A 5-week-Old Boy with Failure to Thrive, Marked Hyperkalemia and Hyponatremia

Min Xu,1,2* Carolina Di Blasi,3,4 Jane Dickerson,1,2 Rhona Jack,1,2 and Joe C. Rutledge1,2

1 Department of Laboratories, Seattle Children's Hospital, Seattle, WA; 2 Department of Laboratory Medicine, University of Washington, Seattle, WA; 3 Department of Endocrinology, Seattle Children's Hospital, Seattle, WA; 4 Department of Pediatrics, University of Washington, Seattle, WA.

* Address correspondence to this author at: Department of Laboratories, OC.8.731, Seattle Children's Hospital, 4800 Sand Point Way NE, Seattle, WA 98105. Fax 206-987-3840; e-mail min.xu@seattlechildrens.org.

CASE DESCRIPTION

A 5-week-old Caucasian boy presented to the emergency department after referral from his primary care physician with concerns for failure to thrive.

The patient's birth weight was 2892 grams at 37 weeks of gestation (10^{th} percentile). He was 2665 grams at 5 weeks of age ($<2^{nd}$ percentile). He had been exclusively breastfed for the first 3 weeks of his life. At 3 weeks of age, he was introduced to a bottle with expressed breast milk, but showed little interest in the bottle. At a well child check-up at 4 weeks of age, his weight was down, and formula supplementation was recommended. In the last 3 days before the admission, he had been lethargic and had difficulty feeding. He had two episodes of projectile vomiting during that week. There was a noticeable reduction in the amount of stool and urine output during the last two days before admission without fever or diarrhea.

The patient was born at 37 weeks of gestation by spontaneous vaginal delivery. Mother was found to be group B streptococcus (GBS) positive but was not treated prior to delivery, so the patient was monitored for 48 hours in the hospital before discharged home. Mother also had a history of bilateral vesicoureteral reflux and reimplantation surgery. There was no other history of genital, urinary or kidney abnormalities.

On exam he was cachectic and lethargic but did have warm extremities, brisk capillary refill (<2 seconds) and good distal pulses. No skin hyperpigmentation was seen and the genitalia were normal. Laboratory tests were significant for potassium (K) 10.2 mmol/L, sodium (Na) 116 mmol/L, chloride (CL) 91 mmol/L, BUN 64 mg/dL (22.9 mmol/L), creatinine 0.7 mg/dL (61.9 µmol/L), and glucose 88 mg/dL (4.9 mmol/L), pH 7.29, pCO_2 29 mmHg, total CO_2 15.0 mmol/L (**Table 1**). The specimen was not hemolyzed. Contamination of the specimen with K was originally suspected by the medical technologist. Communication with clinical staff confirmed the quality of the specimen. Electrocardiogram showed no electrolyte-related changes. His blood pressure was 75/41 mmHg (normal interval 65-85/45-55 mmHg). The patient received normal saline boluses to supplement Na, kayexalate to chelate K, and calcium gluconate to antagonize the membrane actions of hyperkalemia. Although he had 2 normal newborn screens, the possibility of congenital adrenal hyperplasia (CAH) was still in the differential diagnoses owing to such high K and low Na. He was started on stress dose steroids after measurement of baseline adrenocorticotropic hormone (ACTH), renin, aldosterone, 17-hydroxyprogesterone (17-OHP), and cortisol. Urinalysis for the possibility of urinary tract infection (UTI) was also performed. One hour later, Na increased to 125 mmol/L and K decreased to 7.9 mmol/L. He was admitted to the neonatal intensive care unit for further evaluation and management.

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Table 1. Relevant laboratory results during inpatient period.

Test	At admission	1 h after admission	4 h after admission	20 h after admission	Day of discharge, 1 week after admission	Reference interval
Sodium, mmol/L	119 C[a]	125 L	125 L	137	141	135-145
Potassium, mmol/L	10.2 C	7.9 C	6.8 C	5.0	5.0	3.4-5.6
Chloride, mmol/L	93 L		101	110	108	96-110
Carbon dioxide, mmol/L		15 L	12 L	13 L	26	18-27
Anion gap, mmol/L[b]			18.8	19	12	8-22
Glucose, mg/dL	88 (4.9 mmol/L)	71 (3.9 mmol/L)		68 (3.8 mmol/L)	89 (4.9 mmol/L)	60-105 (3.3-6.0 mmol/L)
BUN, mg/dL	64 H (22.9 mmol/L)		49 H (17.5 mmol/L)	24 H (8.6 mmol/L)	<4 L (<1.4 mmol/L)	6-20 (2.1-7.1 mmol/L)
Creatinine, mg/dL	0.7 H (61.9 μmol/L)		0.6 (53.1 μmol/L)	0.4 (35.4 μmol/L)	0.4 (35.4 μmol/L)	0.1-0.6 (8.8-53.1 μmol/L)
pH			7.29 L			7.32-7.42
Pco_2, venous, mmHg			29 L			41-51
Cortisol, random, μg/dL	38.3 H (1057 nmol/L)					4.5-23 (124-635 nmol/L)
17-OHP, ng/dL	29 (0.88 nmol/L)					11-170 (0.33-5.1 nmol/L)

[a] Flags: C, critical; L, low; H, high.
[b] Calculated as (sodium and potassium) − (chloride and CO_2).

QUESTIONS TO CONSIDER
• What is the differential diagnosis of a patient with marked hyperkalemia, hyponatremia and metabolic acidosis?
• What are the diagnostic tests for CAH?
• What are the consequences of neonatal infection of GBS?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the November 2016 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol62/issue11 and follow the link to the Clinical Case Study and Commentaries.

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