JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH

How to cite this article:

ABBAS MT , KHAN FY,BAIDAA A,ERRAYES M, HALEEM AL H AL. RESPIRATORY FAILURE SECONDARY TO OBSTRUCTIVE UROPATHY Journal of Clinical and Diagnostic Research [serial online] 2008 October [cited: 2008 October 6]; 2:1107-1109.
Available from
http://www.jcdr.net/back_issues.asp?issn=0973-709x&year=2008&month= October &volume=2&issue=5&page=1107-1109 &id=269

CASE REPORT

Respiratory Failure Secondary To Obstructive Uropathy

ABBAS MT *, KHANFY,BAIDAA A***,ERRAYES M****, HALEEM AL H AL*******

ABSTRACT

A 34 year-old male patient who presented for the first time with quadriparesis and type 2 respiratory failure, was found to have severe hypokalaemia (1.1 mmol/L) associated with renal failure, normal anion gap metabolic acidosis and respiratory acidosis .Imaging studies showed left hydronephrosis secondary to urinary bladder neck obstruction. Diagnosis of respiratory failure due to hypokalaemia, which resulted from obstructive uropathy, was established.
After relief from obstruction and administration of intravenous fluid and oral potassium supplement, his renal function and his serum potassium level was normalized, and his condition improved. On one of the following days, the patient was discharged.

Key Words: hypokalaemic paralysis, respiratory failure, obstructive uropathy.

*(MD) (MRCP),** (MD),*** (MD), ****,
*****Hamad General Hospital,P O Box 3050 Medical Deptt.Doha-Qatar
Corresponding Author
Dr. MTAbbas (MD) (MRCP)Senior Specialist Deptt. of Medicine,Hamad general Hospital
PO Box 3050, Doha-Qatar
Tel: 009745220486,Fax 009744392273
E Mail: amushtak@hotmail.com

Introduction

Hypokalemaic paralysis is a medical emergency due to the risks of cardiac arrhythmia, respiratory failure, and rhabdomyolysis. Besides supplementing patients with potassium to hasten recovery, the astute physician must search for the underlying cause, to avoid missing a treatable and curable disorder. We report a 34 year old patient who presented with quadriplegia and then passed into respiratory failure secondary to hypokalaemic paralysis, which proved to be secondary to obstructive uropathy.

Case Report

A 34- year old Sri Lankan patient, not known to have any chronic disease before, presented to the Accident and Emergency Department of our hospital because of the inability to move his lower and upper limbs for one day . The patient admitted that he had difficulty in passing urine since the past one month, and had to strain to pass urine. He also had on and off vomiting for one month, which increased 2 days before admission. He denied fever, palpitation, previous similar attack, drug intake, or a family history of similar conditions. Upon examination, the patient was conscious, oriented, dehydrated, with a blood pressure of 102/62, a pulse rate of 80/minute, temperature of 37.2ºC and a respiratory rate of 26/minute. His chest, heart and abdomen were normal, apart from a distended urinary bladder. He had power grades, one in both upper and lower limbs, his reflexes were normal, and his planters were down going. There was no sensory deficit and his cranial nerves and ocular muscles were intact.

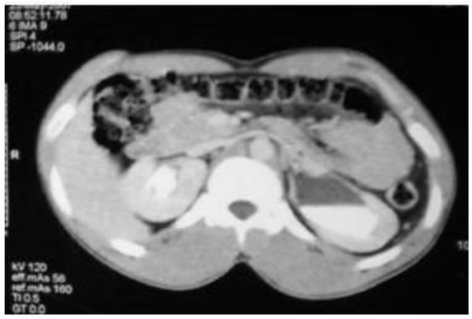
His investigations showed white blood cells: 18700/µL, haemoglobin: 12.2g/dl and platelets:368000/µL. Biochemistry showed potassium: 1.1 mmol/L, calcium: 1.8mmol/l, chloride: 94mmo/l, glucose: 6.6 mmol/l, creatinine: 523 µmol/L, urea: 26.7 mmol/l, bicarbonate: 15 mmol/l, sodium: 120 mmol/l, magnesium: 1.14 mmol/L, phosphorus: 1.37 mmol/L. Serum

