Hypokalemic Paralysis from Distal Renal Tubular Acidosis (Type-1)

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Case Presentation

Chief Complaints

A 39 Years gentleman with no comorbid, NKDA and with a chief complaints of :

1. Tightness in bilateral calf muscles 2 weeks ago that settled on taking rest

2. Bilateral lower limb cramps 3 days ago, treated in outside clinic with intravenous fluids

3. Ascending weakness in both legs, thighs, hips and low back initially gradually worsened involving both hands since last 2 days

4. Mild breathing difficulty since morning

Patient was initially treated elsewhere and brought here for further Management.

Investigations done outside showed CPK - 491, Na+ - 149, K+ - 3, Cl - 115, urea - 36, creatinine - 1.6.

No past medical/surgical history.

On examination (1:45 PM)

Conscious, oriented, afebrile, hydration-fair, no PICCLE.
Vitals:

1. PR - 78/min
2. BP - 160/100 mmHg
3. RR - 18/min
4. SpO2 - 98% RA
5. Temp - 97.80 F
6. CBG - 136 mg/dl
7. GCS - 15/15

Systemic Examination

CVS - S1S2 +, no murmur, JVP - normal.

RS - B/L air entry equal, NVBS, SBC - 30.

PA - Soft, non- tender, bowel sounds sluggishly heard.

CNS: UMN- intact, EOM- full, B/L pupil 3mm-ERTL

VITALS	RIGHT	LEFT
Power UL	2/5	2/5
Power LL	1/5	1/5
Tone	Hypotonic	Hypotonic
DTR	Absent	Absent
Plantar	Mute	Mute
Sensory	Intact	Intact

Primary Management

1) After Neuro opinion, on suspicion of GBS, started on IVIG around 4pm.

2) Around 5pm patient had worsening breathing difficulty with desaturation around 89% RA - started on 2L O2 NP.

3) At 7pm due to further desaturation (80% with O2) and poor respiratory effort, pt electively intubated in ER.

4) Medipack was sent and reports awaited. .

Differential diagnoses

1) GBS

2) Ascending polyneuritis, Miller Fischer syndrome, Stroke

3) Neuro muscular junction disorder like MG, botulism

4) Spinal cord disorder like Transverse Myelitis, compression myelopathy

5) Myositis

6) Metabolic and thyrotoxic myopathies

7) Heavy metal toxicity

8) Infection

9) Hypokalemia related weakness

At 8:30pm pt had a sudden drop in heart rate (HR=30/min),

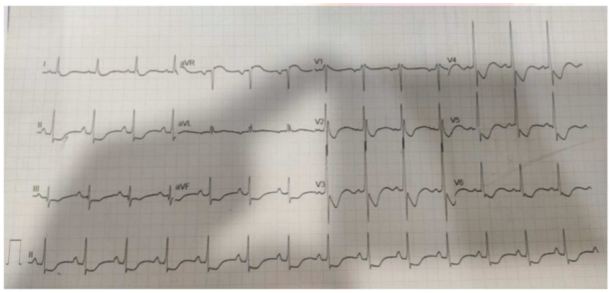
BP=70/50mmHg) - treated with Inj. Atropine 0.6mg IV stat - HR picked up to 86/min.

Immediate ABG and ECG done.

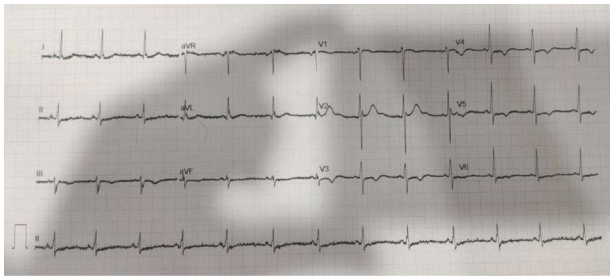
Arterial Blood Gas

PH	7.28
PCO2	24 mmHg
PO2	220 mmHg
Na+	141 mmol/L
K+	0.9 mmol/L
Cl-	113 mmol/L
Lactate	1.4 mmol/L
hco3	11.3 mmol/L
SO2	97%
AG	18

Initial ECG



Post correction ECG



Lab Investigations

Complete Blood Count	
Hb	16.8
WBC	18300
Platelet	209000
ESR	10
Neutrophil	91.4
Lymphocyte	6.1
Monocyte	2.5
Basophil	0

Renal Function Test	
Urea	55.7
Creat	2.03
Sodium	143.4
Potassium	1.4
Chloride	114.9
Bicarb	16
Uric acid	5.96

Urine Routine	
pH	7
Specific gravity	1.01
Glucose	NEG
Protein	TRACE
Blood	+++
Ketone	NEG
Nitrite	NEG
Pus cells	10-12
RBC	2-4
Sodium spot	105
Potassium spot	13.8
Creatinine spot	22.26
Chloride spot	109

Serology: Negative

Nerve Conduction Study

1) Sensory responses are well preserved.

