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Fetal Metabolic Alkalosis Resulting from Maternal Vomiting

William M. Curtin, MD[1,2] Emily A. O'Brien, BS[3] Rachel M. Mauro, MD[1]
Elizabeth A. Lucarelli-Baldwin, MD[1] Serdar H. Ural, MD[1] Christina T. DeAngelis, MD[4]

[1] Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, PennState Health Milton S. Hershey Medical Center, PennState College of Medicine, Hershey, Pennsylvania
[2] Division of Maternal-Fetal Medicine, Department of Pathology, PennState Health Milton S. Hershey Medical Center, PennState College of Medicine, Hershey, Pennsylvania
[3] Department of Medical Education, PennState College of Medicine, Hershey, Pennsylvania
[4] Division of Women's Health, Department of Obstetrics and Gynecology, PennState Health Milton S. Hershey Medical Center, Hershey, Pennsylvania

Address for correspondence William M. Curtin, MD, PennState Milton S. Hershey Medical Center, Mail Code H103, 500 University Drive, PO Box 850, Hershey, PA 17033-0850 (e-mail: wcurtin@pennstatehealth.psu.edu).

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Abstract

We describe a pregnant patient with severe compulsive water ingestion and vomiting that lead to metabolic alkalosis and preterm delivery. A 21-year-old patient was hospitalized multiple times throughout pregnancy for symptoms initially thought to be related to hyperemesis gravidarum. Overtime, it became apparent that the patient induced vomiting by rapidly drinking large volumes of water. At 32 weeks' gestation, rapid ingestion of water caused 3 days of vomiting with findings of hyponatremia, hypokalemia, hypochloremia, metabolic alkalosis, and compensatory respiratory acidosis. Fetal monitoring showed minimal variability and recurrent decelerations; subsequent biophysical profile score of 2/10 prompted urgent cesarean section. A male newborn was delivered and cord blood gases reflected neonatal metabolic alkalosis and electrolyte imbalances identical to those of the mother. Compensatory hypoventilation in both mother and fetus were treated with assisted ventilation. With saline administration and repletion of electrolytes, metabolic alkalosis resolved for both patients within days.

Metabolic alkalosis was transplacentally acquired by the fetus. This case demonstrates the development of metabolic alkalosis in a pregnant woman caused by vomiting severe enough to prompt preterm delivery for nonreassuring fetal status. It also demonstrates fetal dependence on both placenta and mother to maintain physiologic acid–base and electrolyte balance.

Keywords
- pregnancy
- hyperemesis
- alkalosis
- newborn

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Table 1 Maternal and newborn chemistry with reference values in parentheses

Analyte	Maternal value	Newborn value
Na^+ (mmol/L)	129 (136–145)	123[a] (138–146)
K^+ (mmol/L)	2.8 (3.5–5.1)	3.0 (3.5–5.1)
Cl^- (mmol/L)	< 60 (98–107)	62 (98–107)
BUN (mmol/L)	12.49 (2.14–8.21)	12.85 (2.14–8.21)
Creatinine (μmol/L)	280.60 (45.75–76.25)	268.40 (22.88–76.25)
HCO_3^- (mmol/L)	43 (22–29)	38 (22–29)
Ca^{2+} (mmol/L)	2.10[b] (2.10–2.55)	1.70 (1.90–2.60)
Ionized calcium (mmol/L)	0.20 (0.29–0.32)	0.17[a] (0.28–0.33)
PO_4^{3-} (mmol/L)	2.39 (0.94–1.45)	1.97 (1.45–2.91)

Abbreviation: BUN, blood urea nitrogen.
[a]Whole blood.
[b]Corrected for albumin.

Metabolic alkalosis is characterized by alkalemia with an increased serum bicarbonate and compensatory elevation of PCO_2. Alkalosis begins with the loss of hydrogen ions from the body or the addition of alkali. Protracted vomiting causes the loss of H^+ and Cl^- and the gain of HCO_3^-. Respiratory compensation occurs through hypoventilation, resulting in an elevation in PCO_2 that buffers the rise in pH. The process is maintained by the effect of Na^+, K^+, and water loss on the kidney and is therefore treated by normal saline volume repletion and correction of hypokalemia, in addition to correcting the cause of vomiting.[1] We present a case of metabolic alkalosis caused by induced and protracted vomiting in pregnancy associated with nonreassuring fetal status, which prompted an indicated preterm delivery. Metabolic alkalosis in both the mother and newborn was a complicating factor managed to optimize recovery.

Case Presentation

A 21-year-old gravida 3, para 0 presented to the emergency room at 32 weeks' gestation with lethargy, nausea, and vomiting over a 3-day period. Early in the pregnancy, the patient had multiple admissions for hyperemesis gravidarum. The process did not resolve with time, and her pregnancy was complicated by a total of 10 hospital admissions for vomiting with volume depletion and electrolyte disturbances. During one of her latter admissions, the patient was observed rapidly drinking large volumes of water, which was subsequently followed by vomiting all the water ingested. She was under the misconception that this action would relieve heartburn, had already trialed usual remedies for gastroesophageal reflux, and received both gastroenterology and psychiatry consultations. Despite this education, 2 days prior to this admission, the patient reported that she had consumed large quantities of water resulting in vomiting.

