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Metabolic alkalosis in a newborn infant

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INTRODUCTION

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

CASE REPORT

A 9-hour-old newborn female infant, born at 38 weeks of gestational age by vaginal delivery, birth weight 3200 g, was admitted to the intensive care unit for hypoventilation (respiratory rate 20-30 breaths per minute) and desaturations. Postnatal adaptation had been uncomplicated with Apgar scores of 8, 9 and 9 at 1, 5 and 10 minutes, respectively. Rupture of membranes occured 5 hours before birth and the amniotic fluid was clear. The mother was afebrile during labor and delivery. The infant did not become apneic and breathing seemed to be regular. There were no signs of upper airway obstruction. When the infant was awake, color, activity, and breathing were normal. Glucose level was normal, and neither hypercalcemia or hypermagnesemia were present. Blood gas analysis showed a pH of 7.48, pCO2 of 7.2 kPa, a BE of + 12.8 mmol/l, and a bicarbonate concentration of 39.9 mmol/l. Serum electrolyte values were as follows: sodium 133 mmol/l, potassium 3.5 mmol/l, chloride 88 mmol/l. On the basis of theses results, hypochloremic metabolic alkalosis was felt to be responsible for the infant's hypoventilation and desaturations. Alkalosis was present at birth with umbilical arterial and venous pH values of 7.45 and 7.60, respectively.

Maternal chloride deficiency was suspected to be the cause of the patient's initial hypochloremic alkalosis. On examination, the mother appeared cachectic but otherwise well. Maternal serum laboratory values showed hypochloremic alkalosis with a chloride of 90 mmol/l and a potassium of 2.3 mmol/l. The mother admitted that she was suffering from bulimia, she denied diarrhea, recent illnesses and the use of diuretics or laxatives. The eating disorder was present at the beginning of this pregnancy and the woman experienced worsening of symptoms during her pregnancy.

The infant's alkalemia was treated by the administration of sodium and potassium chloride. 24 hours later, alkalosis and chloride concentration were normalized. Breathing was regular at a frequency of 40 to 60 per minute and no desaturations were observed. On the fourth day, the infant was discharged from the hospital.

DISCUSSION

In this patient, metabolic alkalosis was present at birth. During fetal life, the placenta compensates for many metabolic disorders of the fetus. The transport of a substance from the mother to the fetus depends on the concentration gradient between mother and fetus, the transport mechanism across the placenta (Fig. 1), the availability of appropriate carrier proteins if they are required, as well as the placental modification of the transported substance (Fig. 2). Alkalemia at birth is always secondary to maternal abnormalities and not fetal in origin. Later, many causes of hypochloremic alkalosis in the

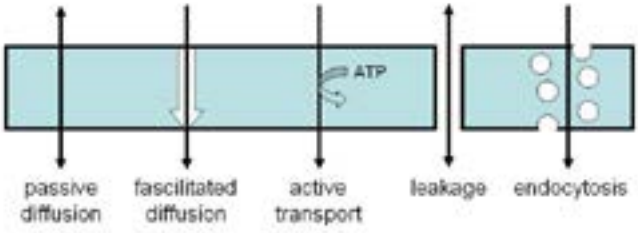

Fig. 1

Different placental transport mechanisms

infant are known (Tab. 1) (1, 2). In the presented case, maternal hypochloremic alkalosis was secondary to bulimia (chloride depletion due to chronic loss of gastric fluids). With the help of Stewart's concept it is possible to quantify the contribution of chloride to changes in bicarbonate and base deficit (3-5). This concept applies the laws of mass action, mass conservation and preservation of electroneutrality to all components of human plasma that influence hydrogen ion concentration (Fig. 3). The chief source of hydrogen ions is via water dissociation, which is in turn affected by three independent variables: pCO_2, total amount of weak acids and the strong ion difference (Fig. 4). The effect of chloride can be quantified by solving simultaneously all of the required Stewart equations: with regression equations

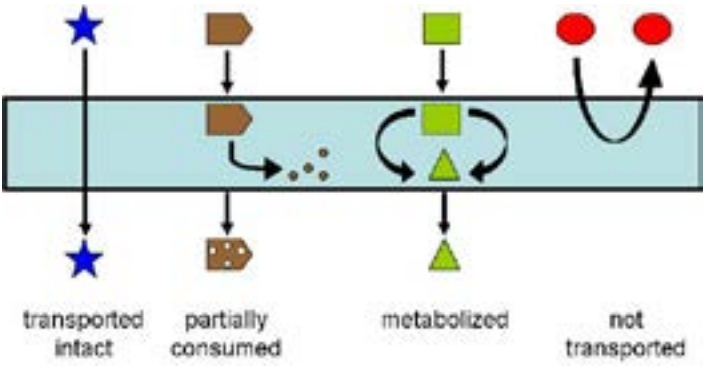

Fig. 2

Placental modification of transported substances.

