CASE REPORTS

Hyperventilation of Pregnancy Presenting with Flaccid Quadriaparesis Due to Hypokalaemia Secondary to Respiratory Alkalosis

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Abstract
Hyperventilation in pregnancy is a cause of chronic respiratory alkalosis. Alkalosis either metabolic or respiratory may cause intracellular shift of potassium ions that may lead to hypokalaemia. However, the resultant hypokalaemia in respiratory alkalosis is usually mild and does not cause much clinical features. A five-months-pregnant female of the age 25 years presented with sudden onset flaccid weakness of both lower limbs associated with thigh muscle pain followed by weakness of both upper limbs within three days. Subsequent investigation revealed severe hypokalaemia due to acute exacerbation of chronic respiratory alkalosis secondary to hyperventilation of pregnancy, other causes of hypokalaemia being ruled out. Respiratory alkalosis causes tetany and other clinical manifestations. But hypokalaemia and such weakness is rarely found. This is probably the first report of this type from India.

Introduction
Alkalosis either metabolic or respiratory may cause intracellular shift of potassium ions that may lead to hypokalaemia. Hypokalaemia in respiratory alkalosis is usually mild and does not cause much clinical features. Hyperventilation in pregnancy is a cause of chronic respiratory alkalosis. It is usually not associated with significant hypokalaemia and muscle weakness. We here report a case of severe hypokalaemia secondary to hyperventilation induced respiratory alkalosis in a pregnant woman leading to quadriaparesis. As far as we could search, this is probably the first report of this type from India.

Case Report
A 25 year old, five months-pregnant female presented with sudden onset flaccid weakness of both lower limbs associated with thigh muscle pain followed by weakness of both upper limbs within three days (Figure 1). There was no history of sensory abnormality, bladder-bowel abnormality, cranial nerve involvement, headache, convulsion, altered sensorium, trauma or pain in the spine. There was no history of similar weakness of limbs in the past or any thyroid disorders. She had one past history of anxiety disorder, but no treatment was given. On admission, the patient was found to be hyperventilating with a rate of 46/minute. Examination of the nervous system revealed hypotonia of all four limbs with power 3/5. All tendon jerks were decreased with bilateral flexor plantar responses. No sensory signs were found.

Laboratory tests revealed haemoglobin of 9 gm% with total leucocyte count of 8900/µL (neutrophil 74%, lymphocyte 20%) and platelet count of 3 lakh/µL. Blood urea and creatinine were 14 and 0.6 mg% respectively. Liver function tests were normal. Analysis of serum electrolytes revealed sodium (Na⁺) 141 mEq/L, potassium (K⁺) 1.8 mEq/L, calcium (Ca²⁺) 9.2 mg/dL (normal 8.4—10.2 mg/dL) and magnesium (Mg²⁺) 1.7 mg/L (normal 1.9—2.5 mg/L). Arterial blood gas (ABG) analysis revealed a pH of 7.57 with pCO₂ of 20 mm of Hg, serum bicarbonate of 18 mEq/L and pO₂ of 100 mm of Hg.
The picture was suggestive of partially compensated respiratory alkalosis. Urinary potassium excretion was 8.93 mmol/L and the transtubular potassium gradient (TTKG), calculated from urinary osmolarity, and came as 3.11. Thus, any renal loss of potassium was ruled out. ECG showed prominent U waves with attenuated T waves (Figure 2). Serum cortisol and aldosterone levels were normal. Thyroid function test was normal.

The patient was treated with intravenous (IV) potassium chloride solution in 100 ml normal saline (0.9%) through peripheral channel at the rate of 20 mEq/hr. Oral potassium supplementation was also started. Again, considering the respiratory alkalosis and hyperventilation, respiratory physiotherapy was started. Patient counselling was done to reduce the anxiety. By the second day, the patient started to have gradual improvement of weakness with return of serum potassium to normal (2.8 mEq/L on day 3; 3.6 mEq/L on day 4). Her respiratory rate also came down to 22/min on day 3 and 16/min by day 5. Repeat ABG analysis showed pH 7.51 on day 3 and 7.47 on day 5. Although serum magnesium was slightly low (1.7 mg/L) it did not need any treatment and repeat Mg on day 5 showed value of 2 mg/L. Fortunately, her foetus did not suffer during this episode and an ultrasonography before discharge showed single live foetus (Figures 3 - 4). She had not developed any other similar episode in follow up though mild chronic respiratory alkalosis persisted.

Discussion

Human pregnancy is characterised by significant increases in minute ventilation. This hyperventilation is due to excess blood progesterone hyperstimulating the respiratory centre, along with pregnancy induced increased wakefulness drives to breathe. Hyperventilation can lead to respiratory alkalosis that causes tetany and other clinical manifestations like tingling of hands and fingers or feeling light-headed or dizzy. But hypokalaemia and resultant weakness due to respiratory alkalosis is rarely reported.

A case report from Korea described a case of severe hypokalaemia and hypocalcaemia following hyperventilation in spinal anaesthesia. Their patient developed severe hypokalaemia at operation theatre (OT) after giving spinal block, with suggestive ECG changes. The patient was reversed with closed mask ventilation and IV potassium infusion. Hyperkalaemia in that patient needed urgent IV therapy. Our patient also responded with potassium infusion. However, our patient did not manifest any hypocalcaemia. In a study from New Delhi, they found that hyperventilation can cause significant hypokalaemia, especially of sudden onset. Animal study also revealed occurrence of hypokalaemia in respiratory alkalosis. In contrary to these, acute respiratory alkalosis in hyperventilation resulting in hyperkalaemia has also been reported. Hyperkalaemia was induced by a hypobicarbonataemia mediated increment in alpha-adrenergic activity and modulated in magnitude by counterbalancing enhanced beta-adrenergic activity. Abrupt cessation of respiratory alkalosis resulted in rapid fall in plasma potassium with hypokalaemic overshoot. So there may be variable response regarding plasma potassium level in respiratory alkalosis in hyperventilation.

Our patient developed severe hypokalaemia due to acute exacerbation of respiratory alkalosis. Hypokalaemia in pregnancy can also be due to various reasons. It can be
caused by recurrent vomiting, excess fluid retention or rarer causes like geophagia. However, our patient had none of these features and respiratory alkalosis was the only possible cause of her symptoms. Also, urinary potassium excretion and osmolarity tests ruled out any possibility of inherited conditions like Gitelman syndrome. Our patient presented with weakness, but the main concern in these cases is the sudden cardiac arrhythmias that can cause death. For every 10 mm of Hg decrease in PaCO2, usually there is 0.5 mmol/L drop in serum K+. Thus, normally in mild respiratory alkalosis of pregnancy, the drop in K+ is not significant, but in patients with pre-existing electrolyte disorders and in severe respiratory alkalosis, this drop may be fatal. Hence, in any patient suspected of hyperventilation, regular serum electrolytes and cardiac monitoring is essential. Hypokalaemia can adversely affect the foetus by causing bradycardia and decreased movements. Thus, prompt treatment is needed.

Weakness following hyperventilation can also be caused by transient severe hypocalcaemia. Thus, checking of both serum K+ and Ca²⁺ are essential. However, usually, hypocalcaemia causes spasticity whereas hypokalaemia causes flaccidity.

This case highlights a rare complication of pregnancy hyperventilation. In any pregnant patient presenting with neurological symptoms, an examination of the respiratory pattern can be helpful in elucidating the cause in many cases. Thus, unnecessary investigations can be avoided. Analysis of ABG in hypokalaemia cases can point towards the diagnosis.

References