osmolality was 263 Os/kg, urine osmolality was 315 Os/Kg , myoglobin was 3308ng/ml, SGOT was 74U/L, , lactic acid was 1.19 mmol/L (0.5-2.2) and lactate dehdrogenase (LDH) was 1534U/L. His initial blood gas showed ph 7.136 pCO_2 42.9 mm/Hg, PO_2 91.1mm/Hg, oxygen saturation was 92.6% and electro cardiogram showed flat T wave with U wave. Urine examination showed pH 7, WBC 10/HPF; Myoglobin in urine was positive. Urine and blood culture was negative. Spot urine showed: Potassium 25 mmol/L with transtubular potassium gradient of 18.97, magnesium 1 mmol/l and chloride 48 mmol/l. 24 hours urine: Volume was 5104 ml/24 hr , creatinine was 7.5 mmol/L, creatinine clearance was 30ml/min(serum creatinine reach 175mmol/L), sodium was 194 mmol/L(Normal 27-287), potassium was 128 mmol/L(25-125), phosphorus was 31.64 mmol/L, magnesium was 5.1 mmol/L (3-5) and chloride was 245mmol/L (110-250). Free T4 was 15.2 and TSH was 4.19(normal).

The patient was admitted to the medical intensive care unit (MICU) as a case of hypokalaemic paralysis with possible renal tubular acidosis and renal failure. Trial of Foleys catheter insertion failed because of obstruction and so a supra pubic catheter was inserted, and the patient passed 800 ml of urine immediately. The patient was started on an intravenous fluid with a high dose of potassium through the central line followed by sodium bicarbonate. After a few hours, the patient became drowsy, and his arterial blood gas showed acute type 2 respiratory failure with PH 6.84, PCO2 115.7mm/Hg, PO2 158 mm/Hg and so the patient was intubated and initiated on a ventilator. During intubation, the patient passed about 4-5 liters/ day of urine daily, secondary to relief of obstruction, and had electrolyte disturbance with hypomagnesaemia, and hypophosphataemia for which he had replacement therapy. Moreover, he had 2 attacks of seizure for which he was given midazolam. After 5 days, his potassium and electrolytes improved, and he was extubated and started to move all four limbs.

Further Investigations

Renal Sonography showed: right kidney 8.5 × 4.2cm with dilated pelvis of 13 mm and left kidney measured 10.7×5.0 cm and 30 mm pelvic dilatation with distended urinary bladder and trace of perinephric fluid. Spiral computed tomography scan examination of abdomen and pelvis with delayed scan for urinary tract with intravenous contrast showed dilated extra renal pelvis of the left kidney with normal renal parenchyma [Table/Fig 1].



Thus, as urethral stricture suspected, flow cytometry, urodynamic micturating cystourethrogram and ascending urethrography were not conclusive, so cystoscopy was done which showed trabeculated bladder with thickening of mucosal fold from verumontanum, ending distal to sphincter, almost occluding the lumen. So resection was done, and Foleys catheter was inserted. The patient's condition improved and he was discharged in good condition with a Creatinine of 136mmol/l and a potassium level of 3.9 mmol/L.

Discussion

The probable causes of hypokalaemia are either reduced intake of potassium, or trans cellular shift of potassium or loss of potassium (non renal or renal)[1].The well known causes of hypokalaemic paralysis (HP) are thyrotoxicosis, familial and sporadic hyperaldosteronism, gastrointestinal loss and renal tubular acidosis (RTA)[2].Gastrointestinal loss is usually evident from history, in addition to

the laboratory test which shows low urine potassium.

Our patient did not have signs or symptoms of hyperthyroidism, as well as he had normal thyroid function test. Also, he had no family history, nor was his case sporadic, because of the electrolyte disturbance and high trans tubular potassium gradient.

