prolonged CRT > 3 seconds and +++/+ pulses.

How will you manage this child at the ED, at this point of first contact?

After an hour of fluid resuscitation, the child's peripheries became warm, pulses became well felt and the child was able to drink ORS and fluids when offered.

How will you manage this child further?

METABOLIC ACIDOSIS

Contributors: Dr Indira Jayakumar, Dr. V.P.Anitha, Dr. Muthiah Pariyakaruppan, Dr. Anita Tarigopula

A fall in pH is termed acidemia, and the underlying disorders that lead to acidemia is acidosis. A primary metabolic acidosis is a pathophysiologic state characterized by an arterial pH of less than 7.35 (acidemia) in the absence of an elevated $PaCO_2$.

Traditional Henderson-Hasselbalch theory - $[H^+] = 24 \times PaCO_2/[HCO_3^-]$. a change in either HCO_3^- or $PaCO_2$ changes the other variable in the same direction (compensation) within certain limits.

Limitations of this approach:

- Though it does show the change that occurs in $Paco_2$ and Hco_3 it does not necessarily state that they are the cause of the underlying acid base abnormality.
- The role of plasma proteins, specifically albumin, in acid-base balance is neglected.

The **Modern Stewart Approach** – The modern physical- chemical approach introduced in 1980 by Peter Stewart states that there are only 3 independent variables controlling H+ concentration and that changes in Hco_3 and $Paco_2$ are the consequence of these in an attempt at maintain pH in the normal range .

- PaCo2
- SID- Strong Ion Difference (strong cations – strong anions)

  (Na+K+Ca+ Mg) – (Cl+lactate) Normal – 40 mmol/L

  Narrowing of SID causes Acidosis and Widening of SID causes Alkalosis
- Atot – weak acids (albumin, phosphate)

  Albumin and phosphate act as weak acids the latter contributing to acidosis in renal

failure. Hypoproteinemia causes a base excess.

Eg: Metabolic Acidosis occurring with large volume saline administration is because of excess chloride administration and narrowing of SID. When large volumes of saline are administered it has a proportionally greater effect on total body chloride than on sodium.

Metabolic Alkalosis with Vomiting occurs due to loss of Chloride. Replacement with Saline or Ringers Lactate corrects its

Classification of Lactic Acidosis (Cohen & Woods):

TYPE A: It occurs in hypoperfusion and hypoxia.

- Tissue hypoxia is seen in carbon monoxide poisoning, severe asthma and severe anemia.
- Hypoperfusion occurs in state of shock- cardiogenic, hemorrhagic, septic, regional (mesentric , limb) ischemia) , cardiac arrest

TYPE B: It occurs when there is NO clinical evidence of hypoperfusion.

It is further subdivided into 3 subtypes:-

- **B1 -** Acquired diseases - diabetes mellitus, seizures, ARDS , sepsis, malignancies, pheochromocytoma, post cardiopulmonary bypass, renal failure, thiamine deficiency, thyroid storm (all causing increased production)hepatic failure, (decreased clearance) etc.
- **B2** - Medications and Toxins - acetaminophen, epinephrine, isoniazid, nitroprusside etc.
- **B3 -** is due to Inborn errors of metabolism

Laboratory assays estimate only L- lactic acidosis. D-lactic acidosis is rare and is caused by d-stereo isomer of lactic acid which is synthesized by pathological gut flora

In Metabolic Acidosis, check for Anion Gap, Delta Gap, Osmolar Gap

Anion Gap (correct for low albumin)

NAGMA - Normal Anion gap acidosis (Cl high)

(diarrhoea, renal tubular acidosis, saline, acetazolamide)

HAGMA -Wide Anion Gap acidosis > 12 (Cl -N)

GOLDMARK mnemonic G- glycols,O- oxoproline, L- lactic acidosis, D- D lactate , M- methanol, A- aspirin, R-renal failure, K-Ketoacids, Oxoproline - Acetaminophen use

Low Albumin - a correction factor of 2.5 must be multiplied to every 1mg/dl reduction of albumin below 4 mg/dl and this added to Anion Gap (AG) to get the **True anion gap.**

eg- Albumin – 1.6 mg/dl and Anion Gap - 12

Corrected (True) AG = (4 - 1.6) × 2.5 + AG = 6 +12 = 19

Another formula- Na – (Chloride + Bicarbonate) + 2.5 (4 – Sr.albumin)

When the anion gap is not corrected in hypoalbuminemic pts, abnormally elevated anion gaps could be missed.

DELTA RATIO & DELTA GAP

Universal rule- The increment in anion gap (AG-12) = decrease in bicarbonate (24-$NaHco_3$)

DELTA RATIO. (Delta AG / Delta HCo_3)

(AG - 12 / 24 - $NaHCo_3$)

Metabolic Alkalosis

Change in **AG > drop in HCO3** (24 - $NaHco_3$)

(For the given anion gap increase , bicarbonate did not fall as much)

N anion gap metabolic acidosis

Change in AG **<** drop in **HCO3** (For the given anion gap increase , bicarbonate fell much more)

Values - < 0.4 - NAGMA

1.4-0.8 - NAGMA + HAGMA

1- 2 - HAGMA

>2 - HAGMA + Metabolic Alkalosis

DELTA GAP = (AG - 12) + HCo_3

If Delta Gap < 18 = Non Anion Gap Metabolic Acidosis

If Delta Gap > 30 = Metabolic Alkalosis

OSMOLAR GAP

Osmolar Gap = Measured Osmolarity - Calculated Osmolarity. (Normal = < 10 meq/L)

Calculated Osmolarity = 2xNa + Glucose/ 18 + Urea/ 6

Treatment of Metabolic Acidosis – Correction of acidosis with bicarbonate may be warranted in patients of myocardial dysfunction as acidosis can cause catecholamine refractoriness. Adverse effects of bicarbonate can be reduced by giving slow infusions in preference to rapid boluses, by correcting hypocalcemia and ensuring adequate ventilation

Bicarbonate side effects

- Hypernatremia, Hyperosmolality (osmolality is 2,000 mOsm/L equal to 5.8% NaCl.
- Impaired oxygen unloading due to left shift of the oxyhaemoglobin dissociation curve
- Hypercapnia with paradoxical intracellular & CSF acidosis -Ventilation must be adequate to eliminate CO2 produced from. HCo_3
- Ionized hypocalcemia & Low K due to alkalosis causing shift into cells decreasing myocardial contractility.

Always treat the underlying cause

- Shock - Restore perfusion with fluids and adequate tissue oxygenation / ventilation, vasoactives, early antibiotic treatment, source control (surgical debridement , central line removal , ischemic gut)

- Status asthmaticus - taper high dose of beta $_2$ agonist to reduce lactate levels

- Changing from Normal Saline to Balanced fluids (Ringer Lactate, PlasmalyteA) to reduce hyperchloremic normal anion gap acidosis. Normal Saline- Na- 154 meq/l, Cl- 154meq/l Ringer Lactate – Na-130 meq/l, Cl- 110 meq/l

- Antidotes for toxins, drugs (Paracetamol even in normal doses can be toxic in hepatic dysfunction).
- Institute dialysis early in Renal failure for persistent acidosis

Analysis with ABG

Measured values - pH, paCo2 , paO2, tCO2 : **Calculated values** – Hco3, BE, SBE

1. History & Physical. gives an idea of what acid base disorder might be present
2. Look at the pH
   - If pH < 7.35, then academia
   - if pH > 7.45, then alkalemia
   - 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?
   - 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 respiratory process, is it acute or chronic?
   - To assess if acute or chronic, determine the extent of compensation.

     10 mmHg change in PaCo2 – Bicarbonate changes by 1 (Acute)

     10 mmHg change in PaCo2 – Bicarbonate changes by 4 (Chronic)
5. If metabolic process, is degree of compensation adequate?
   - Calculate the estimated PCO2, this will help to determine if a separate respiratory disorder is present. In a primary metabolic acidosis, the degree of acute respiratory compensation (pCo2 rise) can be predicted by the following relationship:

     Expected PaCO2 = (1.5 X [HCO3-]) + 8 ± 2 Winters Formula

     If the measured PaCO2 is higher than the expected PaCO2, a concomitant respiratory acidosis is also present. Another formula.....

