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Pediatrics 2010;125:e973-e977; originally published online Mar 15, 2010;

DOI: 10.1542/peds.2009-1906

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<http://www.pediatrics.org/cgi/content/full/125/4/e973>

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American Academy of Pediatrics

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Fresh Goat's Milk for Infants: Myths and Realities—A Review

AUTHORS: Sangita Basnet, MD, FAAP,^a Michael Schneider, MD,^a Avihu Gazit, MD,^b Gurpreet Mander, MD, FAAP,^a and Allan Doctor, MD^b

^aDepartment of Pediatrics, Southern Illinois University School of Medicine, Springfield, Illinois; and ^bDepartment of Pediatrics, Washington University School of Medicine, St Louis, Missouri

KEY WORDS

goat's milk, infant feeding, hypernatremia

ABBREVIATION

G-tube—gastrostomy tube

www.pediatrics.org/cgi/doi/10.1542/peds.2009-1906

doi:10.1542/peds.2009-1906

Accepted for publication Dec 15, 2009

Address correspondence to Sangita Basnet, MD, FAAP, Southern Illinois University School of Medicine, Department of Pediatrics, Division of Pediatric Critical Care, PO Box 19676, Springfield, IL 62794-9676. E-mail: sbasnet@siumed.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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FINANCIAL DISCLOSURE: *The authors have indicated they have no financial relationships relevant to this article to disclose.*

abstract

Many infants are exclusively fed unmodified goat's milk as a result of cultural beliefs as well as exposure to false online information. Anecdotal reports have described a host of morbidities associated with that practice, including severe electrolyte abnormalities, metabolic acidosis, megaloblastic anemia, allergic reactions including life-threatening anaphylactic shock, hemolytic uremic syndrome, and infections. We describe here an infant who was fed raw goat's milk and sustained intracranial infarctions in the setting of severe azotemia and hypernatremia, and we provide a comprehensive review of the consequences associated with this dangerous practice. *Pediatrics* 2010;125:e973–e977

The developed world does not lack nutritious food; nevertheless, infants may still suffer from inadequate and inappropriate nutrition because of parental and cultural beliefs. Furthermore, easy access to the Internet exposes women to false information in regards to alternative foods for their infants, such as raw goat's milk, that may cause severe morbidity and even death. We describe here the case of an infant with severe electrolyte imbalance, renal dysfunction, and stroke as a result of being fed goat's milk.

CASE REPORT

A 5-month-old white boy with CHARGE (coloboma, heart defect, atresia choanae, retarded growth and development, genital hypoplasia, ear anomalies/deafness) association from an Amish family was admitted to the PICU from an outside emergency department after presenting with respiratory failure that required endotracheal tube placement and mechanical ventilation. He had a 1-day history of increased work of breathing and depressed level of consciousness that was preceded by a 4-day history of diarrhea. His past medical history was significant for tracheoesophageal fistula repair with gastrostomy tube (G-tube) placement, multiple esophageal dilatations, and repair of choanal atresia. He also had a history of atrial and ventricular septal defects. In the neonatal period, because of parental consanguinity, testing for plasma amino acids and urine organic acids and the state-expanded tandem mass spectroscopy screen were performed; no abnormalities were found. There had been no previous episodes of acidosis or hyponatremia.

The infant's respiratory distress seemed principally to be the result of severe metabolic acidosis with respiratory compensation. A comprehensive metabolic panel revealed aci-

demia, severe hyponatremia, and azotemia, with significant hyperosmolality. He had hyperchloremia, hyperphosphatemia, hyperuricemia, and an elevated creatinine kinase level. Results of lactic acid and liver function tests were within normal limits. Urinalysis was significant for proteinuria, hematuria, and glucosuria. A complete blood count showed leukocytosis with left shift but was otherwise unremarkable. Abnormal initial serum laboratory values are shown in Table 1.

On arrival to the PICU, the infant was pharmacologically sedated and on mechanical ventilation with a hemodynamic profile that was normal for his age. His respiratory rate was in the 80 breaths per minute range. His capillary refill time was prolonged. Results of his chest radiograph were normal except for minimal right perihilar infiltrates. An echocardiogram on admission revealed normal segmental anatomy and left ventricular systolic function, a small patent foramen ovale, and tiny atrial and ventricular septal defects.

Nutritional history revealed that the infant was initially fed breast milk through a G-tube. However, for 3 to 4

weeks before admission, he had been exclusively fed raw goat's milk because his mother was unable to pump sufficient volume.

The infant's hyponatremia and dehydration were corrected slowly over 96 hours to reduce the risk of cerebral edema and central pontine or extrapontine myelinolysis, which can occur rarely.¹⁻³ He required substantial amounts of intravenous bicarbonate to correct the metabolic acidosis, with his serum level normalizing after 4 days of replacement. He also required intravenous boluses and intermittent supplementation of calcium, magnesium, potassium, and albumin. He was started on low-sodium, low-protein formula through his G-tube on the second hospital day. His serum amino acids, urine organic acids, carnitine profile, ammonia levels, and lactate level were not diagnostic of an inborn error of intermediary metabolism; the acidosis quickly corrected and has not recurred.

