

Metabolic Stridor: A Case Report

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ABSTRACT

The clinical features of severe hypokalaemia include constipation, ileus, ventricular and atrial tachycardias, weakness, hypotonicity and rarely an ascending motor paralysis with ventilatory failure. We describe a patient who presented with severe diarrhoea, hypokalaemic normal anion gap metabolic acidosis and hypophosphataemia. He developed stridor and respiratory failure following early correction of his hypokalaemia which was successfully managed with endotracheal intubation, mechanical ventilation and further correction of his fluid and electrolyte abnormalities. (Critical Care and Resuscitation 2004; 6: 277-279)

Key words: Hypokalemia, hypophosphataemia, skeletal muscle weakness, stridor

Stridor and respiratory distress due to hypokalemia or hypophosphatemia is a rare clinical feature. We present a case of an intravenous drug user who was being treated for profound diarrhoea complicated by severe hypokalemia, hypophosphataemia and hyperchloremic metabolic acidosis. Despite treatment he developed progressive stridor and respiratory distress requiring intubation and mechanical ventilation.

CASE REPORT

A 35 year old Caucasian male was admitted to the emergency department with a six week history of diarrhoea. The volume and frequency of the diarrhoea had increased over the preceding three days and was associated with vomiting and increasing fatigue. The stools were watery and offensive with no visible blood or mucous. He had lost 20 kg in weight over the past two months. His social history revealed that while he had a stable homosexual relationship he had been an intravenous drug user. He had been treated for schizophrenia and depression and had attempted suicide on several occasions.

On examination he was conscious and orientated. His heart rate was 70 beats per minute, blood pressure was 120/70mmHg and he was afebrile. Clinical examination of the abdomen revealed minimal hepatomegaly and apart from a subtle hand tremor and generalised weakness the examination of the other major systems revealed no abnormality.

Laboratory investigations revealed a haemoglobin of

117 g/L, white cell count of $9.2 \times 10^9/L$ and a platelet count of $365 \times 10^9/L$. The plasma urea was 10.1 mmol/L, creatinine 0.17 mmol/L, sodium 132 mmol/L and potassium 2.3 mmol/L. The plasma liver function tests were within normal limits. His electrocardiogram demonstrated U waves which were predominant in the precordial leads.

He was admitted to the general medical ward with a provisional diagnosis of profound diarrhoea with dehydration causing severe hypokalemia and prerenal azotaemia. However, the profuse diarrhoea continued despite being treated with intravenous potassium chloride and fluids. A plasma potassium measured within a few hours of admission was 2.3 mmol/L. Viral serology for hepatitis A, B and C were negative and HIV antibodies were not detected. The thyroid function tests and plasma lactate levels were within normal limits. Stool microscopy revealed a few polymorphs although there was no pus, blood or parasites noted and cultures for enteral pathogens were repeatedly negative. Stool rotavirus antigen analysis was negative as was Clostridium difficile toxin.

After 24 hours, his condition deteriorated. He became lethargic and hypotonic with profound weakness. He was unable to move his limbs or raise his head from the bed and was experiencing difficulty in speaking, although he remained conscious and alert. Plasma biochemistry revealed a potassium of 1.7 mmol/L (despite intravenous supplementation) and a phosphate of 0.3 mmol/L. He was subsequently

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transferred to the intensive care unit. The ECG revealed sinus rhythm of 55 beats per minute with generalised T wave flattening and precordial U waves. Plasma biochemistry revealed a sodium of 145 mmol/L, potassium 1.7 mmol/L, bicarbonate 8 mmol/L, chloride 129 mmol/L and anion gap of 8.7 mEq/L, reflecting a hypokalemic normal anion gap metabolic acidosis. His other plasma biochemical results included a urea of 7.7 mmol/L, creatinine 0.10 mmol/L, phosphate 0.2 mmol/L, magnesium 1.05 mmol/L and corrected plasma calcium of 2.34 mmol/L.

A central venous catheter was inserted to facilitate rapid correction of his hypokalaemia, however the patient subsequently developed worsening stridor and respiratory failure requiring intubation and mechanical ventilation. Despite correcting his potassium and phosphate levels his weakness persisted. In light of the persistent muscle weakness the possibility of myasthenia gravis was also considered. Both the edrophonium test and acetylcholine receptor antibodies were negative.

Early attempts at weaning failed and after 10 days a percutaneous tracheostomy was performed. A slow respiratory wean was successful and his condition stabilised. His early days in intensive care were complicated by a *Staphylococcus aureus* pneumonia and his final few days were complicated by a gastrointestinal haemorrhage secondary to gastric ulceration. The latter required a laparotomy from which he made a rapid recovery and he was transferred to the general medical ward a few days following his surgery.

