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An Interesting Case of Fatigue

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Clinical History

A 27-year-old fruit vendor presented to Internal medicine casualty with history of fatiguability for 2 days, acute in onset and gradually progressive that restricted his activities of daily living.

- No h/o fever or night sweats
- No loss of weight or appetite, diarrhoea or vomiting
- No chest pain, dyspnoea on exertion, orthopnoea or PND
- No weakness of limbs/deviation of angle of mouth
- No palpitation or sweating
- No h/o alcohol intake.
- No illicit drug abuse or stressor.

Past History- No DM, Hypertension, TB, Asthma or recent COVID-19 infection

Family History- 4th child of non-consanguineous marriage
No h/o disease that run-in family

Personal History- Mixed diet, normal bowel and bladder
Sleep and appetite normal, occasional alcoholic and smoker

Drug History- No known drug allergies, no h/o drug intake for chronic disease

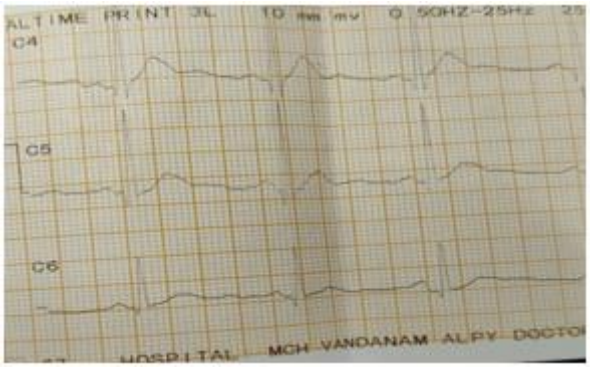
Examination Findings

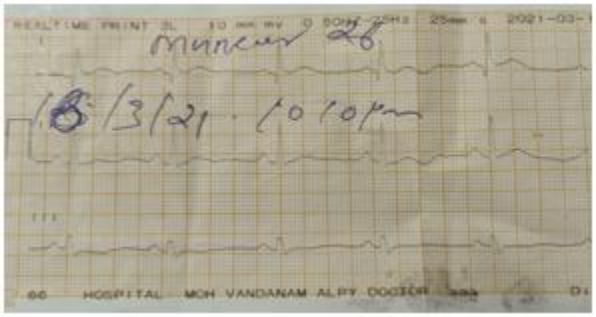
- General examination – Normal
- vitals -stable, a febrile
- Chest-clear, Air entry equal on both sides
- CVS-S1+ S2+ No murmur
- GIT-No organomegaly
- CNS
  - ▶ Higher Functions- NORMAL
  - ▶ Cranial nerves – NORMAL
  - ▶ Motor System
    BULK-NORMAL
    TONE – NORMAL B/L
    POWER-GR 5 IN B/L Upper limb
    GR 5 IN B/L Lower Limb
  - ▶ Reflexes- Superficial and deep all normal
    B/L flexor plantar
  - ▶ Test for coordination- NORMAL
  - ▶ ANS- Normal

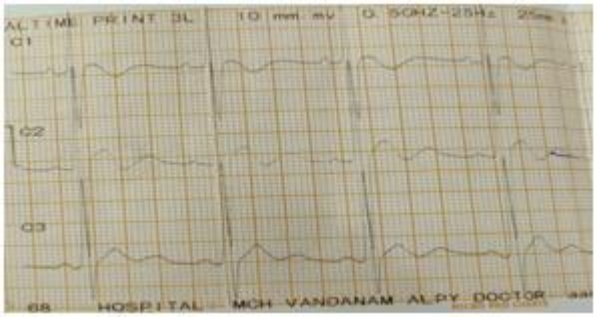
- No signs of meningeal irritation
- H/O AND EXAMINATION WISE EVERYTHING –NORMAL
- ARRANGED FOR ECG AND SENT ALL ROUTINES….

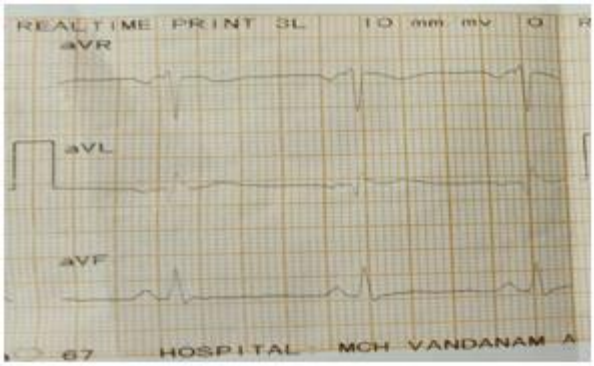
Investigations

Hb-14.5
Tc-8900
Plt-3.5 L
Na/k- 133/2
Ca- 8.7
Mg-1.5
UA-3.5
TSH-4.83,fT3-0.4,fT4-1.1
COVID-19 rtpcr- NEGATIVE
CXR-NORMAL
USG abdomen-Liver – normal echotexture.
Right kidney-10*8*2
Left kidney -10*4*3
CMD maintained, no abnormalities
ECG









Findings

- 1.Presence of U wave.
- 2.Progressive Flattening of T wave.