Renal ultrasound showed normal architecture and anatomy and demonstrated a normal Doppler flow signal in the renal veins. A renal duplex scan showed no evidence of renal artery occlusive disease and normal intrarenal vascular perfusion. The ongoing losses of electrolytes and bicarbonate were attributed to acute tubular necrosis. The infant's serum urea nitrogen and creatinine slowly normalized.

MRI of his brain showed acute and subacute infarcts within the left posterior cerebral artery territory and right temporo-occipital periventricular white matter and chronic infarctions involving bilateral occipital lobes (Fig 1). There was no history of seizures or neurologic deficits, and there was no seizure activity noted during hospitalization. A hypercoagulability workup, including protein C, protein S, and antithrombin levels, was normal. The

TABLE 1 Initial Serum Laboratory Values at Presentation

Blood Test	Result	Reference Range
pH (arterial)	6.90	7.35–7.45
Pco ₂ , mm Hg	13	35–45
HCO ₃ , mmol/L	3	18–23
Sodium, mmol/L	176	135–147
Chloride, mmol/L	154	96–107
Anion gap	18	4–16
Measured osmolality, mOsm/kg	384	262–286
Serum urea nitrogen, mg/dL	112	10–25
Creatinine, mg/dL	2.1	0.7–1.3
Calcium, mg/dL	7.2	9–11
Ionized calcium, mmol/L	1.35	1.10–1.30
Glucose, mg/dL	132	70–109
Uric acid, mg/dL	13.5	3.4–7.4
Creatinine kinase, U/L	1000	0–200
Phosphorus, mg/dL	9.6	3.5–6.7

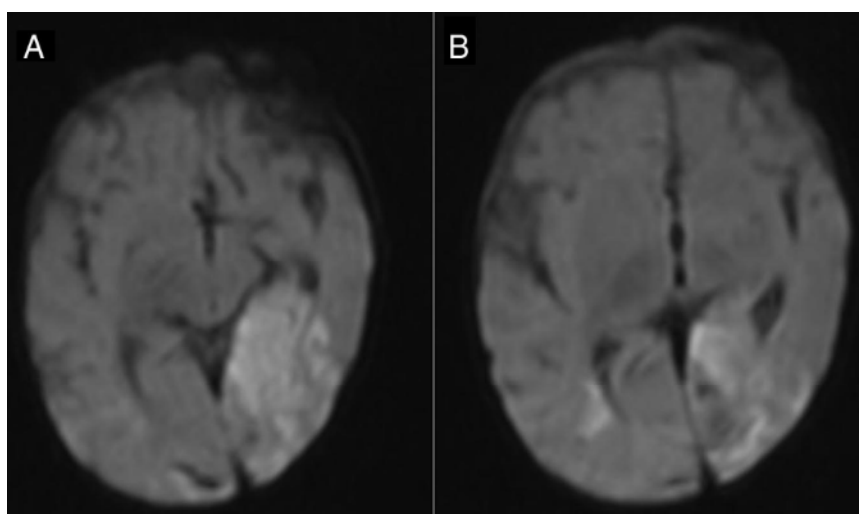


FIGURE 1

Acute stroke: restricted diffusion (bright-white signal) of the left occipital lobe (A) and the white matter along the occipital horn of the right lateral ventricle (B).

strokes were attributed, therefore, to severe hypernatremic dehydration.

The infant had severe respiratory distress on extubation, was diagnosed with having severe tracheomalacia, and underwent tracheostomy. Cardiac angiography demonstrated a vascular ring that was subsequently repaired. The infant was discharged from the hospital with a tracheostomy and mechanical ventilatory assistance. However, he is gaining weight on regular infant formula, and his electrolyte levels have normalized without the need for supplementation.

DISCUSSION

The first case in the literature to report concerns with goat's milk feeding in infants described a 7-month-old boy who had been fed for 6 months on goat's milk, weighed only 4 lb, and was thought to have died of malnutrition because "goat's milk is lighter than skimmed milk."⁴ The author ridiculed the physician who made the diagnosis saying that after cow's milk, "goat's milk closely approximates to that of a woman."

As a result of information technology, it is very easy for parents to read and

be influenced by false and potentially dangerous information. A Google search of the terms "goat's milk" and "infant" and "benefits" yielded 9490 hits; these pages provide information such as "[g]oat's milk is the ideal food for babies. . . . Beneficial for the treatment of asthma, eczema, migraines, stomach ulcers, liver complaints and chronic catarrh, goat's milk also helps babies with colic, habitual vomiting and those not gaining weight."⁵ This same site suggests that the first few feeds be given at half strength, increased to two thirds, then three quarters, reaching full strength in 2 to 3 days' time mixed with honey (another concerning recommendation).

