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Case Report

A Rare Case of Neonatal Hypochloremic Metabolic Alkalosis Causing Hypoxia and Hyperlactatemia Secondary to a Probable Underlying Maternal Reason

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Abstract

Metabolic alkalosis in neonates within few hours of life is extremely rare. The most common causes of metabolic alkalosis in neonates can be attributed to vomiting, diuretic use, Bartter syndrome and maternal hypochloremia. There have been a few reported cases describing neonatal metabolic alkalosis.

We will be presenting a unique case of a male neonate that was found have tachypnea with shallow breathing, and desaturations at 7 hours of life, extensive workup was done and revealed normal serum sodium (Na 134) mmol/L and potassium levels (K 3.9 mmol/L), low serum chloride (Cl 79 mmol/L), urine chloride of < 50 mmol/L, high lactate of 9.6 mmol/L and metabolic alkalosis (blood gas pH 7.54, PCO2 35.2 mmHg, HCO3 44 mmol/L, BE 16.1 mmol/L). Within three days of life lactate level and electrolytes started to normalize. Maternal history was significant for frequent hospital admissions due to undiagnosed hypokalemia. Eventually postnatal as part of the neonatal management, the requested maternal labs showed hypokalemia of 2.3 mmol, hypomagnesaemia of 0.64 mmol/L, hypochloremia of 77 mmol/L and metabolic alkalosis (pH 7.55, PCO2 53.2 mmHg, HCO3 47 mmol/L). She is currently under workup for possible Gitelman syndrome.

Our case highlights the importance of evaluation of maternal electrolyte derangement and acid-base disturbances during neonatal management of metabolic acidosis in the first few days of life. Metabolic alkalosis may present with hypoxic and hypoperfusion status.

Keywords: *Metabolic Alkalosis; Hypochloremia; Hyperlactatemia; Neonate; Neonatal Hypoxia*

Introduction

Significant metabolic alkalosis in the early postnatal period is an uncommon problem. Etiologies include renal, gastrointestinal, metabolic and endocrine causes, maternal abnormalities, and exogenous sources.

Metabolic alkalosis in neonates is very rare and attributed to gastric fluid losses, diuretics and congenital chloride diarrhea (CCH). There were few cases reported about that these are due to maternal bulimia, Bartter's, vomiting and CCH.

Metabolic alkalosis, a disorder that elevates the serum bicarbonate, can result from several mechanisms: intracellular shift of hydrogen ions; gastrointestinal loss of hydrogen ions; excessive renal hydrogen ion loss; administration and retention of bicarbonate ions; or volume contraction around a constant amount of extracellular bicarbonate (contraction alkalosis) (Table 1) [1-4].

Case Presentation

We are discussing a case of a late preterm 35 weeks' baby boy, who was born by normal vaginal delivery, the birth weight was 2.35 kg, APGAR score after birth was normal.

At 7 hours of life he was admitted to the NICU with tachypnea shallow breathing and desaturations no retraction nor nasal flaring.

Upon initial evaluation, his lab investigations were showing hypochloremia with metabolic alkalosis, and elevated lactate. Na 134 mmol/L, K 3.9 mmol/L, Cl 79 mmol/L, urine chloride of < 50 mmol/L, lactate 9.6 mmol/L, ammonia 22.6 micromol/L, arterial blood gas showed pH 7.54, PCO_2 35.2 mmHg, HCO_3 44 mmol/L, BE 16.1 mmol/L.

Echocardiography showed suprasystemic pulmonary HTN, not requiring any treatment, acceptable for age, planned for follow up.

He was having signs of respiratory distress; therefore, he was started on nasal cannula 2 L/min chest X-ray was normal.

The impression at that time was the presence of a possible metabolic/renal disease causing the abnormal lab values, since cardiac causes were ruled out and initial inflammatory markers were reassuring.

The possibility of neonatal sepsis was also entertained, initial blood culture showed no growth antibiotics were stopped after 48 hrs.

His physical examination and remainder of vital signs remained normal throughout the time.

He didn't have polyuria, vomiting, nor diarrhea. The tachypnea resolved gradually over two days.