2) Motor responses are reduced in amplitude in upper, lower limbs motor response delay in distal motor latencies with reduction in conduction velocity.

3) Findings point to motor predominant neuropathy mixed axonal and demyelinating features

Secondary management

1) With the clinical picture and labs we have, our DD narrowed down to:

a) GBS
b) Hypokalemia related weakness
c) Stroke
d) Transverse myelitis, compression myelopathy

2) Patient started on Inj. KCl 60 mEq in 100ml NS over 1 hour followed by 60MEq in 500ml RL @150ml/hr.

3) Inj. Sodium Bicarbonate 100ml in 5% Dextrose @300ml/hr.

4) In view of HR=220/min, started on Inj.Esmolol 2mg IV stat followed by 4ml/hr infusion.

5) Pt condition improved the next day morning, hence extubated and weaned off from other supports

Other Investigations

USG Abdomen: Suggestive of medullary nephrocalcinosis.

Repeat NCS: No evidence for generalised peripheral neuropathy.

MRI Brain: No evidence of haemorrhages, infarcts, mass lesion or demyelination seen.

Discussion

Hypokalemia classification and management in ER

Mild: 3-3.4 mmol/L

a) S Oral KCl syrup.
b) S Potassium sparring diuretics if on diuretic therapy.

Moderate: 2.5-2.9 mmol/L

a) Inj.Kcl 20 mEq in 100ml NS or 500ml NS/RL over 4-5 hours.

Severe: <2.5 mmol/L

a) S Inj.Kcl 40mEq in 100ml NS or 500ml NS/RL over 4-5 hr.

How will you manage cardiac/peri arrest patient secondary to severe hypokalemia?

In case of cardiac arrest or life threatening arrhythmia secondary to hypokalemia:

1) S Inj. KCL 20mEq IVI over 20 - 30mins
2) S Repeat dose if needed

Hypokalemic Periodic Paralysis

1) Condition that causes episodes of extreme muscle weakness.

2) Episodes involve temporary inability to move muscles in the arms and legs.

3) Attacks causes severe weakness or paralysis which are reversible with appropiate treatment.

4) Early recognition is vital as hypokalemia can lead to dangerous VT and sudden cardiac arrest

Renal Tubular Acidosis

1) Renal tubular acidosis (RTA) is acidosis and electrolyte disturbances due to impaired renal hydrogen ion excretion (type 1), impaired bicarbonate resorption (type 2), or abnormal aldosterone production or response (type 4). (Type 3 is extremely rare and is not discussed.)

2) ABG shows normal anion gap metabolic acidosis.

3) Patients may be asymptomatic, display symptoms and signs of electrolyte derangements, or progress to chronic kidney disease.

4) Diagnosis is based on characteristic changes in urine pH and electrolytes in response to provocative testing.

5) Treatment corrects pH and electrolyte imbalances using alkaline agents, electrolytes, and rarely drugs.

Type 1 Distal RTA

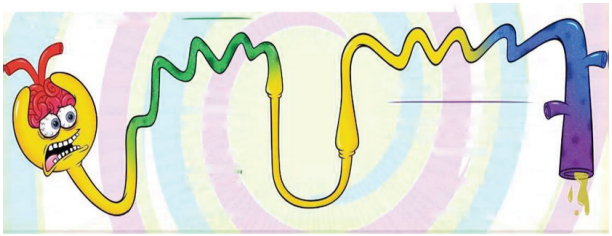
1) Impaired Hydrogen Ion Secretion
2) Urine pH > 5.5
3) Hypokalemia
4) Renal stones

Type 2 Proximal RTA

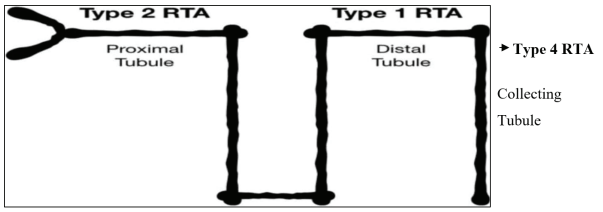
1) Impaired bicarbonate reabsorption
2) High urine ph initially later < 5.5
3) Hypokalemia

Type 4 Hyperkalemic RTA Decreased Aldosterone

1) Secretion or aldosterone
2) Resistance
3) Urine pH < 5.5
4) Hyperkalemia



Classification and Interpretation



	K	Ca	pH	Nephrolithiasis?
2	low	normal	<5.5	No
1 (ODD)	low	high	>5.5	Yes
4	more	normal	<5.5	No

Conclusion

1) Hypokalemia is a commonest electrolyte imbalance we see in our day to day routine in ER.

2) In this patient, the cause of hypokalemia is distal renal tubular acidosis (type-1).

3) Hypokalemic periodic paralysis is not uncommon but easily misdiagnosed.

4) Checking for any reversible causes and treating it first helps both patient and physicians