Provisional Diagnosis

- **Hypokalemia ? Cause**

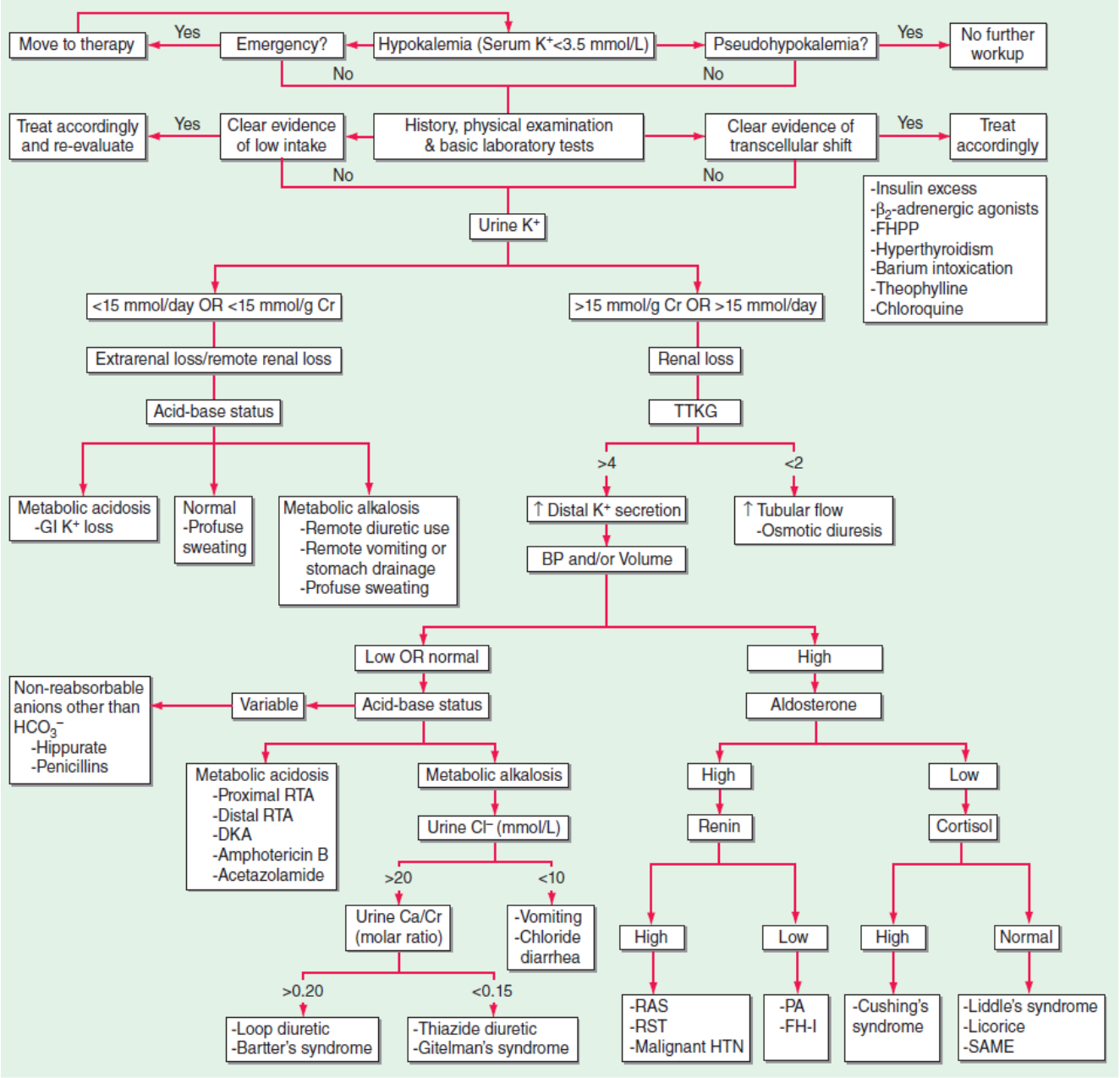
Further Workup

We retook the history.

