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ACUTE ASCENDING HYPOKALAEMIC PARALYSIS

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CASE DESCRIPTION

First patient A fifty-year-old male presented with weakness, difficulty in talking and breathing along with ascending paralysis. He gave prior history of excessive sweating in hot weather and had been administered many intravenous drips of dextrose solutions over the last three days. On clinical examination he was fully conscious, but was restless and tachycardiac. Motor power was 1/5, there was no obvious muscle wasting or fasciculation and the tendon reflexes along with abdominals and plantars were absent. Fundoscopy was unremarkable. Blood biochemistry revealed marked hypokalemia of 2.2 mmol along with an elevated blood and CSF sugar. Electrocardiography revealed flattened T waves and non-specific ST changes. His ventilation was supported and he was given I/V fluids with potassium supplementation and hyperglycaemia was managed with appropriate doses of insulin. The patient rapidly improved over the following few days with serum levels of potassium and ECG changes reverting to normal. The patient was discharged home in an ambulant state and he did not report back with any recurrence of symptoms

Second patient was a fifty-year-old male presented with a few days history of profuse diarrhoea and vomiting. Progressive ascending weakness and difficulty in breathing followed this. On examination he was also fully conscious with no obvious muscle wasting or fasciculation and the tendon jerks were not elicitable. His ECG showed flat T waves. Blood biochemistry reveled hypokalemia with a serum potassium level of 2.3 mmol. His ventilation was supported and I/V fluid with supplemental potassium were administered along with parenteral antibiotics for fever and diarrhoea. He made a quick recovery over the next couple of days and was discharged in less than a week's time. No recurrence of the symptoms occurred over a follow up period of three years.

Third patient was a twenty seven year old soldier who was brought to the Hospital while he was being ventilated with an Ambu bag. He also had history of prior vomiting and diarrhoea for the last five days and over the last twenty four hours had developed difficulty in talking and breathing along with rapid ascending paralysis. On examination he also had no motor wasting with absent tendon jerks. He was also febrile. ECG was suggestive of hypokalemia because of T wave flattening but serum potassium was 4 mmol/L. Chest X ray also revealed right basal collapse and consolidation. He was also placed on a ventilator and supplemental potassium was administered along with parenteral antibiotics and other ancillary supportive treatment. He made a rapid recovery, and has had no recurrences over the last three years.

DISCUSSION

Hypokalaemia is caused by a variety of disorders, but acute weakness has been reported with some and not all the causes of hypokalaemia. Various causes of hypokalemia can be divided into two broad groups. First group would comprise of entities resulting in transcellular potassium shift without actual potassium depletion. The important conditions in this group include familial periodic paralysis, thyrotoxic periodic paralysis and barium poisoning. Second group of conditions resulting in actual potassium depletion can be divided into

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entities where there is renal loss like RTAs, Sjögren's syndrome, medullary sponge kidney, chronic toluene exposure, Fanconi's syndrome, primary hyperaldosteronism, licorice ingestion, water intoxication, Nephrotic syndrome, diuretic phase of acute tubular necrosis, Barter's syndrome, treatment phase of diabetic ketoacidosis, chlorthiazide associated hypokalaemia and hypokalaemia following uretero-sigmoidostomy. Another group would consist of conditions with extrarenal loss of potassium like celiac disease, tropical sprue, acute gastroenteritis and short bowel syndrome [1,2,3] .In our cases potassium loss was aggravated by injudicious use of potassium free fluid replacement in subjects who were also losing electrolytes due to sweating in the hot and humid weather.

The most prominent clinical features of hypokalaemia or potassium depletion are neuromuscular, although other systems, such as cardiovascular and gastrointestinal, may also be affected. Some patients complain of muscular weakness, especially of the lower extremities, while marked and generalised weakness of skeletal muscles is common with more severe potassium depletion. Very severe hypokalaemia may lead to virtually total paralysis including respiratory, bulbar and cranial musculature. Deaths from respiratory failure and arrhythmia have been reported. On physical examination, in addition to decreased motor power, the patient may demonstrate decreased or absent tendon reflexes. The sensations and level of consciousness are generally unaffected.

The cardinal laboratory manifestation is serum potassium of less than 3.5 mmol/l during an attack, although it is usually much lower. Abnormalities in electrocardiogram (ECG) are common. The typical changes include flattening and inversion of T waves, appearance of U waves and ST segment sagging. ECG changes are, however, not well correlated with the severity of the disturbances in potassium metabolism. Symptomatology results from the increased ratio between intra- and extra cellular potassium concentrations, which modifies membrane polarization and thereby alters the function of excitable tissues such as nerve and muscle. Diagnosis of hypokalaemic paralysis should be considered in any patient presenting with a sudden onset, areflexic, pure motor weakness involving one or more limbs, without alteration in the level of consciousness or sphincter function, and laboratory evidence of hypokalaemia.

Management of hypokalaemia [1,4,5,6] is almost always by potassium replacement, with the amount of potassium supplementation depending on the severity of hypokalaemia. Potassium can be given orally in mild to moderate hypokalaemia and intravenously in severe hypokalaemia. When given intravenously the rate of potassium administration should not exceed 20 mmol/hour. To calculate the amount of potassium supplementation one should have an estimate of potassium deficit. On average a reduction of serum potassium by 0.3 mmol/L suggests a total body deficit of 100mmol/L. Based on this formula, a patient with serum potassium of 2.6 mmol/L needs atleast 300mmol of potassium. Potassium phosphate can be used in patients with combined potassium and phosphate depletion (e.g. in patients with cirrhosis of liver and diabetic ketoacidosis). Potassium bicarbonate in patients with hypokalaemia and metabolic acidosis (e.g. in renal tubular acidosis).

Usually 50-100 mmol of potassium chloride is required per day to maintain serum potassium with in normal range in patients with an increased potassium loss (that is in patients receiving diuretics).

Monitoring of clinical improvement by estimation of biochemical parameters frequently and repeated ECG's which should return to normality, is essential to the management. In addition the cardiac rhythm

should be continuously monitored during the above treatment.

CONCLUSION

In these patients severe hypokalaemia developed after few days of mild to moderate diarrhoea and reduced oral intake followed by injudicious use of intravenous fluids. In one patient it was the result of injudicious use of intravenous fluids following excessive sweating in the hot weather of that desert area. This resulted in life threatening hypokalaemia causing ascending paralysis, which was initially confused with Guillain Barre Syndrome. So it is essential to consider hypokalaemia in the differential diagnosis of ascending paralysis as its prompt diagnosis and appropriate treatment can result in dramatic improvement over a short span of time and save precious human lives.

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