Causes of hypochloremic metabolic alkalosis in the infant.

Tab. 1

Renal causes Diuretics Bartter syndrome Gitelman syndrome	**Gastrointestinal** Chronic loss of gastric fluids (vomiting) Diarrhea Laxative abuse
Endocrine cause Hyperaldosteronismus	**Metabolic cause** Recovery from acidosis
Maternal cause Exogenous cause	**Exogenous cause** Bicarbonate, acetate infusion

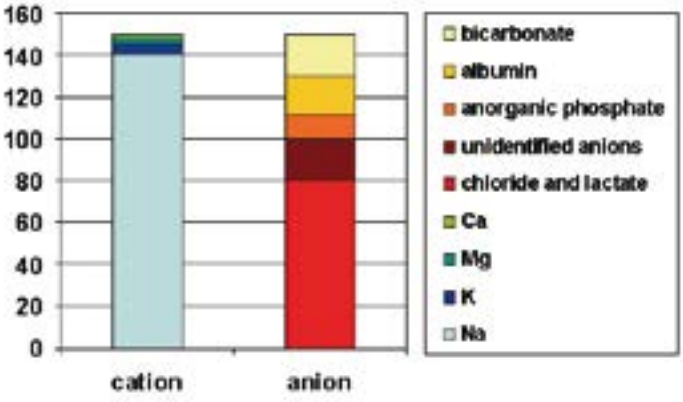

Fig. 3

Stewart methodology: Preservation of electroneutrality.

DISCUSSION

it is possible to predict how much base deficit and bicarbonate concentration varies with changes in chloride: chloride-induced change in base deficit = 0.99 x (Na-Cl) - 31.9 and chloride-induced change in bicarbonate concentration = 0.92 x (Na-Cl) - 5.8 (4). The regression equations in the presented case therefore are: chloride-induced base deficite = 0.99 x (133-88) - 31.9 = 12.6 mmol/l and chloride-induced bicarbonate = 0.92 x (133-88) - 5.8 = 35.6 mmol/l.

Maternal hypokalemia did not significantly affect fetal serum potassium levels, because the placental potassium pump functions unidirectionally: maternal hyperkalemia produces fetal hyperkalemia, but maternal hypokalemia generally does not produce fetal hypokalemia. The placental chloride transit is coupled to several ion pumps, mostly in exchange to bicarbonate. Under conditions of hypochloremia it is possible that chloride and bicarbonate ions diffuse passively through the placenta with closely matched maternal and infant serum levels of chloride and bicarbonate (1).

In the 1980's, chloride deficient formula milk caused severe hypochloremia during infancy and lead to neurologic sequelae (6). The impact of hypochloremia on the fetus is not known, but gestation is obviously a sensitive time for CNS development. Fortunately, in our patient neurodevelopment is normal at the age of 2 years.

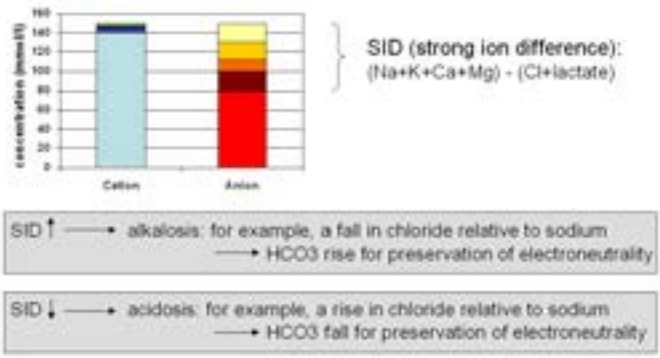

Fig. 4

Stewart methodology: Strong ion difference and the influence on alkalosis and acidosis (strong ion: molecules which are completely dissociated at physiological pH, the predominant components being sodium and chloride).

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