- Revealedh/o 1 episode of acute onset of weakness of B/L upper limbs in 2016
- Consulted local hospital
- Given some IV fluids
- Symptoms relieved in one day….
- No further workup / No details of treatment available
- Patient also ignored that as there were no further episodes

Hence we planned to evaluate the cause for hypokalemia and hence his fatigue

Diagnostic Approach for Hypokalemia Workup



- 24hr urine K-82.9 meq/day
- TTKG-9.6
- ABG
- Ph-7.49
- Pco2-41
- Po2-36
- Hco3-32
- So2-95
- K-<2

Metabolic alkalosis

- Urine chloride-252meq/l
- Urine calcium-3mg/dl
- Urine creatinine -64 mg/dl
- Urine ca/cr-0.04 (<0.3)

Clinical Clues

- **Hypokalemia**
- **Metabolic Alkalosis**
- **Hypocalciuria**
- **Hypomagnesemia**

Diagnosis

Gitelman Syndrome

Treatment

Patient was started on Syrup potchlor and Inj KCL

Symptoms of patient improved dramatically
Serial measurements of potassium showed ;

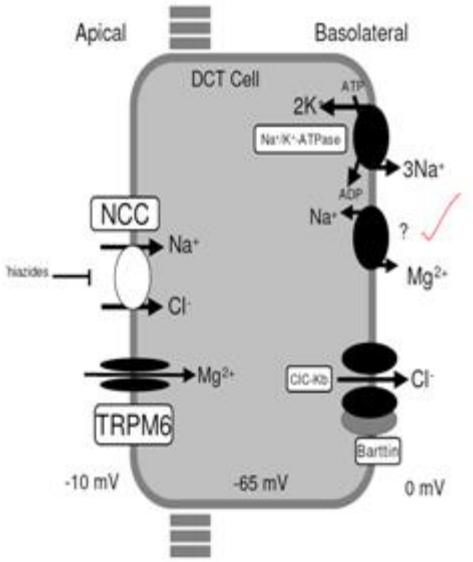
- DAY 1-2
- DAY 2-3.1
- DAY 3-3.1
- DAY 4-3.5
- DAY 5-3.9

Nephrology consultation was sent. Advised to discharge patient on Syrup KMac and to keep patient under follow up.
Patient was counselled and discharged
Came for followup and had K levels corrected and fatiguability improved.

Discussion

- Syndrome first described By American nephrologist **HILLEL.J.GITELMAN** whofirst identified condition in 1966 after observing a pair of sisters
- familial hypokalaemia-hypomagnesemia syndrome
- characterized by hypokalemic metabolic alkalosis in combination with significant hypomagnesemia and low urinary calcium excretion
- Prevalence: 1 in 40,000
- Symptoms do not appear before the age of six years and the disease is usually diagnosed during adolescence or adulthood.
- Follow AR Pattern of inheritance
- caused by mutations in the solute carrier family 12, member 3, ***SLC12A3* gene**, which encodes the renal thiazide-sensitive sodium-chloride cotransporter NCC that is specifically expressed in the apical membrane of cells in the first part of the distal DCT

Pathology



- disruption of NaCl reabsorption in the DCT
- less NaCl is reabsorbed, more sodium reach in the collecting duct resulting in mild volume contraction
- RAAS activated and increasing renin activity and aldosterone levels.
- Elevated aldosterone levels give rise to increased electrogenic sodium reabsorption in the cortical CD *via* the ENaC.
- increased secretion of potassium and hydrogen ions, thus resulting in hypokalemia and metabolic alkalosis.
- passive Ca2+ reabsorption in the proximal tubule and reduced abundance of the epithelial Mg2+ channel TRPM6, located in the DCT explains thiazide-induced hypocalciuria and hypomagnesemia
- thiazides are known to inhibit NCC, and there is phenotypic resemblance between GS and chronic thiazide-treatment

Presentation

- usually present above six years of age and in many cases the diagnosis is only made at adult age
- *tetany*, especially during periods of fever or when extra magnesium is lost due to vomiting or diarrhea.
- *Paresthesias*, especially in the face
- experience *severe fatigue* interfering with daily activities, while others never complain of tiredness
- suffer from *chondrocalcinosis*, which is assumed to result from chronic hypomagnesemia.
- It causes swelling, local heat, and tenderness over the affected joints.
- symptoms, such as *ataxia, vertigo, and blurred vision* have been reported

Differential Diagnosis

1) Type 3 Bartter syndrome (CLCNKB mutation)
2) Primary renal hypomagnesemia
3) Chronic thiazide use
4) Chronic laxative abuse or chronic vomiting

Prognosis

- Excellent long-term prognosis.
- severity of fatigue may seriously hamper some patients in their daily activities
- Progression to renal insufficiency is extremely rare in GS
- One patient who developed chronic renal insufficiency and subsequent progression to ESRD has been reported in literature.

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