Clinical Case Study

Fatal Electrolyte Abnormalities Following Enema Administration

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CASE

A 90-year-old man was admitted to the Emergency Department with severe congestive heart failure and kidney failure. His medical history included successful surgery for prostatic carcinoma, aortocoronary bypass surgery, and cardiac pacemaker implantation. The patient was regularly taking digoxin, enalapril, aspirin, transdermal nitrate, and furosemide. At admission, the patient was hyperkalemic (Table 1), and therapy with sodium polystyrene sulfonate and ethacrynic acid was started immediately. The diuretic therapy was continued after his admission to the Nephrology and Dialysis Department on the second day of hospitalization. An ultrasound examination in which the kidneys appeared small and hyperechoic confirmed chronic renal impairment. On the evening of the sixth day of hospitalization, 2 enemas (120 mL each) were administrated 30 min apart to relieve prolonged constipation. The patient vomited the following night, and a nasogastric tube was inserted. The next morning, the patient showed signs of dehydration and was hypotensive (blood pressure, 90/40 mmHg). The patient's abdominal distension prompted an abdominal radiograph, which showed signs of intestinal obstruction. Laboratory findings revealed severe hypocalcemia [3.7 mg/dL (0.93 mmol/L); reference interval, 8.5–10.5 mg/dL (2.13–2.63 mmol/L)] and alterations in the plasma concentrations of other major plasma ions (Table 1). An intravenous infusion of calcium gluconate was started immediately, and an abdominal computed tomography evaluation was requested. The severe electrolyte abnormalities were confirmed after analysis of a second blood sample drawn after 1.5 h, which revealed severe hyperphosphatemia [30.0 mg/dL (9.69 mmol/L); reference interval, 2.5–4.5 mg/dL (0.81–1.45 mmol/L)] without signs of overt acidosis. In the meantime, the abdominal computed tomography scan revealed paralytic ileus. Emergent hemodialysis was planned, but despite intensive treatment, the patient's electrocardiogram showed an increased QT interval. He finally went into cardiac arrest and died before hemodialysis could begin.

Questions to Consider
• Which laboratory tests are useful in the evaluation of a patient with severe hypocalcemia?
• What are several causes of severe hypophosphatemia?
• What treatment should be used in patients with severe hypophosphatemia and hypocalcemia?

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Table 1. Laboratory findings for the patient during hospitalization. The enemas were given on the evening of day 6.[a]

Test	Reference interval	Day 1 (ED)[b]	Day 2 (ED)	Day 3 (Dialysis)	Day 4 (Dialysis)	Day 7 (10:00 AM, Dialysis)	Day 7 (11:30 AM, Dialysis)
Creatinine (P), mg/dL	0.7–1.2	**5.57**	**5.35**	**4.92**	**4.59**	**3.69**	
Urea (P), mg/dL	19–50	**300**	**306**	**318**	**304**	**223**	
Uric acid (P), mg/dL	2.5–7.0				**17.3**		
Sodium (P), mmol/L	135–145	**132**	136	138	139	**153**	**155**
Potassium (P), mmol/L	3.5–5.0	**7.7**	**5.3**	5.0	3.8	**2.7**	**2.9**
Chloride (P), mmol/L	98–110	**93**	**96**	99	**97**	102	102
Bicarbonate (P), mmol/L	22–29	23				23	23
pH	7.35–7.45	7.43				7.36	7.36
Total calcium (P), mg/dL	8.5–10.5		9.6	9.5	9.4	**3.7**	**3.1**
Albumin (P), g/L	35–50				**33**		
Ionized calcium (B), mg/dL[c]	4.64–5.28	**4.24**				**1.28**	**1.60**
Phosphate (P), mg/dL	2.5–4.5				**5.9**		**30.0**
Magnesium (P), mg/dL	1.4–2.4						**1.8**
C-reactive protein (P), mg/L	<10	2.0	**20.9**	**21.4**	**17.8**	**76.3**	**84.7**
Hemoglobin (B), g/L	130–175	**95**	**88**			**90**	
Leukocytes (B), ×103/μL	4.00–10.00	6.14	7.09			**14.00**	
Neutrophils (B), ×103/μL	2.00–8.00					**11.83**	

[a] Abnormal results are in boldface. Conversion factors: calcium: mg/dL × 0.25 = mmol/L; creatinine: mg/dL × 88.4 = μmol/L; magnesium: mg/dL × 0.411 = mmol/L; phosphate: mg/dL × 0.323 = mmol/L; urea: mg/dL × 0.166 = mmol/L; uric acid: mg/dL × 59.48 = μmol/L.
[b] ED, Emergency Department; Dialysis, Nephrology and Dialysis Department; P, plasma; B, whole blood.
[c] Normalized to pH 7.4.

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the November 2012 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol58/issue11 and follow the link to the Clinical Case Study and Commentaries.

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