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Case Report

Obstetrics and Gynaecology Section

Hypokalemic Paraplegia in Pregnancy

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ABSTRACT

Hypokalemic myopathy may range from numbness/weakness to complete paralysis. The aetiology may be congenital or acquired. It is characterized by acute muscular weakness with low levels of potassium (<3.5 meq/L). We present a case of 26-year-old multigravida at 36 weeks of gestation with gestational hypertension on treatment, who came with acute onset of pain, numbness and weakness of both legs which worsened following betamethasone injection. She was diagnosed to have Hypokalemic paralysis with potassium levels of 2.1 meq/L. The medical profile remitted promptly on intravenous potassium replacement. Pregnancy was continued till 37 weeks with oral potassium supplements, antihypertensives and regular monitoring of serum potassium levels. The pregnancy was terminated after 37 weeks in view of gestational hypertension. Postpartum period was uneventful, patient was discharged after two weeks when potassium levels and BP returned to normal.

Keywords: Hypokalemia, Myopathy, Paralysis, Pregnancy

CASE REPORT

A 26-year-old Gravida 2 Para 1 Living 1 with previous normal delivery four years back, a booked case in our hospital, presented with eight months of amenorrhea with h/o increasing pedal oedema, pain and numbness in both the legs since five days. Patient was being treated for gestational hypertension since 7th month with nifedepine 10 mg one BD. There was no history of similar episodes of weakness in the past. On admission clinical examination revealed bilateral pedal oedema of grade 3 and BP of 160/100 mm of Hg. Obstetric examination revealed 34 week gravid uterus with a longitudinal lie and cephalic presentation. Liver function tests were within normal limits. An ophthalmologic examination revealed grade II hypertensive retinopathy. The dose of nifedepine was increased to 10mg TID and Inj. Betamethasone was advised for fetal lung maturity. Following 1st dose of Inj. Betamethasone, patient started complaining of worsening of numbness and weakness and inability to walk. Initially the electrolyte profile was normal, therefore the cause remained inconclusive. A neurological opinion was sought, after neurological examination a diagnosis of spastic paraplegia was made, a repeat electrolyte profile showed potassium levels of 2.1 meq/L and ECG showed U waves. A diagnosis of Hypokalemic paralysis was made and patient was administered 40mg potassium in 500ml of normal saline and later followed by potassium oral syrup 15 ml TID along with tab. nifedepine 10mg TID for hypertension with daily potassium monitoring. The pregnancy continued till 37 weeks, a decision to terminate the pregnancy was made in view of hypertension. The lady delivered, following induction of labor, a male baby weighing 2.85 kg, with an APGAR score of 8 and 9 at the 1st and 5th min respectively. Postpartum period was uneventful. Potassium levels were monitored daily and the daily dose was tapered and then gradually stopped. Following delivery, antihypertensives were continued for a week and once BP stabilized it was tapered and stopped. Patient was discharged after two weeks, at discharge BP was normal and potassium levels were normal. The lady was reviewed in the outpatients department after six weeks, she had no complaints, the potassium levels were normal, and the growth of the infant was also normal.

DISCUSSION

Hypokalemic paralysis during pregnancy has a rare occurrence. It manifests as acute muscular weakness associated with low potassium levels. Any pregnancy associated with paralysis is a high-risk pregnancy, and must be treated as such. If a woman is experiencing significant weakness or attacks of paralysis the associated cardiac, respiratory and muscular problems may well pose risks, and such a patient will require careful monitoring and informed care. A thorough cardiac evaluation is essential. The medical team must be prepared to appropriately handle an episode of weakness or paralysis, if one develops during labor and delivery.

The aetiology of hypokalemia may be varied, ranging from congenital to acquired causes [1]. A careful insight into the past history regarding the age of onset, precipitating factors like, episodes of weakness, following exercise, carbohydrate load, increased salt intake etc, may be helpful to make a diagnosis of congenital abnormalities. The lady in the present scenario neither gives any such history in the past, nor there are, any other members in the family who suffer from similar condition, thereby familial conditions like, Familial Hypokalemic Periodic Paralysis (FHPP), Thyrotoxic Periodic Paralysis, Anderson Tawill Syndrome ruled out. Investigations like ictal potassium measurements, T3, T4, TSH levels and an ECG, may be helpful in differentiating these conditions [2]. It is imperative to diagnose these conditions before pregnancy, as worsening of FHPP has been reported during pregnancy [3]. The electrolyte profile was normal in this lady except for hypokalemia. The rennin-angiotensin-aldosterone axis was normal, so also the urinary calcium levels, hence Bartter's syndrome [4], Gittleman syndrome [5] and Bartter like conditions were ruled out. There was no metabolic acidosis and renal function tests were normal, not suggestive of distal or proximal renal tubular acidosis [6-10]. There was no history of pica in this lady to suggest ingestion of any unusual substance [11]. Case reports of Hypokalemic paralysis following glucose screen also exist, but no such precipitating factors exist in this lady [12]. The only precipitating factor noted was injection of betamethasone, following which the lady complained of increasing weakness followed by paraplegia, a similar case has been cited in the literature [13].

The occurrence of myopathy in a pregnant woman is, often of much concern to the patient and her relatives, and challenging for the treating clinician [14]. Due to a wide variety of the causes, there is often a delay in recognition and treatment. The myopathy may be trivial and sometimes severe enough to warrant ventilation [4]. The challenge lies in timely recognition of the cause and prompt correction. The acute episode may be managed with intravenous potassium replacement and maintenance with oral potassium supplements [2]. Even in our case, a high index of suspicion led to the early recognition of this seemingly alarming condition, hence preventing more serious complications like respiratory paralysis or fatal arrhythmias.

Whatever may be the cause of hypokalemic paralysis, the condition is promptly reversible with potassium administration and vaginal delivery is usually possible with careful monitoring, epidural analgesia, avoiding active maternal efforts (passive descent and elective outlet forceps) and other stimulatory factors [3], as is the case of this lady. However, the neonate may have episodes of flaccid paralysis, respiratory distress at birth. Feeding and respiration may be a problem; therefore a close watch has to be kept on the neonate, under expert supervision [15].

CONCLUSION

Neuromuscular disorders in pregnancy are often alarming for patients and challenging for the clinicians. The heterogeneity of the causes makes it more difficult to diagnose and manage the underlying condition. Behavioral and environmental management, trigger identification and dietary modification may be helpful along with drugs to maintain the potassium levels within normal limit in familial conditions. Though the aetiology was uncertain in this case, the condition aggravated following administration of injection betamethasone. Therefore, steroids as a cause of Hypokalemic paralysis should always be borne in mind. The seemingly alarming myopathy may be amenable to simple correction of electrolytes, undetected may lead to dangerous respiratory paralysis with patient ending up on a ventilator. With timely detection and prompt management there is a scope for vaginal delivery without any maternal and fetal complications.

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