Hypokalemic Paralysis in Leptospirosis

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Case Report

A 40 year old male farmer presented to us with fever and body ache of 1 week, progressive motor weakness of all 4 limbs and difficulty in walking of 4 days duration. There was no history suggestive of sensory, bladder or bowel involvement.

On examination patient was febrile and had stable vital signs. He was icteric, had pericorneal conjunctival congestion and severe calf and quadriceps tenderness. There was no facial puffiness, pedal oedema or an elevated jugular venous pressure. The central nervous system examination revealed a flaccid quadriparesis with greater involvement of the proximal muscles of the lower limb (Lower upper limb grade 3/5, lower limb grade 2/5) He also had a hepatomegaly but no splenomegaly or ascites. All other systems were within normal limits.

Investigations on admission

Hb- 11.0 g/dL: Total count – 18,860, Platelet count – 1,00,000/mm3, FBS – 92 mg/dL, PPBS – 134 mg/dL, T.Bili – 4.5 mg/dL, D.Bilirubin – 2.5 mg/dL, AST – 309 u/l, ALT – 283 u/l, urea – 56 mg/dL, S. creatinine – 2.9 mg/dL, Na – 139 meq/L, S Potassium – 1.7 meq/L, HCO3 – 24 meq/L, ABG – PH – 7.45, PaO2 – 94 mmHg, PaCO2 – 44 mmg, HCO3 – 26 meq/L.

Urine analysis – pH – 5.3, alb – Nil, WBC – 1-2/Hpf, occasional hyaline casts. 24 hour urine potassium was 85 m.eq/day (Normal < 20 m.eq/day)

Leptospiral Antibody(IgM ELISA) - Negative (< 0.9 Index value) on admission, but was strongly positive (>1.3) on 12th day of admission. (>1.1 index value is positive). Viral markers for hepatitis were negative. ECG– flattening of ‘T’ waves; ‘U’ waves present.

Discussion

Leptospirosis, a spirochetal infection, is one of the most widespread zoonosis. It is highly prevalent in India, especially during rainy season. The clinical presentation can vary from subclinical infection and self limited anicteric febrile illness to severe and fatal disease. Leptospirosis can affect the kidney by different mechanisms. Interstitial nephritis and tubular necrosis are common renal lesions seen. Acute renal failure associated with leptospirosis is often non-oliguric with high fractional urinary excretion of potassium and hypokalemia.1 Acute tubular necrosis with oliguric renal failure can also occur;2 usually secondary to hypotension and shock, release of endotoxins and pro-inflammatory cytokines such as TNF-α, disseminated intravascular coagulation, intravascular hemolysis and rarely hemolytic uremic syndrome. Immune mediated glomerulonephritis and drug induced interstitial nephritis may also cause renal failure. This case had a non-oliguric renal failure with marked hypokalemia which led to hyporeflexic paralysis.

Hypokalemia caused by kaliuresis is noted in 26% - 40% of patients with leptospirosis. If significant, it can lead to muscular weakness. The outer membrane proteins of leptospira inhibit Na-K ATPase. This causes an increase in intra-cellular Na resulting in decreased Na transport at the luminal border of all parts of renal tubules. The resulting increase in Na delivery to the distal nephrons for Na-K exchange causes kaliuresis. Increased plasma aldosterone and cortisol levels further enhance K excretion.

Anand Krishnan et al reported a case of paralysis due to renal potassium wasting.7 That patient too was managed conservatively and his paralysis improved following correction of hypokalemia and resolution of the disease. Lin CL et all from...
Taiwan reported a case of leptospirosis induced non-oliguric acute renal failure who developed hypokalemia during the recovery phase. This patient too had inappropriately high urinary potassium excretion in the presence of hypokalemia, suggesting that renal potassium wasting played a role in the pathogenesis.

Paraparesis may also occur due to leptospirosis independent of electrolyte abnormality. A case of sudden onset paraparesis with acute renal failure in a patient with leptospirosis was reported by Ramakrishna et al. Although the patient had hyperkalemia initially, the paraparesis did not improve following the correction of the hyperkalemia. Guillain-Barre syndrome with paraparesis can rarely occur following infection with leptospira.

We report this as a case of reversible flaccid weakness in leptospirosis secondary to hypokalemia caused by renal potassium wasting which improved following potassium supplementation.

References