1 mEq/L change in HCo3 – PaCo2 changes by 1 (Acute)

1 mEq/L change in HCo3 – PaCo2 changes by 4 (Chronic)

6. If metabolic acidosis, then look at the Anion Gap.
   - If elevated (> 12), then acidosis due to. (Ketoacidosis, Uremia, Lactic acidosis, Toxins)
   - If anion gap is normal, then acidosis likely due to diarrhea, RTA, saline
7. If anion gap is elevated, then calculate the Delta-Ratio (Δ/Δ) to assess for other disorders.
   - Δ/Δ compares the change in the anion gap to the change in bicarbonate.
   - If ratio between 1 and 2, then only wide anion gap acidosis
   - If < 1, then there is a coexistent Normal anion gap acidosis
   - if > 2, then there is a coexistent Metabolic alkalosis present (or rarely a compensated chronic respiratory acidosis.)
8. If normal anion gap and cause is unknown, then calculate the Urine Anion Gap (UAG).
   - In RTA, UAG is positive.
   - In diarrhea and other causes of metabolic acidosis, the UAG is negative. (neGUTive in diarrhea)

Questions

1. What are the causes of High anion gap metabolic acidosis?
2. How do we classify lactic acidosis?
3. Does bicarbonate help in high anion gap metabolic acidosis?
4. Is there any correction of anion gap for serum albumin levels?

EXAMPLE

Ques - 3 yr boy with pneumonia, diarrhea with severe shock

– 7.22 / $PaCo_2$ 44 / PaO_2 65 / 90%

- Na 139 / K- 4.0 / Cl- 110 / Hco_3- 10

  What is the underlying acid base disturbance , cause and treatment ?

- pH (7.22) = acidemia
- **Winters formula** - $PaCo_2$ = 1.5 (HCO3) + 8 1.5 (10) + 8 = **23 but is higher**
- **AG** = 139 - (10 + 110) = **19 (high)**
- **Delta gap -** HC03 + change in the AG **,** 10 + (19 - 12) = 17 is **Low**

Ans- AG acidosis / Respiratory acidosis / Non AG acidosis

Wide Anion Gap acidosis due to Pneumonia, Septic shock

Narrow Anion Gap acidosis due to Diarrhea

Rx- with fluids, vasoactive, oxygen, ventilation antibiotics

If significant diarrhoea (bicarbonate losses in stool) with worsening acidosis and shock, can change IV maintenance fluid to D5 425 ml + Soda bicarbonate 75 ml + Kcl to improve catecholamine responsiveness.

Case 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, SpO_2: 96% RA. Peripheries are cold with feeble pulses. VBG done shows pH: 7.29, pCO_2: 38, pO_2: 100, HCO_3: 18, BE: -6 , Cl: 88, Na^+: 121, K^+: 2.8, Glu 80, Lac: 2.4

Questions

a. Interpret the VBG

b. what is the fluid of choice for this child to correct dehydration.

Case 2.

An 8 year girl has been brought for acute exacerbation of asthma.She's a known asthmatic, obese and poorly compliant with therapy. She is given 3 back to back nebulisations of 5mg of salbutamol. There was only marginal response, hence IV Magnesium sulphate was given, IV steroids administered and Salbutamol 5mg nebulisation given every 30 min. Ipravent nebulisation added and as SpO2 was 89%, O2 supplemented via Venturi at 40% FiO2.CXR showed no parenchymal infiltrates.A baseline ABG is done - pH 7.41, pO2 46, pCO2 34, HCO3 21, BE -2.3, Lactate 3.9, Glucose 103, Na 141, K 4.2, Cl 105,CBC - Hb 15.2, TLC 6.58, Platelets 312, Creat 0.6.

Three hours later she complains of feeling faint, though wheeze has improved and she's now on hourly nebulisation of salbutamol.

A repeat ABG is done - pH7.33, pCO2 35, pO2 52, HCO3 18, BE -6.2, Lactate 5.3, Glucose 213, Na 141, K 2.6, Cl 109

Questions

a. Interpret the ABG

b. What type of acidosis is this ?

c. How will you manage this patient ?

Case 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, SpO_2: 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, pCO_2: 24, HCO_3: 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 4.

15 years boy, dev normal went out with his friends at 5 pm. He was disoriented and drowsy after he returned home at around 8pm. There is no h/o trauma/anyone witnessing seizures? There are no fang marks. He was received in ED where he was intubated in view of GCS (7/15). VBG: pH 7.329 / pO_2 118/ pCO_2 20/ HCO_3 18 / BE: -10, Lac 3.6, Na 141 / K 4.7/ Cl 108. RBS: 107gm/dl. S. Osmolarity: 311, Calculated osmolality: 290, Urine ketones: negative.

Questions

a) Comment on the VBG
b) Calculate the osmolar gap.
c) What is the significance of osmolal gap
d) Mention some underlying causes which could result in this type of ABG changes

Case 5

A 2 year old boy is found lethargic with bluish discolouration 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

a. What is the cause of acidosis in this child?

b. Calculate delta gap

c. How do we treat the acidosis ?

Case 6

A 14 year old girl was found unresponsive at home by her mother when she returned from work. On the way to the hospital she developed seizures and vomited red coloured material. She was referred as a case of vatical bleed.

ABCs were managed in the ER, she was intubated and given benzodiazepines for the seizures, followed by Fosphenytoin loading dose.

When she was catheterised to monitor urine output, urine was also coloured reddish.

ABG and labs were as follows

pH 6.9, PCO2 43, pAO2 85, HCO3 4.8, BE -27.5

Na 142, K 3.4, Cl 104

Glucose 229, BUN 12, Creat 1.3

SGPT 37, SGOT 55, Alk PO4 345, GGTP 17, LDH 334 U/L

Questions

a) Interpret the ABG

b) Calculate the anion gap and delta gap

c) How will you manage this patient ?

Case 7

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 nonconsanguinous 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

a. Interpret the ABG
b. Calculate the anion gap and delta gap
c. How will you manage ?

Case 8

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

a) What is the cause of metabolic acidosis here?

b) Calculate the anion gap

c) Does soda bicarbonate correct acidosis here?

Case 9

10 year old female child (30Kg) presented with vomiting for 2 days, Pain abdomen for 1 day, breathing difficulty from last night. H/o recent onset increased thirst, increased urination, weight loss, No significant past illness. O/E HR 140/min, RR 40/min, BP 90/50mmHg, SpO2 98% RA, minimal WOB, chest b/l clear, cold peripheries, feeble peripheral pulses, lethargic but conscious. Blood gas showed pH 6.98, Pco2 9.4, Hco3 2.2, base excess -12, lactate 0.74.CBG 394mg/dL, blood ketones 7 mmol/dL.

Questions

a) Interpret the blood gas. What is the expected pco2 here?

b) What is the relationship between measured sodium levels and glucose?

c) What is delta ratio and its implication here?

d) What is Strong ion difference and its clinical application?

Case 10

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

a) What is the cause of metabolic acidosis here?
b) How do we manage this acidosis?
c) Is there a role of renal replacement therapy here?

Case 11

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

a) Interpret the blood gas and probable primary cause.
b) How do we manage this acidosis?

Approach to hypernatremia in children

Contributors: Dr. V Poovazhagi, Dr. Shobana Rajendran, Dr. V.Vimalraj, Dr. L.Chidhambharam

Definition: Serum Na > 145 mmol/ L or mEq/L. Some references mention as > 150 mEq/L

Salt and water physiology

60% of body weight is constituted by water. Between extracellular fluid (ECF) and intracellular fluid (ICF) is the cell membrane. Only water permeates through the cell membrane. This transport is from lower tonicity to higher tonicity which means in hypernatremia it is from intracellular to extracellular compartment. The electrolyte composition of ECF and ICF has almost equal osmolality but electrolyte concentration is different. ECF: Na 135-145 mEq/L K 3.5 -4.5 mEq/L and ICF: Na 10-20 mEq/L K 120 - 150 mEq/L. Sodium is closely related to water balance. Thirst and ADH release are the major defences associated with hypernatremia. Threshold for thirst begins at 5-10 mosm/kg higher than that for ADH release

Causes of hypernatremia (Table 1)

Hypernatremia develops only by two mechanisms

1. Loss of free water (most common) – could be as loss of water or hypotonic fluids leading to hypovolemic hypernatremia
2. Gain of sodium (less common) – mostly iatrogenic; rarely accidental leading to hypervolemic hypernatremia

Table 1 showing the causes for hypernatremia

Hypotonic fluid or electrolyte free water loss	Excess salt or gain of salt
GIT : Diarrhea	Improper ORS or formulae
Renal: osmotic agents Diabetes Insipidus Chronic kidney disease Acute tubular necrosis	Iatrogenic Bicarbonate or hypertonic saline

Skin :Burns , increased sweating	Salt poisoning Child abuse Salt with water instead of sugar Pica Hyperaldosteronism

Clinical features

1. Dehydration and shock: Not obvious as ECF water content is near normal. Subtle findings – thirst and “doughy” skin, tachycardia, wt loss
2. CNS symptoms: Convulsions, irritability, high-pitched cry and. Some alert infants are very thirsty.
   a. Brain haemorrhage : Brain shrinks, results in tearing of bridging blood vessels - subarachnoid, subdural, parenchymal bleed .
   b. Thrombosis: Stroke, dural sinus thrombosis, peripheral thrombosis, and renal vein thrombosis - possibly due to hypercoagulability.

