Severe hypokalemia as a rare presentation of Disseminated Tuberculosis

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Abstract

Disseminated tuberculosis (TB) results from a massive lymphohematogenous dissemination of Mycobacterium tuberculosis**.** We report a 38-year-old patient who presented with fever , severe weakness for 5 days , his initial workup in the Emergency showed severe hypokalemia resistant to multiple doses of potassium replacement , later the patient showed feature of meningitis , his CSF analysis confirmed Tuberculous meningitis , after starting on Anti tuberculous medications, his hypokalemia improved significantly. Later sputum culture came positive for TB and the patient labelled as Disseminated Tuberculosis. Despite Severe Hypokalemia being a rare association with Tuberculosis, Disseminated Tuberculosis can be considered as the possible cause in patient presenting with severe hypokalemia and fever. Renal tubular defect could be the proposed mechanism causing potassium losing nephropathy.

Keywords: TB, Hypokalemia, Renal tubular defect, Bartter.

Introduction:

Disseminated Tuberculosis (TB) results from a massive lymphohematogenous dissemination of Mycobacterium tuberculosis bacilli. Clinical manifestations of Disseminated TB are most likely to be subacute or chronic, less commonly, acute presentations also occur {1, 2}. Patients with subacute or chronic may present with fever of unknown origin, night sweats, fever, rigor and / or dysfunction of one or more organ. Acute Presentation, including multisystem failure, septic shock and Acute Respiratory Distress Syndrome {3}.

Severe Hypokalemia as presenting feature of Disseminated or Miliary Tuberculosis is a rare entity, and the association between Disseminated Tuberculosis and hypokalemia is poorly

understood. To highlight this rare association and to explain the possible association between these two clinical conditions, we report a case of a 38-year-old male patient who present with weakness Found to have severe hypokalemia and further investigations revealed Disseminated Tuberculosis.

Case Report:

38-year-old patient previously healthy presented with generalized body weakness for 5 days Described as general weakness more in the lower limbs associated with generalized fatigability, he denied any muscle pain, specific proximal or distal distribution, or any variation during the day . Over the next 5 days his weakness was progressively increasing, to extent he had difficulty walking leading to a fall. This weakness was associated with on and off fever, occasional dry cough and 2 episodes of vomiting. Patient had polyurea, polydipsia and nocturia for 2 days which were first time to develop such complains.

Patient denied having headache, photophobia, phonophobia, neck pain or stiffness, abnormal jerky movements, palpitations, sweating, weight loss, shortness of breath, sputum production or dysuria, as well as no history of diarrhea or poor dietary intake. Patient is doesn't smoke, drink alcohol or use illicit drugs. He is not sexually active. Works as a salesman. He denied sick contacts or recent travel.

Upon presentation to the hospital ED patient was febrile but other vitals were stable. He needed support to walk to bathroom. Upon exam his lower limbs power was 4/5 that improved to 5/5 after IV potassium infusion temporarily. Meningeal signs were negative, and the rest of physical exam was unremarkable. Initial labs showed severe hypokalemia of 2.1mEq/l (Normal 3.5-5 mEq/l) and hyponatremia of 127 (Normal 135-145mEq/l) , creatinine 73 umol/L (normal 62-106 umol/L) , chloride 83 mmol/l (normal 90-105 mmol/l) , bicarbonate 29 mmol/l (normal 22-29mmol/l) , corrected Ca 2.54mmol/L (normal 2.2-2.55mmol/L) , glucose 7.9mmol/L (3.5-5.5mmol) repeated random glucose readings were normal ,HBa1c 5.6% (normal < 6.5 %), Lactic acid 2.0 (Normal < 2), Hb 13.5 gm/dl (normal 13-17 gm/dl) , WBC 19.10 x10^3/uL ((normal 4-10 x10^3/uL), HIV negative, urine dipstick unremarkable. ECG showed regular sinus rhythm, T wave inversion in inferolateral leads, U wave and epsilon wave. The patient was admitted as a case of severe symptomatic hypokalemia and fever for workup and management.