DISCUSSION

Stridor is an auditory manifestation of disordered respiratory function due to air flow changes in the larynx, trachea or bronchi secondary to partial obstruction.¹ Stridor arising from obstruction to air flow through the larger airways can be a complication of either a localised obstruction such as a foreign body or neoplasm, or it can be the sequela of a more generalised disorder such as tetany or some other metabolic disturbance causing laryngospasm.² Potassium is the most abundant intracellular cation and is a major determinant of intracellular osmolality. Only 2% is extracellular and most intracellular potassium is contained within the skeletal muscle. Intracellular potassium has a central role in the excitability of nerve and muscle. The resting potential, generation of action potential, repolarisation and endplate potentials in both nerve and muscle are all dependant on intracellular potassium concentrations. Profound hypokalemia usually causes muscle weakness of the trunk and/or extremities.³ Severe hypokalemia can cause respiratory failure.

The blood and urine electrolytes along with the arterial blood acid base parameters can provide valuable

clues in simplifying the differential diagnoses of hypokalemia.⁴ Hypokalemia with a metabolic alkalosis may be due to vomiting, excess nasogastric fluid loss, hyperaldosteronism, Bartter's syndrome,⁵ Gitelman's variant,^{6,7} Liddle's syndrome (pseudoaldosteronism),⁸ Cushing's syndrome, ectopic corticotropin producing tumours, VIPoma, mineralocorticoid excess, excess liquorice intake and diuretics. Hypokalaemic metabolic acidosis may be associated with a normal or raised anion gap. Normal anion gap metabolic acidosis can again be classified into low and high NH₄ excretion states. The former include distal and proximal renal tubular acidosis. The causes with high NH₄ excretion include toluene abuse, diarrhoea and ureteral diversions. Hypokalaemia with a raised anion gap metabolic acidosis may be seen with diabetic ketoacidosis. Hypokalemia without total body potassium depletion is a complication of hypokalemic periodic paralysis, beta₂ agonist administration or insulin and glucose infusions. Hypokalemia without an acid-base disturbances may also be encountered after an osmotic diuresis, penicillin or theophylline administration, hypomagnesaemia or a low dietary intake.

Phosphorus is an essential component of phospholipids, nucleic acids and has an essential role in energy metabolism. Approximately 1% of the total body phosphorus is extracellular and the major intracellular store is in bone and intracellular compartments. The ionised phosphorus is the physiologically active form which constitutes about 55% of the plasma concentration. Serum phosphorus is a poor indicator of total body phosphorus in view of rapid intra and extracellular shifts. There is also a poor correlation between plasma phosphate levels and symptoms. Severe hypophosphatemia can cause life threatening complications including myocardial depression, weakness, rhabdomyolysis, respiratory failure, confusion, coma, stupor, seizures, haemolysis and platelet and leucocyte dysfunction.

Sarkar reported a case of stridor due to laryngospasm caused by a drug induced hypokalemic alkalosis.² Moralee *et al*, described a case of bilateral abductor palsy and stridor in an 89 year-old lady with drug induced hypokalemia which responded to correction of the plasma potassium.⁹ Dunn *et al*, reported a case of hypokalemic hyperchloremic metabolic acidosis requiring intermittent positive pressure ventilation in a patient as a complication of combined colonic bladder augmentation and incomplete voiding via a prosthetic sphincter.¹⁰

We believe that, early and aggressive correction of the hypokalemia might have prevented the occurrence of stridor and respiratory failure in this patient, though he had persistent muscle weakness even after the correction of his low plasma potassium level.

Received 13 August 04

Accepted 31 August 04

REFERENCES

1. Cinnamond MJ. Stridor. In Scott- Brown's otolaryngology. eds. Evans JNG, Kerr AG, Butterworths: London 1987; 6:420.
2. Sarkar SK. Stridor due to drug-induced hypokalaemic alkalosis. *J Laryngol Otol* 1987;101:197-198.
3. Young,R., Bradly,W., Adams,R. Approach to clinical myology. In Harisson's Principles of Internal Medicine 10th edition. eds. Petersdof,R., Adams R, Braunwald E, Isselbacher K, Wilson J: Mc Graw-Hill. 1983 p.2171-2173.
4. Lin SH, Lin YF, Halperin ML. Hypokalemia and paralysis. *Q J Med* 2001;94:133-139.
5. Bartter PC, Pronove P, Gill JR Jr, MacCardle RC. Hyperplasia of the juxtaglomerular complex with hyperaldosteronism and hypokalemic alkalosis: a new syndrome. *Am J Med* 1962;33:811-828.
6. Gitelman HJ, Graham JB, Welt LG. A new familial disorder characterized by hypokalemia and hypomagnesemia. *Trans Assoc Am Physicians* 1966;79:221-235.
7. Scheinman SJ, Guay-Woodford LM, Thakker RV, Warnock DG. Genetic disorders of renal electrolyte transport. *N Engl J Med* 1999;340:1177-1187.
8. Gadallah MF, Abreo K, Work J. Liddle's syndrome, an underrecognised entity: a report of four cases, including the first report in black individuals. *Am J Kidney Dis* 1995;25:829-835.
9. Moralee SJ, Reilly PG. Metabolic stridor: bilatetral vocal cord abductor paralysis due to hypokalemia? *J Laryngol Otol* 1992;106:56-57.
10. Dunn SR, Farnsworth TA, Karunaratne WU. Hypokalemic, hyperchloraemic metabolic acidosis requiring ventilation. *Anesthesia* 1999;54:566-568.