In view of the above mentioned data, the most likely cause of hypokalaemia in this patient was renal tubular acidosis; it was found that patients with distal renal tubular acidosis had hypokalaemia in 28-53% of cases[4],[5]. Potassium wasting is due to primary tubular defect[6]. The presentation of the patient with hypokalaemic paralysis secondary to RTA cannot be differentiated from attacks of familial periodic paralysis. In both conditions, the muscle involvement can vary in severity from moderate muscle weakness to almost complete paralysis[3].A negative family history and high urine potassium make familial hypokalaemic paralysis unlikely.

Respiratory failure secondary to hypokalaemia is quite uncommon; a MEDLINE search showed that only few cases[7],[8],[9],[10],[11] of respiratory failure secondary to hypokalaemia have been reported in the literature.

This patient presented to the emergency department with combined metabolic and respiratory acidosis, but respiratory failure became very evident when he was given oxygen.

Emergency treatment of hypokalaemic paralysis due to renal tubular acidosis consists of intravenous potassium and bicarbonate therapy[2].Potassium should be replaced prior to bicarbonate therapy, because bicarbonate can precipitate further hypokalaemia due to intracellular potassium influx. Our patient was given potassium at the beginning and later on, he received bicarbonate. The definitive treatment is to treat the underlying cause; in our case , resection of the mucosal fold improved the condition.

In conclusion, this case highlights the significance of considering hypokalaemia as a significant cause of respiratory failure which needs prompt diagnosis and treatment to save the patient's life.

References

[1]. Singer GG, Brenner BM. Fluid and electrolyte disturbances. In: Harrison's Principles of Internal Medicine. 16th ed. Kasper DL, Fauci AS, Longo DL, Braunwald B, Hauser SL, Jameson JL Eds. McGraw-Hill, New York, 2005: 258-63.

[2]. Ahlawat SK, Sachdev A. Hypokalemic paralysis. Post Graduate Med J 1999; 75: 193-7.

[3]. Koul PA, Saleem SM, Bhat D. Sporadic distal renal tubular acidosis and periodic hypokalaemic paralysis in Kashmir. J Intern Med1993; 233 (6): 463-6.

[4]. Wrong OM, Feest TG. The natural history of distal renal tubular acidosis. Contrib Nephrol 21: 137, 1980.

[5]. 3Caruana RJ, Buckalew VM. The syndrome of distal (type 1) renal tubular acidosis: clinical and labrotary in 58 cases Medicine 67:84,1988

[6]. Sebastian A, McSherry E, Moris RC. Renal potassium wasting in renal tubular acidosis (RTA): its occurrence in type 1 and 2 RTA despite sustained correction of systemic acidosis. J Clin Invest 50: 667, 1971.

[7]. Gombar S, Mathew PJ, Gombar KK, D'Cruz S, Goyal G. Acute respiratory failure due to hypokalemic muscular paralysis from renal tubular acidosis. Anaesth Intensive Care. 2005 ;33:656-8

[8]. Poux JM, Peyronnet P, Le Meur Y, Favereau JP, Charmes JP, Leroux-Robert C. Hypokalemic quadriplegia and respiratory arrest revealing primary Sjogren's syndrome. Clin Nephrol 1992 ;37:189-91

[9]. Haddad S, Arabi Y, Shimemeri AA. Hypokalemic paralysis mimicking Guillain-Barre syndrome and causing acute respiratory failure. Middle East J Anesthesiol. 2004;17:891-7

[10]. Le Corre A, Veber B, Dureuil B.An unusual cause of acute respiratory distress Ann Fr Anesth Reanim. 2000;19: 549-51

[11]. Mishra I,MishraSK,PatiSS,Mohpatra DN: Renal Tubular Acidosis Presenting As Severe Hypokalemia With Respiratory Paralysis: Report Of Two Cases. The Internet Journal of Tropical Medicine. 2006: 3/1.