His 24hr urine potassium after 48 hr of admission was 156 mmol/24hrs in 4.585 L of urine (normal 25-125 mmol/24hr), IV potassium replacement was started, Patient received a total of 110 mEq IV and 60 mEq Oral in the first 24 hours of his hospital stay, his potassium remained low at 2.5 mEq/L (normal 3.5 -5 mEq/L). Patient was kept on continuous IV potassium replacement (4 mmol/hr) in addition to 30 mEq oral q8hrs over the next 2 days. His potassium improved to 3.9 mEq/L and the weakness disappeared completely.

Sepsis screen came unremarkable including 2 sets of blood cultures, urine culture, chest X ray and abdominal ultrasound, Patient was started on ceftriaxone q24hr initially but after 48 hrs of hospital stay he was still having fever and he reported new neck pain, upon exam found to have neck stiffness, positive kerning's and brudzenski signs, new bilateral lower limb weakness (power 3/5) and urine retention. Lower limb weakness (power 3/5) and urine retention. LP showed CSF protein 5.7 gm/L (normal 0.15-0.45gm/L), CSF Glucose 0.97mmol/l (normal 2.2-3.8 mmol/l), CSF WBC 484 /uL (normal 0-5/ul) (78% Lymphocytes, 16% neutrophils). Ceftriaxone changed to q12hr and started on anti TB treatment with prednisolone 60mg and pyridoxine, then CSF TB PCR came positive, so ceftriaxone was discontinued and continued on Anti TB treatment. Sputum for TB Afb smear, PCR and culture was induced with hypertonic saline and sent and Repeated Lumber Punctures after 3 days showed improvement with CSF protein decreased to 1.22gm/L, CSF glucose increased to 1.62 mmol/l.MRI brain showed features highly suggestive of tuberculous meningitis involving the brain and spinal canal with small right frontal as well as left cerebellar tuberculoma. Mild supratentorial hydrocephalus.

Patient's condition improved and became afebrile but with remnant lower limb weakness and urine retention. Transferred to rehabilitation medicine on day 17 of admission. Potassium supplements were stopped on day 13 of hospital stay. 4 weeks from admission his sputum TB culture came back positive (rifampicin and INH sensitive) and patient was labelled as Disseminated Tuberculosis. Patient continued treatment in rehabilitation hospital 5 weeks then discharged on anti TB treatment with Direct Observation Therapy and ID follow up. His potassium remained normal in the whole period of rehabilitation and didn't require any supplements.

Discussion

Disseminated tuberculosis (TB) results from a massive lymphohematogenous dissemination of Mycobacterium tuberculosis. Electrolytes disturbances could be associated with Tuberculosis.

Hyponatremia is frequently observed and reported; it is presumed to be due to disregulation of antidiuretic hormone (SIADH) that causing more water retention with excessive sodium excretion from the body in the urine {4}.

Hypokalemia is one of the adverse reactions commonly found in patients with multi-drugs resistant tuberculosis (MDR-TB) who are treated with tablets or injectable agents. Rifampicin, Amikacin, viomycin-pyrazinamide all are mentioned in the literature to be associated with electrolytes imbalance including hypokalemia {5-8}.

Hypokalemic periodic paralysis also considered as one of the well-known causes of hypokalemia which has an autosomal dominant mode of inheritance, or it can be acquired in patients with hyperthyroidism. Acute attacks which cause sudden movement of potassium into the cells are often are often precipitated by rest after exercise, stress, or a carbohydrate meal. In our patient infection with TB could be the stressful trigger causing hypokalemia.

Hypokalemia as a rare association with pulmonary Tb. One case report mentioned the association of Bartter's syndrome with pulmonary tuberculosis in a middle age lady who presented with polyuria and frequency, wasting syndrome and cramps. Laboratory evaluation showed leukocytosis (12.100/mm³ normal), hyponatremia (123 mEq/L), severe hypokalemia (1.34 mEq/L), BUN (67 mg/dL), metabolic alkalosis pH=7.40, $HCO3^{-}$= 30 moll/L,. A 24-hour urine collection (3500 ml) revealed potassium level of 40 mEq/L, the patient later was treated with regular therapy with oral potassium 15g/day for almost 5 years without significant improvement in her symptoms, The patient was admitted again with fever, chills, cough, wasting syndrome, polyuria and polydipsia found to have severe hypokalemia and further evaluation showed, Surprisingly, CXR showed opacity in her upper left pulmonary lobe and pulmonary tuberculosis was also confirmed by positive smears and cultures. The authors in this case assumed that tubulopathies caused by primary infection can explain the unusual association between Barter syndrome and pulmonary Tuberculosis. {9}

hypomagnesemia contributing to hypocalcemia and hypokalemia, occurring as a primary

