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Case Report

A CASE OF POSTPARTUM QUADRIPARESIS

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CASE DESCRIPTION *: A case of a 37 year old female P3+1 (3A), who presented with an eleven day history of progressive weakness of all limbs and inability to walk. On examination, she was not in obvious distress, however, power in both upper and lower limbs was less than 5/5. She was admitted as a case of Quadriparesis ?cause. Urgent serum electrolytes requested on admission revealed marked hypokalaemia of 1.3 mmol/L. Serum magnesium measured after 24 hours of IV potassium correction for deficit and daily requirements with minimal improvement in serum potassium values revealed hypomagnaesemia. She subsequently developed polyuria and polydipsia with urine output of 4.5 – 10.1 liters a day. She was then managed as a case of severe hypokalemia with nephrogenic diabetes insipidus using Tabs Ibuprofen 400 mg 6 hourly, Tabs Amiloride 5mg daily, and calculated potassium correction. She improved with resolution of all symptoms at discharge and restoration of serum potassium value.*

Keywords*:Hypokalaemia, Hypomagnesaemia, Nephrogenic Diabetes Insipidus, Postpartum quadriparesis*

INTRODUCTION

This is a case of a 37 year old P3+0 (3 alive) woman who presented with an 11 day history of gradual onset generalized body weakness and fever which progressed to quadriparesis at the time of admission. Weakness was insidious in onset, worse in the lower limbs, progressively worsening with no known aggravating or relieving factors. There was associated generalized body pains with concomitant moderate grade fever. There was no history of cough, headache, diarrhea, vomiting or urinary symptoms. There was also no history of trauma or seizures preceding symptoms. She has had no similar symptoms in the past. She is not a known diabetic or hypertensive. She had spontaneous vertex delivery of a life female neonate 2 days

prior to presentation with no immediate obstetric complications; however, the baby was found to have a swelling at the back and was being evaluated for a neural tube defect.

She booked index pregnancy at 14 weeks gestation in a peripheral hospital and had adequate antenatal care with no complications in the antepartum period. Her previous confinements were without complications. She attained menarche at 16 years and menstruates for four days in a regular 28-32 days cycle. She is a computer graphics designer and married in a monogamous family setting. She does not take alcohol or smoke cigarettes.

On examination, she was found to be anicteric, not pale, febrile (T=37.4°C), well hydrated, acyanosed, with mild pitting pedal oedema up to 1/3rd of the mid shin. Cardiovascular examination revealed a pulse rate of 82 bpm, regular, good volume, synchronous; blood pressure was 116/72 mmHg, apex beat was in the 5th left intercostal space, midclavicular line, jugular venous pressure was not raised, heart sounds were first and second only. The central nervous system revealed a fully conscious woman, oriented in time, place and person. Neuromuscular examination revealed symmetrically uniform limbs with no muscle wasting or fasciculations. There was global hypotonia with hyporeflexia. Power in the upper and lower limbs was 3/5 and 4/5 respectively.

MANAGEMENT AND OUTCOME

She was admitted as a case of Postpartum Quadriparesis ?cause to rule out Gullain-Barre syndrome. Intravenous antibiotics and supportive physiotherapy with routine postpartum care was commenced and samples were taken for urgent full blood count (FBC), serum electrolytes, urea and creatinine and a lumbar puncture for cerebrospinal (CSF) analysis was planned. Urgent serum electrolytes estimation revealed severe hypokalaemia of 1.3 mmol/L (3.5 – 5.0 mmol/L), serum sodium of 142 mmol/L, serum chloride of 110 mmol/L, serum bicarbonate of 17.5 mmol/L, and a repeat serum potassium done on the same day was 1.4 mmol/L. She was started on intravenous potassium correction at 120 mmol of potassium chloride in intravenous fluids (0.9% normal saline) per day to correct for deficit and daily maintenance and high potassium containing diet, but after 48 hours of correction there was no significant improvement. Serum magnesium was requested and came out as 0.5 mmol/L (0.7-1.0 mmol/L). Urine electrolytes were also assessed and came out as sodium of 38.4 mmol/L, chloride of 37.8 mmol/L and potassium of 5.7mmol/L. Her urine output was then noticed to be increasing and attained polyuric range on the 2nd day of admission. Random plasma glucose was 10.2 mmol/L, fasting plasma glucose was 4.8 mmol/L and HbA1c was 4%.

The correction for potassium was continued and magnesium correction using intravenous infusion of magnesium sulphate in 5% dextrose saline was commenced. Polyuria and polydipsia however worsened with urine output of 10.1 litres on day 5 of admission. Low dose Tabs Amiloride (5mg) was introduced with Tabs Ibuprofen 400mg 6 hourly to manage the current working diagnosis of severe hypokalaemia with nephrogenic diabetes insipidus secondary to hypokalaemia.

After 48 hours of therapy with the new regimen, she improved clinically with declining urine output, rising serum potassium and improved motor function enabling her to ambulate. Serum potassium was 3.8 mmol/l at discharge and urine output had declined to 3,150 mls per day.