Lab investigations

- CBC: high HCT favours dehydration
- CXR: Can identify volume overload-pulmonary congestion, pleural fluid USG Lungs: presence of B lines. IVC filling will indicate the volume status
- Urine Sodium: < 10 favours dehydration > 20 – Salt excess or renal losses FENA: < 1.0 – hypovolemia > 2.0 Salt excess and hypervolemia
- Blood sugar, urea, creatine, other electrolytes, calcium and magnesium. High serum chloride – Salt excess
- Serum and urine osmolarity
- Serum AVP level and response to AVP
- Neuroimaging: For cause in DI and CNS complications
- ABG: In a setting of edema, hypertension, hypokalemia, hypernatremia and metabolic alkalosis – hyperaldosteronism

Approach to hypovolemic hypernatremia (six steps)

1. Stabilise the child -- ABC control seizures
2. Volume replacement as NS bolus if hemodynamics compromised followed by fluid deficit correction either formula based or simple strategy based and maintenance fluids
3. Monitor the rate of fall (0.5 mEq/L/hr) and adjust fluids accordingly. (Track Sodium 4 th hrly. If drop is more than expected reduce the fluid administration rate or change to less hypotonic fluid and vice versa)
4. Look for specific diagnosis like DI & specific therapy

   a) UO > 4 mLkg/hr. Serum osmolarity > 300; Urine osmolarity < 300

   b) Check plasma AVP level. - Trial of vasopressin
5. Monitor for following
   a) Any time shock appears (CHECK LACTATE) - another bolus of NS 10 mL/kg
   b) If volume overload heart failure, pulmonary edema or AKI – dialysis
   c) Any time seizure –give 3% saline 2-5 mL/kg over 15-30 min
   d) Check CBG – Don't use insulin but adjust dextrose content D5 to D2.5
6. Switch to oral or nasogastric feeds for free water deficit as the child improves

   Deficit correction various formula are available. Eg for a 10kg child with 175 meq/L

   Formula 1:

   Free water deficit in L = Current TBW x ([current plasma Na/140] - 1)

   (10 x 0.6 = 6) x ([175/145]) – 1=6 x (1.21-1.0). = 1.26 L = 1260mL

   Formula 2:

   Free water deficit in mL = (4 mL) x (BW in kg) x (desired change in plasma Na)

   4 x 10 x (175-145 = 30) = 1200 mL

   Formula 3:

   Free water deficit = BW x 0.6 {1 – (145/Current Na)}

   6 x {1-0.83 = 0.17} = 1.02L L or 1020 mL

   For correction add maintenance to the above formula

Time taken to correct (175-145) = 30 mEq/0.5 = 60 hrs

Hourly deficit replacement -1200/60 = 20 mL/hr of free water + 40mL/hr maintenance fluid(NS or ½ NS) =60mL/hr

Formula 4:

Choose the IVF of choice – NS, ½ NS, ¼ NS or 1/5 NS 2)

Calculate amount of drop in Na if one Litre is given E.g ½ NS (77 mEq/L)

$$\text{Change in serum Na} = \frac{\text{Infusate Na - serum Na}}{\text{Total body water + 1}}$$

When one Liter ½ NS is given = (77-175/6+1) = 14.0 mmol drop in Na

(175 -145 = 30 mmol/l correction needs 30/14.0 = 2.0 L of ½ NS

Rate of administration = 2.0 L /60 hrs = 33 ml/hour of ½ NS + 40mL/hr = 77mL/hr

SIMPLE USEFUL FORMULA

1.25 -1.5 times maintenance fluid as ½ NS to 1/4 NS in D5

Approach to Isovolemic /hypervolemic hypernatremia (five steps)

1. Stabilization of ABC and seizure control.
2. Confirm by lab: Urine Na > 20 mEq FENa.> 2. Impaired renal function
3. IV Frusemide as infusion (0.1-0.2 mg/kg/hr) to reduce fluid overload.
   Meticulously measure UO. Replace UO with ¼ or 1/5 GNS. Serum potassium, should be monitored and should be replaced as needed
4. Stop 3% Saline or sodium bicarbonate
5. Consider RRT, if UO low, or creatinine high or very high serum Na> 180 or multiple electrolyte deficiency or fluid overload and CCF

To remember

- Seizure during hypernatremia correction can be due to rapid fall of Na so treat with 3%saline

Hyperglycemia with hypernatremia does not warrant specific therapy.

- Acute hypernatremia can be corrected rapidly if < 24 hours but if duration is more 48hours rapid correction is avoided as idiogenic osmoles will alter the physiology.
- Higher the Na value : more severe dehydration : continue isotonic fluids to avoid rapid drop in NA whilst correcting intravascular dehydration
- Vasopressin is useful in cases of diabetes insipidus to reduce the free water loses.

Scenario 1

14 days old late preterm boy was brought to ER with fever , lethargy and refusal to feed and history revealed the following . He was born at 35 week with birth weight of 3 kg. He was on exclusive breast feeding till day 10, mother had bilateral sore nipples, and he had 3-4 high colored small volume urine, started on formula thrice daily (30 ml water with 2 scoops of formula)

On examination he deeply icteric till legs, febrile with fore head sweating, weight was 2.4kg 18% weight loss, his pulses were well felt with heart rate of 170/min ,BP was 58/40 mmHg.

Enumerate the important findings in the history

What are the probable causes?

What tests to order?

What is the probable diagnosis?

Outline the management

Scenario 2

4 yrs/ Boy, Wt: 14 kg ,presented with fever & cough 4 days, 1 episode of GTCS, and altered sensorium 1 day. Examination revealed the following GCS 12/15, stridulous breathing, neck stiffness + started on AB, AED, antiviral.

GCS deteriorating, how will you proceed? What is the fluid plan? Child developed hypernatremia, raised

urea creatinine

How to proceed further?

Scenario 3

3 year girl/ 10 kg/ Craniopharyngioma -Operated, Pre op TFT, cortisol, and electrolytes were optimized. Call from recovery room 6 hours after surgery – saying child is tachycardia, hypotensive with normal Serum Na (145) and low urine output