Abnormality in patients with pulmonary tuberculosis was reported by Baskaran S. *et. al.* which was worsened by the use of streptomycin he mentioned that hypomagnesaemia is multifactorial in origin including malnutrition, malabsorption, and therapy induced renal loss. (10).

To the best of our knowledge this is the first case report on the association between Disseminated TB and severe hypokalemia. We reemphasized the assumption that Tuberculosis which is in our case disseminated type can lead to tubular defect which can cause potassium losing nephropathy. In the future further studies may highlight the exact mechanism.

Conclusion

Despite Severe Hypokalemia being a rare association with Tuberculosis, Disseminated tuberculosis can be considered as the possible cause in patient presenting with severe hypokalemia and fever. Renal tubular defect could be the proposed mechanism causing potassium losing nephropathy.

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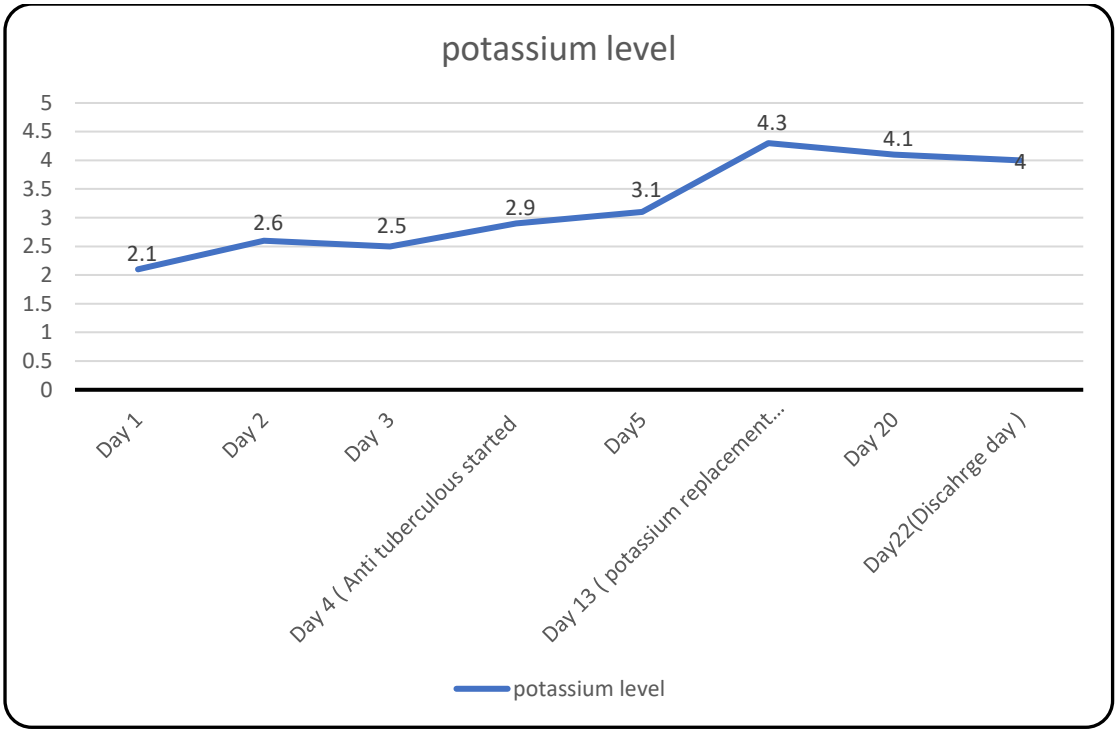

Figure 1: correlation between Potassium level and treatment of the patient.