The diagnosis at discharge was that of refractory hypokalaemia due to hypomagnesaemia complicated by nephrogenic diabetes insipidus secondary to hypokalaemia.

DAYS ON ADMISSION	POTASSIUM	MAGNESIUM	URINE OUTPUT
DAY 1	1.3 - 1.4 mmol/l	0.5 mmol/l	1,500 ml
DAY 2	1.8 mmol/l	0.7 mmol/l	4,800 ml
DAY 3	1.6 mmol/l	-	7,200 ml
DAY 4	1.7 mmol/l	-	8,100 ml
DAY 5	1.9 mmol/l	-	10, 100 ml
DAY 6	2.6 mmol/l	0.89 mmol/l	3,700 ml
DAY 7	3.3 mmol/l	-	3,500 ml
DAY 8	3.8 mmol/l	-	3,150 ml

Table 1; Serum and Urine Electrolyte Values and Urine Output of the Patient.

DISCUSSION

Potassium is the most abundant intracellular anion found primarily in the skeletal muscle and bone, and participates with sodium in the maintenance of body osmotic balance. Potassium homeostasis in the body is regulated by the kidneys through the excretion of urine and regulation of absorption after dietary intake (Scott *et al.*, 2012). Potassium homeostasis is under the control of various hormones like aldosterone and insulin which stimulates renal and cellular potassium uptake respectively (Jensen *et al.*, 2015).

Hypokalaemia is an electrolyte disorder characterized by low serum potassium (<3.5 mmol/l) (Jensen *et al.*, 2015). It is a common electrolyte disorder associated with adverse outcomes which include paralysis (as seen in the index patient), intestinal ileus, cardiac arrhythmias and death (Asmar*et al.*, 2012). It is associated with mortality among hospitalized patients with heart disease on digoxin therapy and has an incidence of 16.8% in first-time admissions (Jensen *et al*, 2015). The body maintains serum potassium within a narrow limit, however, symptomatic hypokalaemia with severe potassium depletion can affect renal tubular function and accelerate progression of chronic kidney disease (Asmar*et al.*, 2012). This was a possibility in the index patient who had defective tubular reabsorption of water noticed after a period of hypokalaemia.

Hypokalemia is usually a symptom of an underlying pathology, or a side effect of diuretic drugs use (Jensen *et al.*, 2015). Long standing hypokalemia may indicate depletion of total body potassium or increased renal potassium loss. In individuals with simple potassium depletion, potassium replacement therapy should correct serum potassium concentration. Potassium replacement may not be adequate when there is renal potassium wasting (Asmar*et al.*, 2012) which was a possibility in the index patient.

The approach to management is dictated by the clinical picture and the serum potassium concentration (Jensen *et al.*, 2015) and the goal is to correct serum potassium deficit without provoking hyperkalaemia.

Diabetes Insipidus (DI) is a clinical condition that results from inappropriate secretion (central) or action (nephrogenic) of serum antidiuretic hormones characterized by polyuria (defined as 24 hour urine output in excess of 40 ml/kg, 3 litres/day) and polydipsia (Shapiro, 2012). Various pathologic conditions underlie Diabetes Insipidus eventually leading to the production of *insipid* urine that is hypotonic and bland in a polyuric state (Karet, 2011). The prevalence of Diabetes Insipidus in the general population is approximately one per 25,000 – 30,000. Diabetes Insipidus could either be congenital or acquired – the congenital forms are the familial neurohypophyseal DI (FNDI) and the congenital nephrogenic DI (NDI) while the acquired forms of DI are also either central or nephrogenic (Moeller *et al.*, 2013).

Acquired Nephrogenic Diabetes Insipidus results from impaired renal response to the activity of antidiuretic hormone (Arthus*et al.*, 2000). Metabolic imbalances like hypokalaemia and hypercalcaemia are associated with decreased sensitivity of the nephrons to ADH (Nephrogenic diabetes insipidus) (Garofeanu*et al.*, 2005) which could be responsible for the polyuric state in the index patient who was initially severely hypokalaemic. The pathogenesis of Nephrogenic Diabetes Insipidus from hypokalaemia has not been fully explained,

however, certain postulates have been advanced; in addition to a decreased sensitivity of the distal convoluted tubules and cortical collecting ducts to ADH, there is also decreased production of the countercurrent gradient via the Na-K-2Cl co-transporter in the thick ascending limb of Henle (Shapiro, 2012). Hypokalemia has also been noted to induce thirst leading to associated polyuria and polydipsia and impaired countercurrent multiplication (Luke, 1985). The reversal of these metabolic derangements usually lead to the reversal of the Nephrogenic Diabetes Insipidus (Garofeanu*et al.,* 2005) which could account for the resolution of polyuria and the improved clinical state of the index patient at discharge.

LIMITATIONS

Serum osmolality, urine osmolality, aldosterone, ADH and genetic analysis would have contributed to the evaluation of the patient but were not available at the time of management of this patient.

Dynamic function test with ADH analogues was however withheld as the patient responded to empirical management of nephrogenic diabetes insipidus

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