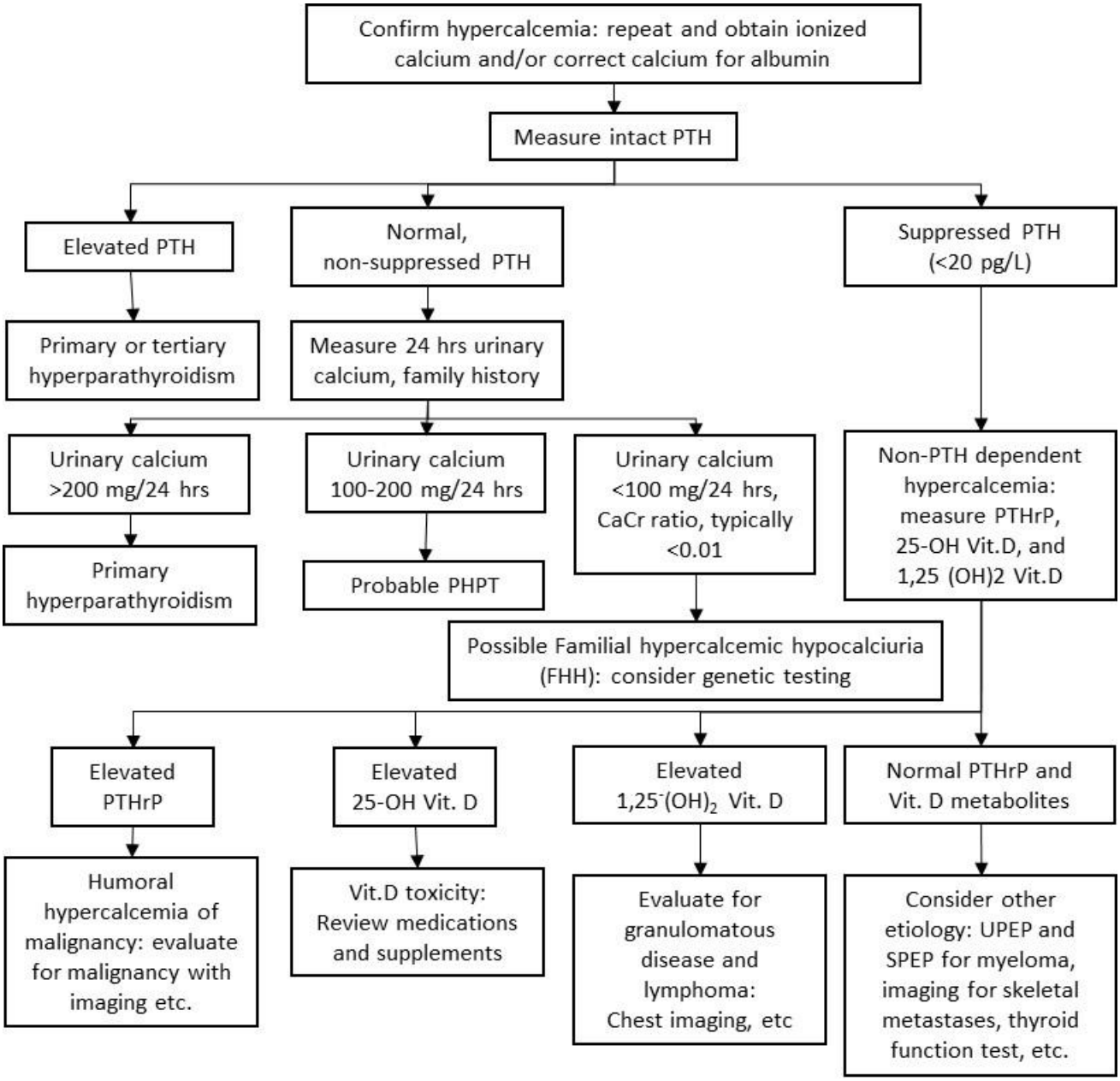
- Possible DD?
- In PICU – 12 hours later – child is irritable suddenly UO increased from 40 – 120 ml /hr
- Next plan of action? Contrast different causes of polyuria with serum Na in respective conditions S.Na -170 (12 hrs) in this child. UO- 100 ml/hr
- Diagnostic criteria for DI ? What type of fluid (NS, RL,0.45 NS) , how much and at what rate should we start?
- Role of vasopressin – IV or SC or ORAL – and how to titrate?
- S.Na 176 (4 hrs later) – possible causes? How to treat?
- D4 – Urine output (10 ml /hr) – child drowsy – S.na – 125 . What are the DD? How to treat
- D7 – S.Na – 150. UO – 3ml/kg/hr .Child playful. What is discharge plan?

What is triphasic response in craniopharyngioma post op child? How to treat permanent DI?

HYPO/HYPERCALCEMIA & HYPOMAGNESEMIA

Contributors: Dr. N.C.Gowrishankar, Dr. L.K.Premkumar, Dr. T.S.Ekambaranath, Dr. R.Venkateshwari

Hypercalcemia- approach



Step 1 Document hypercalcemia

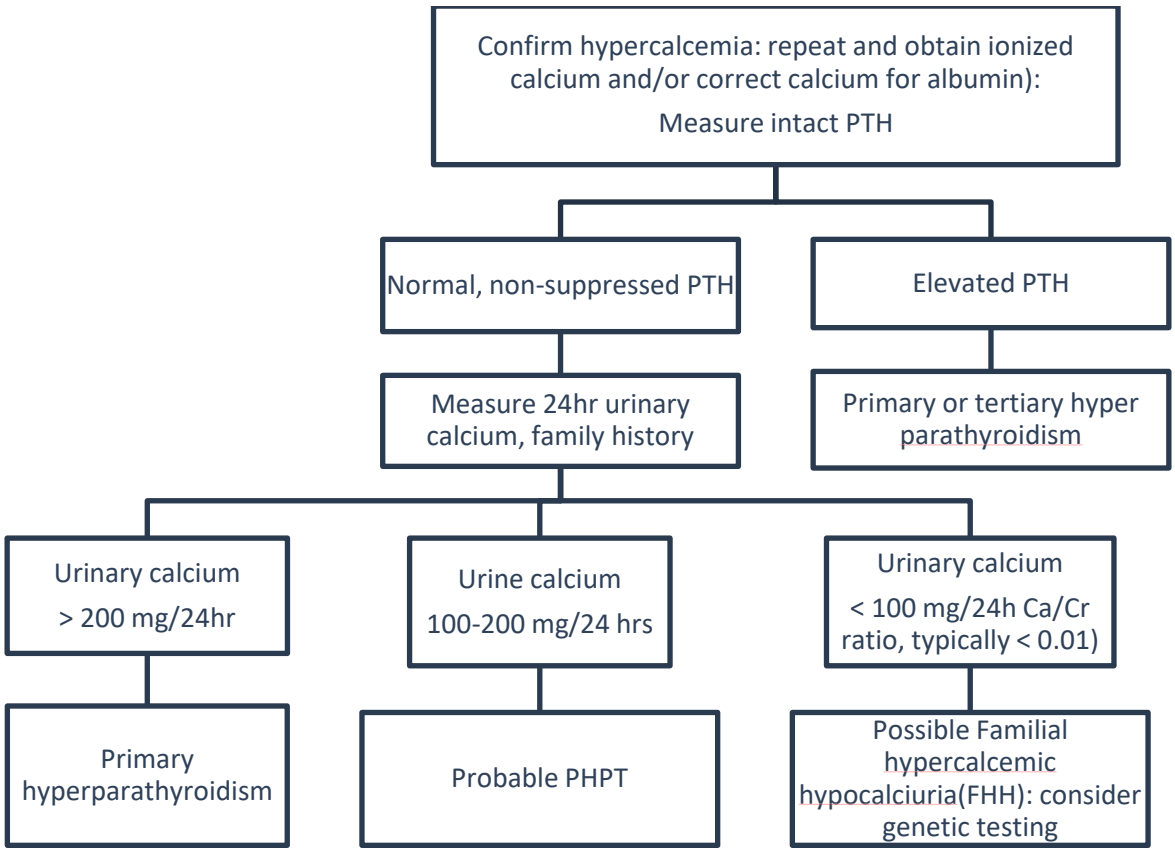
Step 2 PTH level – if high – primary/tertiary hyper parathyroidism

Step 3 PTH level – if normal – measure 24 hr urine calcium

If > 200 mg/24 hr: primary hyperparathyroidism

If 100-200 mg/24hr: probable primary hyperparathyroidism

If <100& urine Ca/Cr ratio: < 0.01: possible familial hypercalcemic hypercalciuria- need genetic testing