The infant in this report presented with severe hypernatremia and azotemia in addition to other electrolyte abnormalities. Goat's milk contains 50 mg of sodium and 3.56 g of protein per 100 mL, approximately 3 times that in human milk (17 mg and 1.03 g per 100 mL, respectively).⁶ The estimated requirements of sodium and protein for infants <6 months old are 100 to 200 mg/day and 9 to 11 g/day, respectively.⁷ The infant described here was receiving ~500 mg/

day of sodium and 30 g/day of protein, with a total intake of 32 oz of goat's milk per day. The immature kidneys in very young infants have difficulty handling the byproducts of foods with a high renal solute load.⁸ Sodium excretion capacity matures more slowly than glomerular filtration rate and does not attain full capacity until the second year of life.⁹ Therefore, infants fed fresh goat's milk are at substantive risk for hypernatremia and azotemia, particularly in the face of dehydration (as in the case described here), which may in turn result in major central nervous system pathology, including diffuse encephalopathy, intraparenchymal hemorrhage, or thromboses¹⁰ as manifested in our patient.

Metabolic acidosis has been described in infants fed undiluted goat's milk.^{11–13} Our patient presented with severe metabolic acidosis with increased anion gap, which seemed out of proportion to the dehydration and hyperchloremia alone. The high protein content of goat's milk may have contributed to this problem. Excessive protein loading may result in accumulation of non-volatile acids and urea,^{14,15} and it has been shown that the incidence of metabolic acidosis increases with increase in dietary protein intake.¹⁶

The main benefit claimed by proponents of fresh goat's milk for infants is that it is less allergenic than cow's milk and is a suitable substitute for infants who are allergic to the latter. However, evidence shows that most infants who are allergic to cow's milk are also allergic to goat's milk. In vitro studies have shown that there is an extensive cross-reactivity of sera from individuals who are allergic to cow's milk with proteins found in goat's milk.^{17–19} In 1 study, 26 children with immunoglobulin E-mediated cow's milk allergy also had positive skin test responses to goat's milk, and 24 of 26 had positive double-blind, placebo-

controlled, oral food challenges with fresh goat's milk.²⁰ There have been case reports of severe life-threatening anaphylactic reactions after the ingestion of commercial goat's milk preparation in infants with documented cow's milk protein allergy.²¹ Furthermore, infants and young children may have signs, symptoms, and serology positive for goat's milk without being allergic to cow's milk.^{22–25} In a retrospective study, children presented with severe allergic reactions, including anaphylaxis, after consumption of goat's milk products but tolerated cow's milk products.²⁶

Folate deficiency with anemia in infants fed homemade formula based on goat's milk has been described.^{27,28} In fact, "goat's milk anemia" was the name given to the macrocytic hyperchromic megaloblastic anemia observed in infants fed goat's milk in Europe during the 1920s and 1930s.²⁹ The anemia was thought to be more severe than that associated with exclusive cow's milk feeding and was cured by giving supplements of liver extracts. The concentration of folate in goat's milk is 6 µg/L in comparison to human

breast milk, which contains 50 µg/L.³⁰ Infants younger than 6 months of age need 65 µg/day of folate, and the recommended daily allowance increases with age.³⁰

There have been reports of infections such as Q fever, toxoplasmosis, and brucellosis associated with feeding raw goat's milk.^{31–33} Consumption of unpasteurized goat's milk has also been implicated in the development of *Escherichia coli* O157:H7-associated hemolytic uremic syndrome.^{34,35} Although raw goat's milk is a proven vehicle for pathogen transmission, the belief persists that raw dairy products are healthier and that pasteurized products are less beneficial and even harmful.⁵

Although infants should not be fed unmodified, raw goat's milk, goat's milk infant formula may be a suitable alternative to cow's milk formula. A study performed in New Zealand showed that there was no difference in weight gain between healthy neonates fed either formulas.³⁶ Sixty-two infants were randomly assigned to either goat's milk formula or cow's milk formula from within 72 hours of birth until

168 days of age. There was no statistically significant difference in average weight gain in the goat's milk formula group versus the cow's milk formula group (309 g [95% confidence interval: –49 to 668]). Furthermore, although infants fed goat's milk formula had higher bowel motion frequency (2.4 vs 1.7 bowel motions per day), both formulas resulted in similar bowel motion consistency and periods of crying and were deemed safe and well tolerated. However, the authors cautioned feeding it to infants with documented allergy to cow's milk infant formula.

CONCLUSIONS

An exclusive, unmodified goat's milk diet can cause significant morbidity and even mortality in infants, including electrolyte imbalances, metabolic acidosis, folate deficiency, and species-specific and nonspecific antigenicity. Unpasteurized goat milk has its additional infectious risks. However, information supporting this practice abounds on the Internet and in specific cultures. Our case report and literature review support the need to strongly advocate against this practice.

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