CASE SCENARIOS ON HYPOKALEMIA

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CASE 1

- A 12 year old girl weighing 40kg presents to you with abdominal pain, vomiting and rapid breathing for 1 day. Parents say she has lost weight over the past few weeks and has been very thirsty and passing urine frequently.
- On examination she is drowsy but arousable dehydrated and tachypneic with deep labored breathing.
- Vitals: HR 130/min, BP 100/70mmHg, RR 32/min. Peripheral pulses feeble. Central pulses well felt.

HOW WILL YOU APPROACH

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

APPROACH

1.Physiological status

- Compensated shock

2.Immediate management

- Care of ABC
- Airway positioning as needed
- Breathing: O2 NRBM 100%
- Circulation: secure 2 IV lines, start on 20ml/kg bolus NS over 20 min and reassess her. Repeat bolus till shock resolves.
- While securing lines take samples for CBG, electrolytes, RFT , venous blood gas
- Cardiac monitoring for hypo/hyperkalemia

3.Probable etiology?

- From history and examination findings, would suspect DKA

INVESTIGATIONS

- CBG: 400 mg/dL
- Blood gas → pH 7.05 HCO_3^- 8 mEq/L anion gap 22.
- Serum sodium :132mEq/L
- Serum potassium: 2.9 mEq/L
- Serum glucose: 410mg/dl
- Urea : 20mg/dl
- Creatinine: 0.7mg/dl
- Urine is strongly positive for ketones and glucose.
- Blood beta hydroxy butyrate (to do if available)
- 12 lead ECG showed flattening of T waves

IMPRESSION: Severe DKA with Hypokalemia

HOW TO PROCEED?

- Fluid therapy Once shock is corrected deficit+ maintenance over 24-48 hours
- Start IV KCl since there is ECG change

WHAT HAPPENS TO POTASSIUM IN DKA?

- Severe **total body K^+ depletion** due to osmotic diuresis, vomiting
- Volume depletion causes secondary hyperaldosteronism, which promotes urinary potassium excretion.
- Although total body potassium is decreased, serum levels at presentation may be normal or high due to redistribution in response to acidosis and insulin deficiency and reduced renal excretion.
- Severity of hypokalemia may be higher in malnourished children.
- In our case, **low value = extreme deficit**.

WHAT HAPPENS DURING TREATMENT?

- Administration of insulin and the correction of acidosis drives potassium back into the cells, decreasing serum potassium levels
- Insulin also has an aldosterone-like effect leading to increased urinary potassium excretion.
- Potassium replacement is required regardless of the serum potassium concentration, except if renal failure is present.
- If the child is hyperkalemic, defer potassium replacement therapy **until urine output is documented and K< 5.5mEq/L**

POTASSIUM REPLACEMENT IN OUR PATIENT

- With baseline K^+ 2.9mEq/L, starting insulin would cause **life-threatening hypokalemia → arrhythmias, muscle weakness and respiratory failure**.
- IV KCL - 0.5 mEq/kg/hr (KCl→1ml=2mEq) with cardiac momitoring
- Recheck K^+ after 1–2 h
- Start Insulin infusion 0.05–0.1 U/kg/hr (no bolus) once potassium normalizes.
- Add 40mEq KCl /Litre IV fluids (10ml KCl in 500ml IVF)

OTHER MONITORING TO BE DONE

- Monitor sugar hourly (expected glucose drop 50–100 mg/dl/hr)
- Continuous ECG (look for U waves, QT changes)
- Urine output hourly, daily weight
- Frequent GCS monitoring for cerebral edema
- Check Mg^{2+}/PO_4^{3-} at 4–6 h, replace if low
- Once acidosis resolves,
- Switch to subcutaneous insulin, overlap with IV for 30–60 min
- No oral K^+ if labs normal and child is eating

CASE 2

A 10 month old male infant is brought with a 3 day history of profuse watery diarrhea ,decreased feeding and excessive sleepiness. Parents say he has passed more than 8–10 stools per day, no blood and has vomited 2–3 times. On examination he is lethargic when undisturbed, has sunken eyes, dry tongue and sluggish skin pinch.

Vitals

- HR 160/min
- BP 70/50mm Hg
- RR 28/min
- Weight: 8kg (Weight loss is about 12% of baseline)
- Capillary refill time is 4 seconds

HOW TO APPROACH

1. What is your clinical diagnosis?

Acute watery diarrhoea with severe dehydration in shock

2. Next step in management?

- Stabilisation of ABC → Secure 2 IV lines , RINGER LACTATE 20ml/kg over 20 minutes . Reassess and repeat as needed.
- Severe dehydration (no SAM)- 30ml/kg RL over 1 hour followed by 70ml/kg RL over 5 hours

3. Do you need any investigations?

- Not for all diarrhea patients. RFT , electrolytes, CBG should be done if in shock or SAM.
- Lab tests show Na^+:132mEq/L, bicarbonate: 12mEq/L, K^+: 3 mEq/L and mild metabolic acidosis. CBG: 92mg/dl

MANAGEMENT

- Child needed 2 boluses and severe dehydration correction.
- ORS started.
- Ongoing losses replaced with ORS
- Breastfeeding and appropriate feeds started

QUESTIONS

Q1: How does acute diarrhea lead to hypokalemia and acidosis?

- Stool contains large amounts of K^+ and bicarbonate → direct losses in profuse diarrhea
- Repeated watery stools cause bicarbonate loss → metabolic acidosis (non-anion gap)
- Severe dehydration activates RAAS/aldosterone → renal potassium wasting
- Vomiting adds further K^+ and H^+ losses

Result: Significant hypokalemia with metabolic acidosis

QUESTIONS

Q2: How should potassium replacement be incorporated?

- Do not add K^+ to initial IV bolus fluids/ dehydration correction fluids while treating severe dehydration
- Most children improve with fluid resuscitation and ORS (potassium (20mEq/L))
- IV supplementation is rarely needed.

TAKE HOME POINTS

- All children with hypokalemia do not need extensive evaluation.
- Acute IV K correction will be needed only in symptomatic patients or those with ECG changes.
- Electrolytes measurement is not needed in all diarrhea cases unless the patient is in shock or has SAM

THANK YOU