Step 1 Document hypercalcemia

Step 2 Measure PTH level

Step 3 if suppressed < 20 pg/mL - Non-PTH dependent hypercalcemia

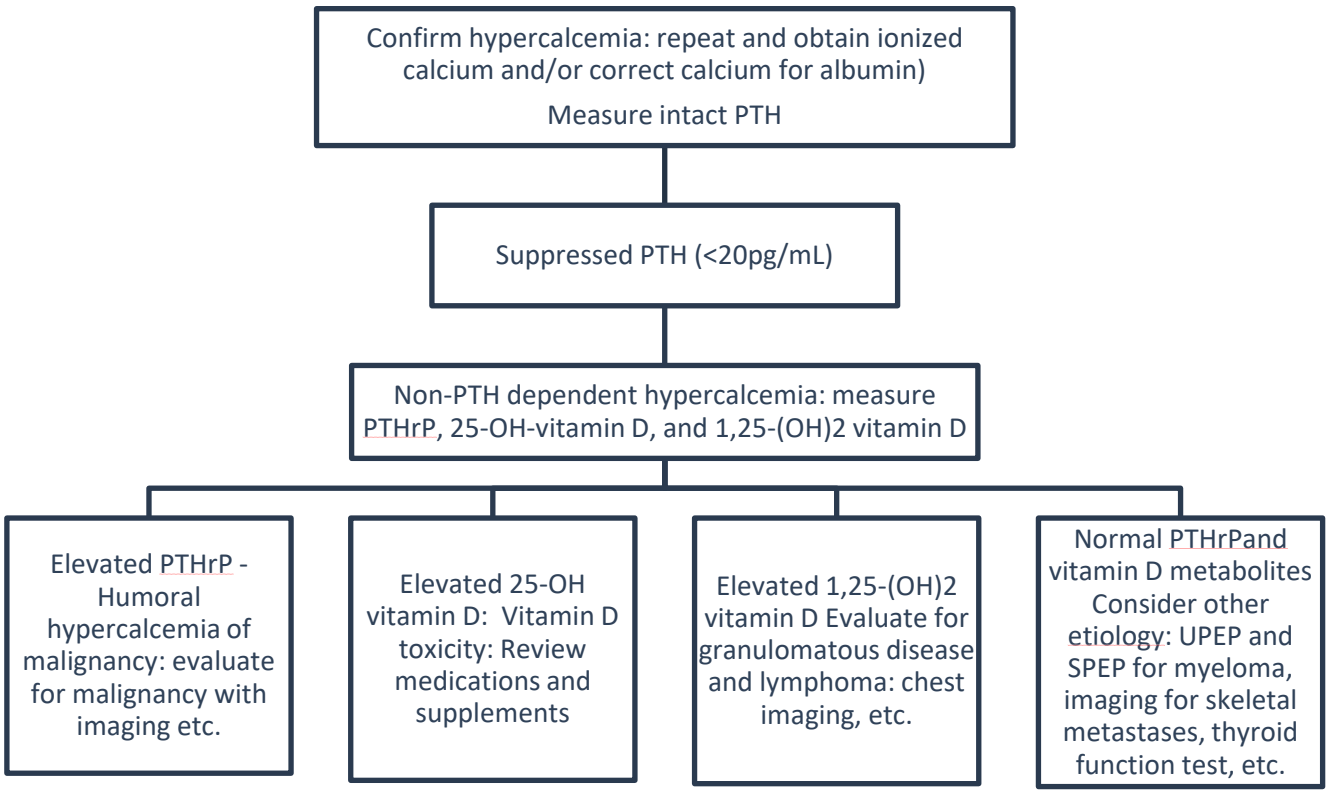
Step 4: measure PTH related protein, 25-OH-vitamin D, 1,25-(OH)2 Vit D

Elevated PTHrP: Humoral hypercalcemia of malignancy: evaluate for malignancy with imaging etc

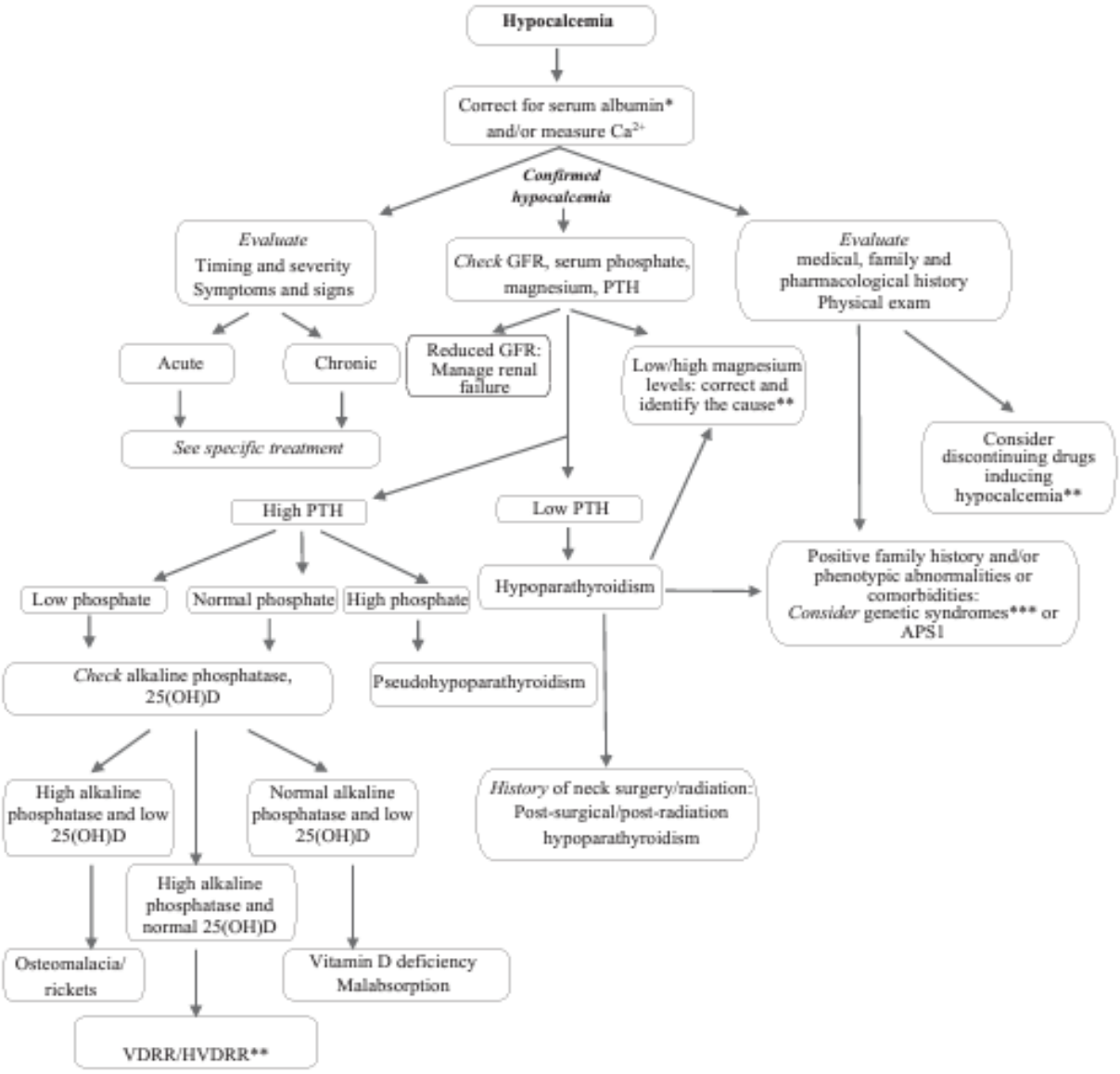
Elevated 25-OH vitamin D Vitamin D toxicity: Review medications and supplements

Elevated 1,25-(OH)2 vitamin D Evaluate for granulomatous disease and lymphoma: chest imaging,etc.

Normal PTHrPand vitamin D metabolites Consider other etiology: UPEP and SPEP for myeloma, imaging for skeletal metastases, thyroid function test, etc.