His antenatal records were reviewed and were only significant for bilateral choroid plexus cysts and echogenic cardiac foci on antenatal scan.

No antenatal history of oligo or polyhydramnios.

Renal ultrasound was done at day 5 of life it showed both kidneys appear normal in size, shape and echotexture.

Maternal history was significant for a hospital admission with hypokalemia, hypomagnesaemia and hypothyroidism, suspected to have Gitelman syndrome. No family history of any renal, metabolic or endocrine disease.

Input from the Nephrology and Metabolic teams were obtained.

From the metabolic point of view, there was no explanation for the metabolic alkalosis, since his ammonia level was normal, and the elevated lactate could have been due to the tachypnea he had at birth, so no further recommendations were given.

The Nephrology team entertained the possibility of Bartter syndrome but it is less likely as neonatal types that usually present with polyhydramnios with normal electrolytes and urine output, and they believed it is too early to diagnose Gitelman syndrome. Other extra-renal etiologies are possible, but very prematurely to present as well, like cystic fibrosis (CF), pyloric stenosis and congenital chloride diarrhea). But he does not have clinical features that may suggest CF like delayed passage of meconium, meconium ileus. No history of diarrhea nor repeated vomiting.

Baby was started with IV fluid 80 ml/kg/day D10% with sodium chloride and potassium chloride supplement. At second day of life He was stable, off nasal cannula, tolerating full oral feeds, blood culture was negative, antibiotics were stopped, and next day levels of chloride, creatinine, lactate and blood gas were normalized.

So, he was discharged from the NICU, with scheduled clinic follow ups.

He was seen in the clinic for his follow ups and well-baby visit/vaccinations and is doing perfectly well.

Currently he is 21 months old, growing and developing appropriate for age.

Discussion

Metabolic alkalosis is an acid base disturbance that is caused by either loss of an acid or gain of a base. It's not a disease by itself, but rather a sign of an underlying disease process.

Metabolic alkalosis in neonatal period is extremely rare. Multiple attributing factors has been discussed in literature that are revised in table 1 [1,2].

1. Renal	2. Gastrointestinal	3. Endocrine
Loop/Thiazide diuretics use	Vomiting (pyloric stenosis)	Hyperaldosteronism
Hypokalemia	Diarrhea (congenital chloride diarrhea)	Cushing Syndrome
Barter Syndrome	Laxative use	Congenital adrenal hyperplasia (ll-beta hydroxylase or 17-alpha hydroxylase deficiencies)
Gitleman Syndrome	Nasogastric suctioning Low chloride formula	
4. Metabolic	**5. Maternal**	**6. Exogenous**
Recovery from acidosis	Maternal hypochloremic alkalosis	Use of bicarbonate, lactate, citrate or acetate infusions

Table 1: *Causes of Hypochloremic metabolic alkalosis in neonates/infants.*

In our case, the presentation was transient, the patient had complete resolution of all symptoms, did not have any gastrointestinal losses nor any evidence of endocrine disorders, no history of diuretic/laxative use nor any alkali infusion use during his hospital stay. In addition, the laboratory findings normalized upon regular visits to our nephrology clinics until the age of 1 year. Thus, the diagnosis was transient neonatal hypochloremic metabolic alkalosis secondary to maternal hypochloremic metabolic alkalosis.

Patients may have different presentations secondary to metabolic alkalosis. Some may present with lethargy and seizure, as alkalosis effect in the brain may lead to cerebral vasoconstriction [3].

Others may present with hypoventilation, apnea and desaturations and this is because the respiratory drive is inhibited via the central and peripheral receptors [3-5]. Decreased myocardial contractility and arrhythmias have been also reported in literature [3].

Conclusion

Neonatal metabolic alkalosis within few hours of life could be secondary to maternal causes. We are highlighting in our paper the importance of evaluating maternal electrolyte derangement and acid-base disturbances during management of neonatal metabolic acidosis. Metabolic alkalosis may present with hypoxic and hypoperfusion status. Treatment is by providing the patient with IV fluid and correction of electrolyte disturbances. Regular follow ups are needed for neurodevelopment evaluation.

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