Admission chemistry (►**Table 1**) showed hyponatremia, hypokalemia, severe hypochloremia, elevated serum bicarbonate, elevated blood urea nitrogen (BUN), and elevated creatinine, all consistent with protracted vomiting resulting in severe electrolyte disturbance, volume contraction, and acute kidney injury. There was hyperphosphatemia reflecting renal insufficiency and low ionized calcium secondary to metabolic alkalosis. Fetal heart rate monitoring showed normal rate with minimal variability and intermittent decelerations prompting a biophysical profile that scored only two points for normal amniotic fluid. A 1,610-g male newborn was delivered by urgent cesarean section with Apgar scores of 4 and 6 at 1 and 5 minutes, respectively. Umbilical cord gases along with reference values[2,3] are provided in ►**Table 2** and showed a metabolic alkalosis with elevated serum bicarbonate levels, hypercapnia, and base excess. The mother remained somnolent after delivery under spinal anesthesia, was transferred to the intensive care unit, and her venous blood gas (►**Table 2**) in conjunction with her serum electrolytes showed a hypochloremic metabolic alkalosis with compensatory hypoventilation as the cause of fetal/newborn metabolic disturbance.

The mother was treated with normal saline replacement, intravenous potassium chloride and calcium gluconate as her acute kidney injury was secondary to hypovolemia. Bilevel positive airway pressure (BiPap) ventilation was used to correct her hypercapnia and improve her mental status. The BiPap was discontinued after 24 hours with return to normal consciousness. Adequate urine output was maintained, and by postoperative day 3, serum sodium, potassium, chloride, bicarbonate, ionized calcium, phosphate, BUN, and creatinine all returned to normal limits. The patient was discharged on postoperative day 4.

The newborn's course began with poor respiratory effort requiring continuous positive airway pressure (CPAP) and

Table 2 Umbilical cord and maternal blood gas results with reference values in parentheses

	Umbilical cord blood gas		Maternal blood gas[a]
	Venous	Arterial	Venous
pH	7.36 (7.35 ± 0.05)	7.36 (7.28 ± 0.05)	7.45 (7.33–7.43)
pCO_2 (kPa)	11.84 (5.08 ± 0.67)	12.64 (5.35 ± 1.12)	9.71 (5.45–6.78)
pO_2 (kPa)	0.93 (3.88 ± 0.52)	0.93 (2.39 ± 0.82)	5.19
HCO_3^- (mmol/L)	50.3 (20.4 ± 4.1)	53.7 (22.3 ± 2.5)	50.7 (24–28)
Base excess (mmol/L)	19.9 (−4.0 ± 2.0)	23.0 (−4.0 ± 2.0)	23.1

[a]Collected 2 hours postpartum.

40% fraction of inspired oxygen (FiO_2). The newborn's electrolyte, acid–base, and serum chemistry pattern mirrored the mother's hypochloremic metabolic alkalosis (►**Table 1**). He received surfactant but required intubation for persistent oxygen requirement and hypercapnia. In addition, 3% saline was used to correct hyponatremia; potassium chloride and calcium gluconate boluses were used to correct hypokalemia and hypocalcemia. He maintained good urine output, and by day 4 of life, metabolic abnormalities corrected to normal, and he was extubated and maintained on CPAP. He was eventually discharged on day 78 of life with bronchopulmonary dysplasia that required 0.5 L/min 100% FiO_2 via nasal cannula.

Discussion

This case demonstrates the development of metabolic alkalosis in a pregnant woman caused by protracted vomiting with volume depletion and loss of sodium, hydrogen, and chloride severe enough to prompt preterm delivery for nonreassuring fetal status. It also demonstrates the dependence of the fetus on both the placenta and mother to maintain physiologic acid–base and electrolyte balance. The fetal kidney primarily maintains amniotic fluid volume and blood pressure.[4] Creatinine levels in newborns likewise reflect that of the mother.[5] Since the mother had metabolic alkalosis that was not corrected and the fetus required urgent delivery, it became a problem for the newborn as well. In both the mother and newborn, the process was managed by fluid and electrolyte restoration with appropriate ventilation to counteract the maladaptive compensatory hypoventilation, allowing gradual renal correction of the alkalosis in mother and newborn.

Conclusion

Metabolic alkalosis of the newborn resulting from maternal metabolic alkalosis has been described in a handful of cases in the literature.[6–8] These cases were believed to occur in mothers with a known or suspected eating disorder. Metabolic alkalosis appears to come to clinical attention in infants because the compensatory respiratory acidosis leads to hypoventilation and oxygen desaturation. In our case, the indication for delivery at 32 weeks' gestation was the nonreassuring fetal status, likely related to maternal hypovolemia and hypercapnia. The metabolic alkalosis of both the mother and fetus was apparent on the umbilical cord blood gases and treated similarly with assisted ventilation, replacement of serum sodium, potassium, and chloride resulting in resolution of metabolic abnormalities within 3 days. Whether or not vomiting in pregnancy is related to an eating disorder, vomiting in pregnancy is common, and the potential for metabolic alkalosis may adversely affect the fetus or newborn. Metabolic alkalosis in a newborn should be considered in newborns with oxygen desaturation and hypoventilation. Schimert et al used the term "transplacental metabolic alkalosis" to describe their case report of a newborn with metabolic alkalosis, and this term succinctly describes our case as well.[6]

Conflict of Interes
None declared.

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