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

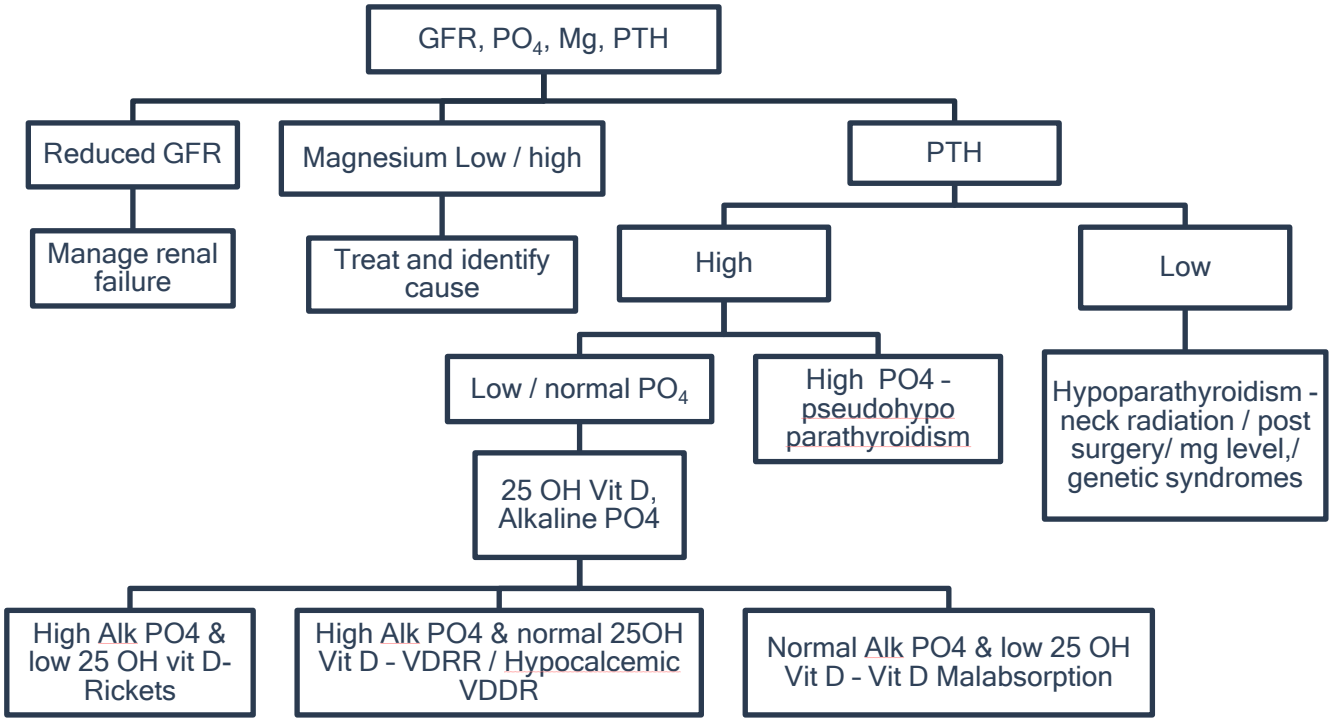
Step 4 IV calcium gluconate (1g= 93 mg elemental Ca) / IV calcium chloride (1g= 100-300 mg elemental calcium) - infusion

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 harmone



Case scenario 1

4 years old previously well male child, was brought with complaints of vomiting, decreased activity and refusal of feeds for 3 days.

Drug history: Given 5000 IU/day of vitamin D for 1month, 16000 IU/day for 1 month.

At admission, child was lethargic, hydrated. His vitals were normal. His weight was 18kg. Systemic examination was normal.

Baseline investigations showed total calcium of 17.6mg/dl.

1. What will be the first step in the treatment of this child?
2. What history and physical findings will support your diagnosis?
3. Have you identified any underlying risk factors in this child?
4. What other diagnostic tests would you like to do in this child?
5. How would you manage this child?

Case scenario 2

12year old girl presented with 2 weeks history of worsening epigastric pain, vomiting.

On examination, she was lethargic with signs of some dehydration. She had bradycardia (60/min). Musculoskeletal examination revealed muscle weakness in both legs with presence of genu valgum. There was no bone pain. No palpable lumps in the neck. Her total calcium was 18mg/dl.

1. What history and physical findings will support your diagnosis?
2. Have you identified any underlying risk factors in this child?
3. What other diagnostic tests would you like to do in this child?
4. How would you manage this child?

Case scenario 3

- 11year old mch, a k/c/o SRNS admitted to ER with seizures lasting for 5 min.
- H/o vomiting 3 days. No fever,BP 100/70
- Was on prednisolone, furosemide, calcium, enalapril-stopped for 3 days
- Urea 81, creatinine 3.8, albumin 1.3, calcium 4.9, Phosphorous 5.6 urine albumin +++,PTH 56pg/ml (10-65),VIT D 2ng/ml
- ABG :PH 7.06PCO2 11.8 HCO3 3.4 iCa 0.7

Q 1 How will calculate corrected calcium in hypoalbuminemia

Q 2 What are the causes of hypocalcemia in this child

Q3 What conditions are associated with reduced ionised calcium levels

- Inj Cal glu 0.5 ml/kg iv tds
- Inj sodabicarb 5ml/kg iv 2 days then oral
- Tetany on day 3

Q 4-What is the reason for recurrence of symptoms?

- Started on calcitriol & followed up

Case scenario 4

- 40 days old baby was admitted with c/o seizure in the form of uprolling of eyes and tonic posturing of limbs.
- Ser cal-6.4mg/dl, albumin 3.7,Alk po4 434, SGOT 24,Na 136,K 5.5,Urea 30, creat-0.6
- Phosphorous 8.8, PTH 180 pg/ml, Vit D 17.4

Q 1 What could be the cause of hypocalcemia in this baby?

- IV calcium correction given
- Followed by oral calcium and Vit D

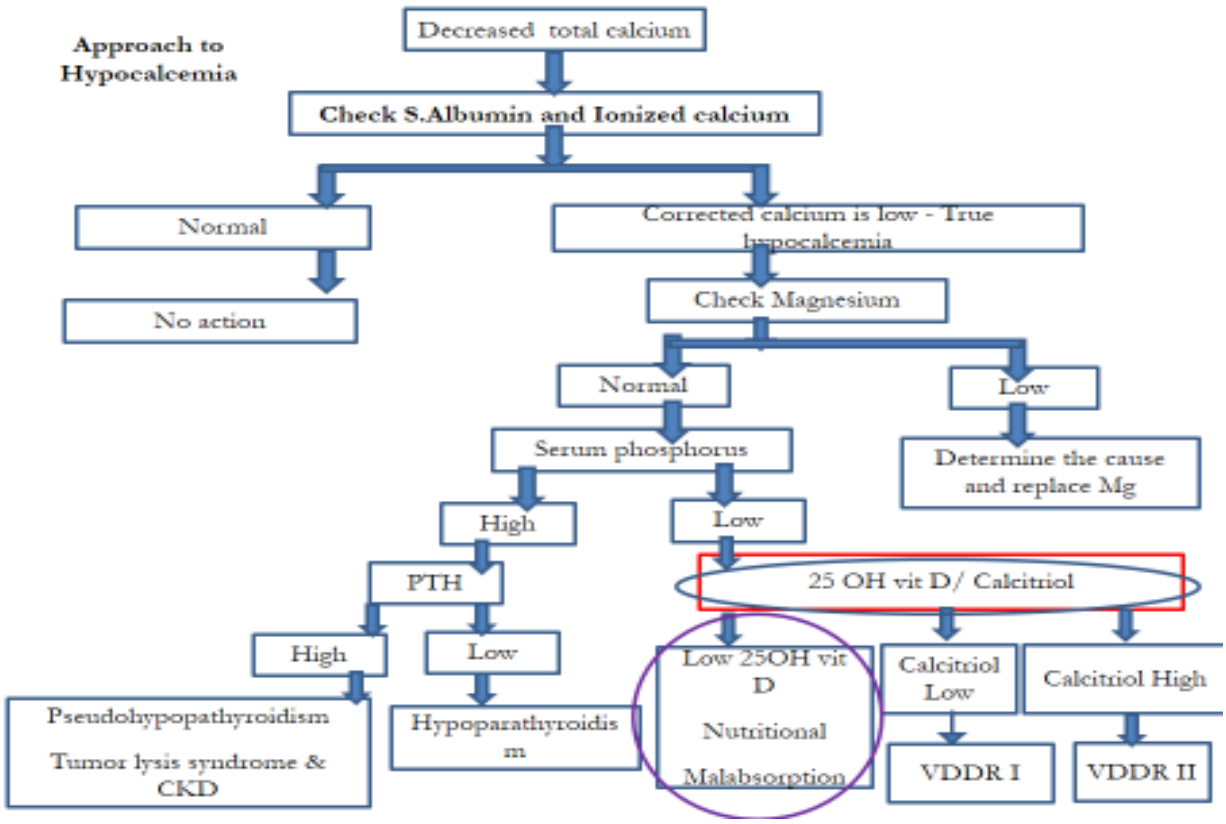
Q 2 What precautions to take while giving IV Calcium?

Q 3 Calcium gluconate is immiscible with what?

