
An Unusual Cause of Metabolic Alkalosis and Hypocalcemia in Childhood

Tony Huynh^{1,2,3*} and Urs Wilgen^{2,3}

1 Department of Endocrinology and Diabetes, Lady Cilento Children's Hospital, South Brisbane, Queensland, Australia; 2 Department of Chemical Pathology, Pathology Queensland, Herston, Queensland, Australia; 3 Faculty of Medicine, The University of Queensland, St. Lucia, Queensland, Australia.

* Address correspondence to this author at: Department of Endocrinology and Diabetes, Queensland Children's Hospital, South Brisbane QLD 4101, Australia. Fax +61-7-33196492; e-mail tony.huynh@uq.edu.au

CASE DESCRIPTION

An 8-year-old boy presented to the emergency department with complaints of intermittent stiffening of his upper and lower limbs. Shortly after his arrival he had a large volume (approximately 500 mL) of nonbilious projectile vomit. He described general weakness and fatigue the previous night and on waking, with no history of headaches, diarrhea, fevers, or other symptoms of infection.

In the past 4 months he had experienced intermittent generalized abdominal pain, nausea, anorexia, vomiting, and mild weight loss (3 kg). He had presented 2 months earlier with vomiting. Investigations including a complete blood count, electrolytes, liver function tests, albumin-corrected calcium, magnesium, and phosphate were within reference intervals at that time. An abdominal ultrasonography was unremarkable. The episodes of vomiting had become increasingly more frequent and forceful. His past medical history was otherwise unremarkable.

On presentation he was afebrile but tachycardic (pulse rate 156 bpm), tachypneic (respiratory rate 32/min), and mildly hypertensive (bp 125/78 mmHg). He was intermittently drowsy but easily rousable. He was described as having "locked hands and curled toes" consistent with carpopedal spasms and twitching movements of his tongue. He had no focal neurological signs, and his power and reflexes were essentially normal. There were no cardiac murmurs, and his respiratory examination was unremarkable. His abdomen was soft and nontender, with normal bowel sounds. Biochemical testing on presentation showed hypokalemia, hypochloremia, and hyperbicarbonatemia (Table 1). He had evidence of dehydration and prerenal azotemia (increased urea, creatinine, and urate) and normal total protein and albumin. His liver function tests, lipase, thyroid function tests, albumin-corrected calcium, phosphate, and magnesium were normal. An electrocardiogram revealed a prolonged corrected QT interval (QTc 480 ms). Despite the normal albumin-corrected calcium, the presence of carpopedal spasms and the prolonged QTc interval prompted the collection of a

venous blood gas, which revealed hypocalcemia [ionized calcium of 0.90 mmol/L (reference interval, 1.15–1.32 mmol/L)], a metabolic alkalosis [pH 7.53 (reference interval, 7.32–7.43), bicarbonate 36 mmol/L (reference interval, 17–31 mmol/L)], and PCO₂ of 43 mmHg (reference interval, 38–54 mmHg). In the setting of metabolic alkalosis, the respiratory drive is typically reduced as part of the process of respiratory compensation, with the rise in PCO₂ blunting the rise in pH. The presence of tachypnea in this patient was thought to be due to hyperventilation related to anxiety and distress and potentially may have further worsened the alkalosis and ionized hypocalcemia, leading to acute symptoms.

Table 1. Biochemical investigations at presentation.

Test	Result	Reference interval
Venous blood gas		
pH	7.53	7.32-7.43
PCO ₂	43 (5.7)	38-54 mmHg (5.1-7.2 kPa)
PO ₂	50 (6.6)	30-50 mmHg (4.0-6.6 kPa)
Bicarbonate	36	17-31 mmol/L
Base excess	12.0	-2.0 to 3.0
Sodium	132	133-144 mmol/L
Potassium	2.8	3.3-4.9 mmol/L
Chloride	89	97-110 mmol/L
Anion gap	7	4-13 mmol/L
Creatinine	1.0 (88)	0.3-0.7 mg/dL (27-58 μmol/L)
Ionized calcium	0.90	1.15-1.32 mmol/L
Glucose	128 (7.1)	54-140 mg/dL (3.0-7.8 mmol/L)
Lactate	2.7	0.5-2.2 mmol/L
Serum and urine biochemistry		
Albumin-corrected calcium	8.8 (2.21)	8.8-10.6 mg/dL (2.20-2.65 mmol/L)
Phosphate	6.2 (2.00)	2.8-6.2 mg/dL (0.90-2.00 mmol/L)
Magnesium	1.92 (0.79)	1.58-2.68 mg/dL (0.65-1.10 mmol/L)
Parathyroid hormone	209 (22)	10-66 pg/mL (1.0-7.0 pmol/L)
25-hydroxyvitamin D	26 (66)	20-60 ng/mL (50-150 nmol/L)
1,25-dihydroxyvitamin D	56 (134)	20-79 pg/mL (48-190 pmol/L)
Urine sodium	94	mmol/L
Urine potassium	253	mmol/L
Urine chloride	17	mmol/L
Urine calcium	1.6 (0.4)	mg/dL (mmol/L)
Urine phosphate	136 (43.9)	mg/dL (mmol/L)

QUESTIONS TO CONSIDER

- What are some of the common causes of metabolic alkalosis in pediatric populations?
- What is the clinical significance of urinary chloride concentration in distinguishing among the causes of metabolic acidosis?
- What is the effect of pH change on the total and ionized calcium concentrations?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the April 2019 issue of *Clinical Chemistry*. To view the case and comments online, go to <http://www.clinchem.org/content/vol65/issue4> and follow the link to the Clinical Case Study and Commentaries.

Educational Centers

If you are associated with an educational center and would like to receive the cases and questions 1 month in advance of publication, please email clinchemed@aacc.org.

All previous Clinical Case Studies can be accessed and downloaded online at <https://www.aacc.org/publications/clinical-chemistry/clinical-case-studies>

AACC is pleased to allow free reproduction and distribution of this Clinical Case Study for personal or classroom discussion use. When photocopying, please make sure the DOI and copyright notice appear on each copy.

AACC is a leading professional society dedicated to improving healthcare through laboratory medicine. Its nearly 10,000 members are clinical laboratory professionals, physicians, research scientists, and others involved in developing tests and directing laboratory operations. AACC brings this community together with programs that advance knowledge, expertise, and innovation. AACC is best known for the respected scientific journal, *Clinical Chemistry*, the award-winning patient-centered web site *Lab Tests Online*, and the world's largest conference on laboratory medicine and technology. Through these and other programs, AACC advances laboratory medicine and the quality of patient care.