Recommendations for vitamin D and calcium deficiency – prevention and treatment

	Vitamin D (IU/day)			Calcium	
Age	Prevention	Treatment	Treatment with large doses	Prevention	Treatment
Premature neonates	400	1000	NA	150-220mg/kg/day	175-200mg/kg/day
Neonates	400	2000	NA	200 mg/day	500 mg/day
1-12 months	400	2000	60K/wk X 6 weeks	250 – 500 mg/day	500 mg/day
1 -18yrs	600	3000-6000	60K/wk X 6 weeks	600-800mg/day	600-800mg/day
At risk group	400-1000	As per age	As per age	As per age	As per age

- IM route with larger doses – consider only when compliance or absorption from the gut is an issue
- Obese children – 2 to 3 times (between 400-1000 IU/day) more vitamin D for their age group
- Vitamin D concentrations of >20 ng/mL – sufficient, 12-20 ng/mL – insufficient & <12 ng/mL - deficient



- For each gram of fall in albumin ser calcium falls by 0.8mg/dl
- Corrected calcium=measured Ca+{0.8 x(4.0-ser albumin)}
- Hyperventilation and metabolic alkalosis also cause hypocalcemia

Case scenario 4

2 yrs old male child presented with history of diarrhea of more than 4 weeks duration. On examination child was lethargic, dehydrated and was afebrile. His vitals were stable except for mild tachycardia. Investigations sent showed normal blood counts and negative CRP. His electrolytes showed Na - 135mg/dL, K - 3.2mg/dL, HCO3 - 18 mg/dL .

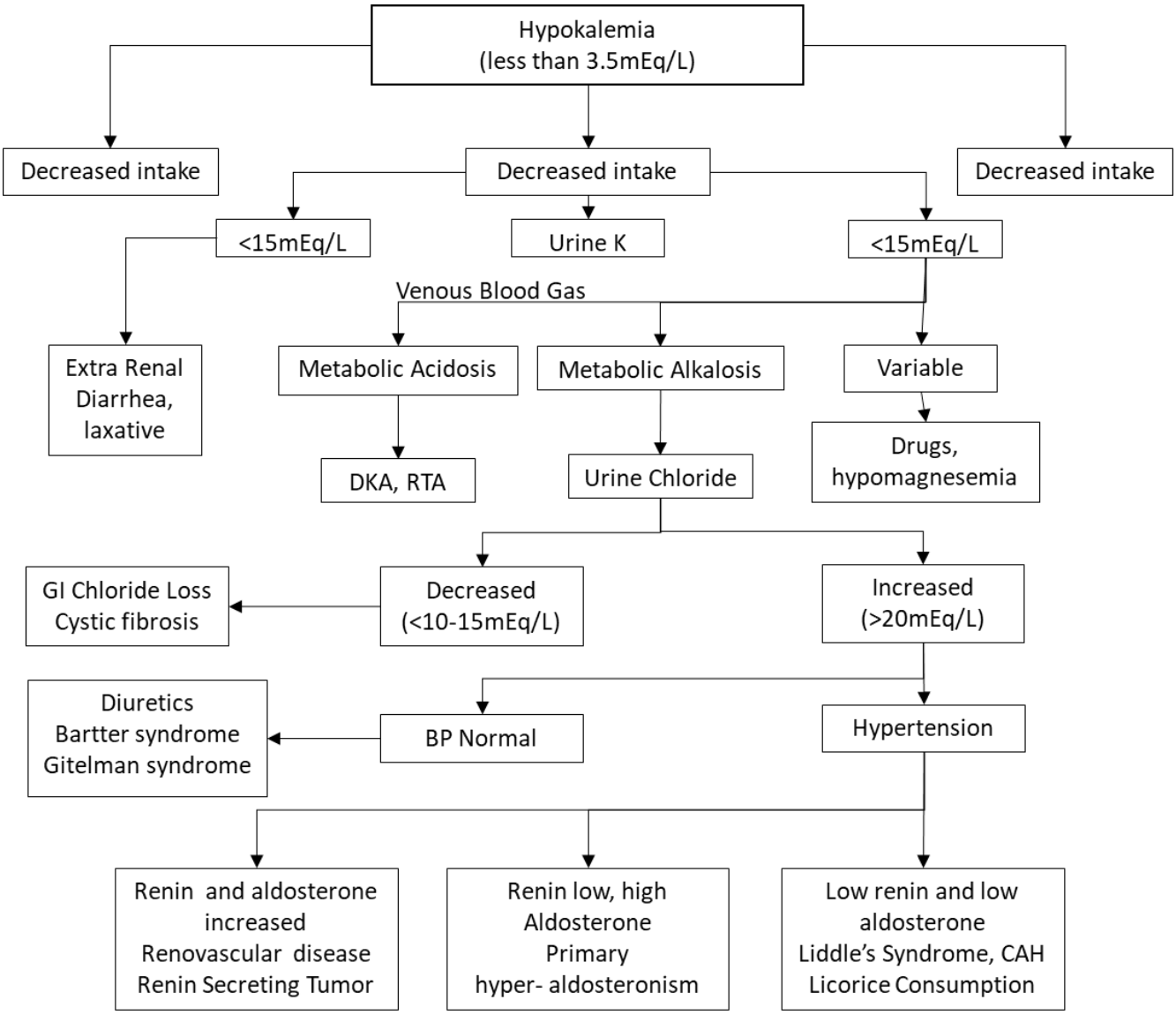
Child was treated appropriately with IV fluids & supportive treatment

On Day 2 of admission, child developed an episode of GTCS

1) What investigation to be done?
2) Child developed another episode of GTCS --- how do we proceed?
3) Dose of IV magnesium
4) Tests to differentiate the causes

HYPO/HYPERKALEMIA

Contributors: Dr. S.Shanthi, Dr. Sridevi A Naaraayan, Dr. M.Karthikeyan, Dr. Manasi Garg



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.

Hyperkalemia

Definition: Serum or plasma concentration of K >5.5mEq/L; in neonates > 6mEq/L (serum K is 0.1-0.7 mmol/L higher)

- Mild hyperkalemia - 5.5-6 mEq/L.
- Moderate hyperkalemia - 6 -7 mEq/L.
- Severe hyperkalemia - >7 mEq/L and or presence of ECG changes

Causes of hyperkalemia

1. Increased K intake- IV/Oral, blood transfusions, parenteral nutrition
2. Increased production followed by transcellular shift-Tumor lysis syndrome, Excessive trauma, rhabdomyolysis, hemolysis, malignant hyperthermia
3. Transcellular shifts- metabolic acidosis, drugs-succinyl choline,beta blockers, digoxin, hyperosmolality(mannitol), hyperkalemic periodic paralysis

Symptoms

May be asymptomatic. Symptoms can range from muscle weakness to ascending flaccid paralysis, palpitations, syncope, arrhythmia and sudden cardiac arrest. Respiratory depression, ileus and paresthesia can occur

ECG changes include tall, peaked T waves, prolonged PR interval, progressive widening of QRS, Sine wave((fusion of QRS and T wave),VT, VF, asystole

A normal ECG does not exclude risk for arrhythmia, as life threatening arrhythmia can occur without warning

Approach

- **Step1:** 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.

Emergent treatment is needed if symptomatic or in the presence of ECG changes, asymptomatic severe hyperkalemia, and in moderate hyperkalemia patients who are likely to have a rise in K as in children with tumor lysis syndrome or rhabdomyolysis.

Care of ABC is the priority. Correct shock with isotonic fluids as this will improve renal excretion of K.

Arrhythmias are treated as per PALS protocol though antiarrhythmic drugs may not be very useful unless emergent measures are taken to reduce potassium levels.

Treatment modalities

1. 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.
2. 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)
3. Increase potassium excretion (cation exchange resin, diuretics, dialysis)

4. Stop all K containing fluids and drugs that cause hyperkalemia. Avoid blood transfusion in a child with hyperkalemia unless absolutely necessary.
5. 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

Step 2: If patient is asymptomatic and does not have setting of hyperkalemia rule out pseudohyperkalemia

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. Blood sampling proximal to an intravenous line with potassium containing fluid can falsely increase the potassium level.
c) Leucocytosis and thrombocytosis also can increase serum K levels but plasma levels are normal. Check plasma K rather than serum K.
d) Repeat potassium levels from a non-hemolysed sample when pseudohyperkalemia is suspected.

Step 3: Take a detailed history and do a clinical examination to find out the etiology.

History

- H/o burns, trauma leading to crush injury, diabetes, excess potassium intake
- Palpitations, fasciculations, syncope and parasthesias
- Drug intake

- H/o renal disease, UTI
- 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
- H/o blood transfusion
- H/o surgery, anesthesia (malignant hyperthermia)
- H/o chemotherapy in a child with a large tumor load
- Color of the urine (cola colored in AGN, hemoglobinuria, rhabdomyolysis)
- Bloody stools may occur in HUS
- Family history of similar disorder (Single gene disorders causing hyperkalemia, malignant hyperthermia, neuromuscular disorders, familial hyperkalemic periodic paralysis)

Clinical examination

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

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
- Secondline: CPK, uricacid , USG abdomen, urine potassium and sodium

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

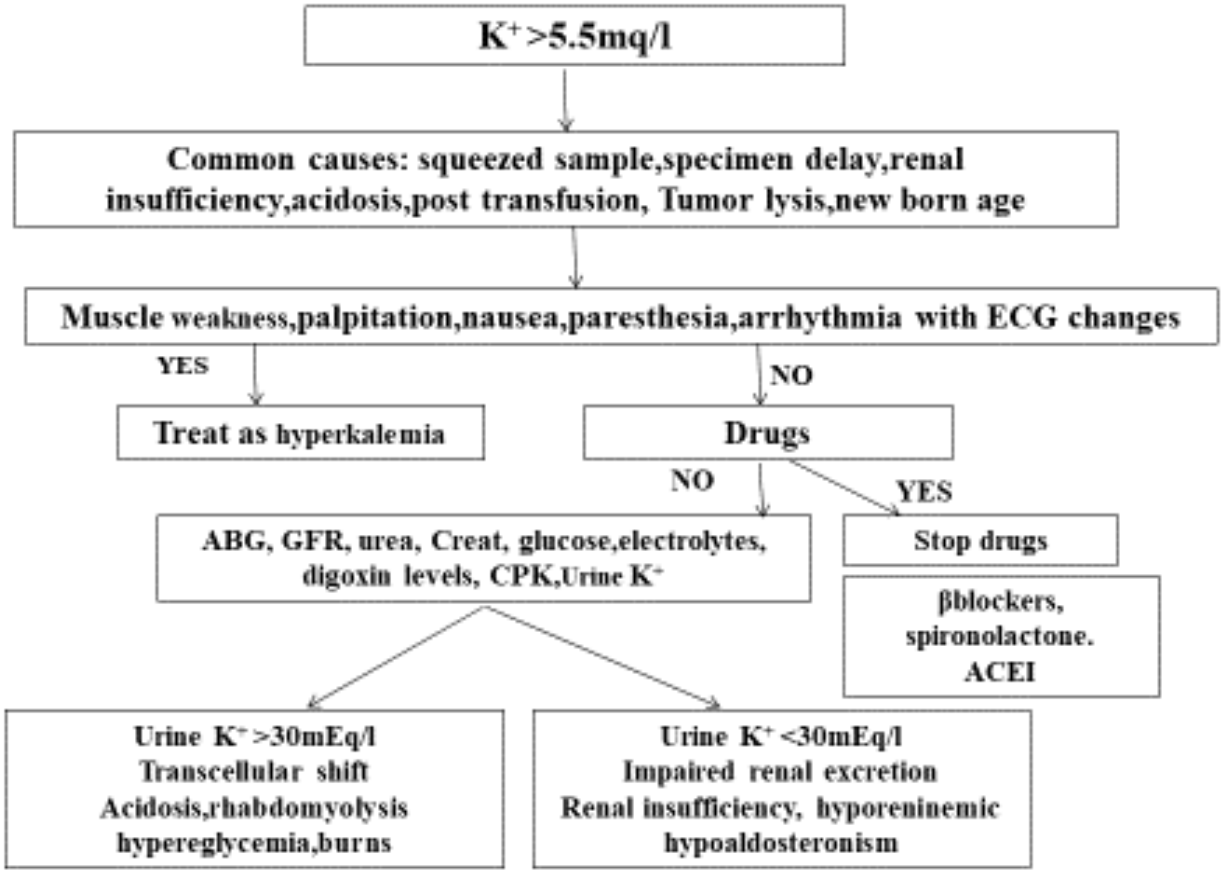
In intrinsic renal disease both renin and aldosterone are decreased; both are increased in pseudohypoaldosteronism. Renin high aldosterone low- CAH , hypoaldosteronism.

Management of moderate hyperkalemia without ECG changes

- Salbutamol nebulisation, Insulin dextrose , Kayexelate , bicarbonate if metabolic acidosis

Management of mild hyperkalemia without ECG changes

- Stop K supplements, salbutamol nebulisation, Kayexelate, diuretics



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

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 investigation 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

1. If renal loss of K is suspected do urinary K TTKG > 4 or urinary K >15mEq/L indicates renal loss.
2. Do a VBG

   Hypokalemia with metabolic acidosis- consider RTA, DKA

3. 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, hypocalciuria), EAST syndrome(normal calcium excretion)

If BP high

Consider hyperaldosteronism, renovascular disease, Cushings syndrome

Case scenario 1

A 3-year-old girl is brought with complaints of passing watery stools 8 to 10 times a day for 3 days, 3 to 4 episodes of vomiting per day for 2 days and abdominal distension for 1 day. She does not have fever. She is passing urine less frequently around 3 to 4 times per day since yesterday. On examination, she is looking lethargic, tongue is dry, has sunken eyes, skin pinch that goes back very slowly, height is 90cms and weight is 9kg, has stable vitals including blood pressure, has abdominal distension with sluggish bowel sounds and hyporeflexia. Her investigations reveal normal hemogram and negative CRP. Her biochemical values are as follows – Blood sugar -96mg/dl, Urea – 16mg/dl, Creatinine – 0.5mg/dl, S. sodium – 142 mEq/L, S. potassium – 2.6 mEq/L, S. chloride – 98mEq/L, S. bicarbonate -26 mEq/L.

What is the complete diagnosis?

How should the child be managed?

Case scenario 2

A 1-year-old girl was admitted due to vomiting and poor feeding past five days. The child's vomiting was non-bilious and occurred frequently around 6 - 8 times a day. Child had history of previous similar episodes, treated elsewhere in nearby health care facilities with IV fluids and medications but parents didn't have any medical records for the same. Physical examination revealed failure to thrive . The child weighed 4 kg with a length of 62 cm. In addition, child had loss of muscle tone. Child was not able to lift her head and limbs. Developmental milestones were abnormal. On evaluation child's hemoglobin was 10g/dl, WBC and platelets were normal, urea 15 mg/dl and creatinine 0.7 mg/dl; Na 136meq/l, K 2.4 meq/l, Cl 93meq/l, bicarbonate 28 meq/l, serum calcium 8.8 mg/dl, phosporus 3.9 mg/dl, serum

magnesium 2.4mg/dl; ABG- pH 7.5 ,Pco2 -48 meq/l, HCO3-32meq/l, PaO2-92mm hg. Urine sodium 58 meq/l Urine potassium – 50 meq/l, urine chloride 40 meq/l, urine calcium creatinine ratio was 0.5 mg/mg

Questions

What is the approach to evaluate the case?

What is the most likely diagnosis?

How would you treat this case?

Case scenario 3

3 years old female child was diagnosed as nephrotic syndrome at 18 months of age-on regular follow up. She was treated as steroid resistant NS . Histopathology was suggestive of FSGS. She was started on calcineurin inhibitors (tacrolimus) and ACEI

Edema was managed with on and off diuretics. Currently she presented to Emergency department with periorbital puffiness, abdominal distension and decreased urine output. On examination she was lethargic, afebrile, HR 96/min, RR 32/min,BP 90/ 60mmHg, perfusion-normal, anasarca present. Urea 90 mg/dl and creatinine 1.4 mg/dl; Na 135meq/l, K 6.8 meq/l, Cl 104meq/L.

- What is your diagnosis?
- What are the probable causes of hyperkalemia in this child?
- How do you manage this child and what are the preventive measures you should take to prevent hyperkalemia

Case scenario 4

A 4 years old boy has c/o cough, cold and fever for 4 days. Mother complained of fast breathing, excessive sleep and not passing urine for > 14 hours. On examination, child was pain responsive, febrile 102 F, HR: 178/min, RR: 72/minute, peripheral pulses weak, CFT: >5 sec, BP: 80/40 mm of Hg, RS: B/l Crepts, occasional rhonchi, liver span normal.

Hb: 9.8, TLC: 38560, DC: P92, L08, Platelet: 142000, Urea-132mg/dl; cr-2.3mg/dL; Na/K/Cl/Hco3-130/ 7.5/92/4; ABG: pH: 6.9, PCO2 35mm Hg, PO2 82mm Hg, Lactate 8.9 mmol/L

1. What are the ECG changes in hyperkalemia?
2. What are the factors contributing to hyperkalemia in this child?
3. What is the probable diagnosis and how do you manage this child?
4. What is TTKG and it's role in hyperkalemia?