

CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

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Case 7-2006: A 47-Year-Old Man with Altered Mental Status and Acute Renal Failure

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PRESENTATION OF CASE

A 47-year-old man was transferred to the emergency department of this hospital at 10 a.m. on a day in early June because of altered mental status and acute renal failure. At approximately 8 p.m. the previous evening, the patient had been behaving normally at dinner with his family. After dinner, he went outside to work on his car. According to his wife, when he later returned to the house, his speech was slurred and he was lethargic. By 9 p.m., the patient was vomiting and becoming increasingly lethargic. He went to bed, and at 3:30 a.m., his wife found him unresponsive. She called emergency medical services, and the patient was taken to the emergency department of a local hospital.

The patient had not had recent fevers, illnesses, or depressive symptoms. He had irritable bowel syndrome (for which he took atropine and diphenoxylate), chronic back pain, and anxiety. He was allergic to penicillin. There was no personal or family history of major medical problems. He had a 30 pack-year history of smoking and a history of alcohol abuse, but he had not consumed alcohol in the past year. His wife did not believe that he used illicit drugs. There were no empty pill bottles found in the house, and the patient did not have known access to other prescription medications.

On arrival in the emergency department, the patient was somnolent and unable to follow commands; the blood pressure was 85/40 mm Hg, and the heart rate 75 beats per minute. His mental status worsened, and the trachea was intubated for airway protection with use of rapid-sequence induction with 20 mg of etomidate and 120 mg of succinylcholine. A chest radiograph showed an infiltrate in the left lower lobe that was consistent with pneumonia. A computed tomographic (CT) scan of the head revealed no abnormalities except for an air-fluid level in the left mastoid air cells that was suggestive of mastoiditis. An orogastric tube was placed, and 50 g of activated charcoal was given; in addition, 900 mg of clindamycin and 500 mg of metronidazole were given intravenously for possible aspiration pneumonia. After endotracheal intubation, the arterial pH was 6.97, the partial pressure of oxygen 182 mm Hg, and the partial pressure of carbon dioxide 34 mm Hg; the level of carbon monoxide was undetectable. The creatinine level was 3.8 mg per deciliter (336 μ mol per liter), and the white-cell count was 30,000 per cubic millimeter. A continuous intravenous infusion of sodium bicarbonate (150 mmol per liter) in a

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solution of 5 percent dextrose in water was started at a rate of 250 ml per hour, and the patient was transferred to the emergency department of this hospital.

On arrival at this hospital, the patient was intubated and sedated and unresponsive to painful stimuli. The patient's blood pressure was 137/88 mm Hg, the heart rate 80 beats per minute, and the temperature 36.2°C; he was ventilated at a rate of 30 breaths per minute with an oxygen saturation of 99 percent and a fraction of inspired oxygen of 1.0. His pupils were equal, round, and reactive at 3 mm. The corneal reflex was present, and the vestibulo-ocular reflex was absent. An endotracheal tube was in place. The neck was supple without lymphadenopathy. Rhonchi were present bilaterally. Cardiac auscultation revealed no abnormalities. The abdomen was soft, with active bowel sounds. A Foley catheter was draining clear urine. The rest of the examination was normal.

The placement of the endotracheal tube was confirmed by bedside end-tidal carbon dioxide calorimetry. A central venous pressure line was placed in the right internal jugular vein, and in the process a transient run of ventricular tachycardia occurred, which terminated with retraction of the guidewire. A chest radiograph obtained with a portable device showed no pneumothorax and a small left perihilar opacity. The central venous pressure was 10 to 13 mm Hg. The white-cell count was 27,000 per cubic millimeter, with 95 percent neutrophils; the rest of the complete blood count was normal. The results of other laboratory tests are shown in Table 1. After a review of the CT scan obtained at the first hospital, vancomycin (1 g) and ceftriaxone (2 g) were administered intravenously for empirical treatment of possible meningitis. An electrocardiogram showed normal sinus rhythm without marked prolongation of the QRS complex or the QT interval.

Table 1. Results of Laboratory Tests.*

Variable	Normal Range	Day 1				Day 2	Day 8	Day 20
		1 hr, 4 min after Admission	1 hr, 9 min after Admission	5 hr, 48 min after Admission	12 hr, 35 min after Admission			
Sodium (mmol/liter)	135–145	147	149	153	140	137	136	142
Potassium (mmol/liter)	3.4–4.8	4.1	2.4	2.6	4.8	3.8	5.2	5.1
Chloride (mmol/liter)	100–108	110			107	100	103	108
Carbon dioxide (mmol/liter)	23–31.9	13.6			25.3	31.5	17.8	26.8
Urea nitrogen (mg/dl)	8–25	23			17	15	96	56
Creatinine (mg/dl)	0.6–1.5	2.8			2.7	2.6	12.6	3.6
Glucose (mg/dl)	70–110	208	141	99	163	130	115	90
Calcium (mg/dl)	8.5–10.5	7.6			8.8	8.0	8.6	9.1
Calcium, ionized (mmol/liter)	1.14–1.30		0.74	0.55	1.22			
Phosphorus (mg/dl)	2.6–4.5	4.3			2.8	1.1	7.0	5.7
Magnesium (mEq/liter)	1.4–2.0	1.5			1.3	1.3	2.1	1.6
Fraction of inspired oxygen (per liter)	No normal value			0.7	0.5			
Arterial pH	7.35–7.45		7.10	7.34	7.45			
Arterial partial pressure of carbon dioxide (mm Hg)	35–42		33	28	36			
Arterial partial pressure of oxygen (mm Hg)	80–100		139	234	105			
Osmolality (mOsm/kg)	280–296	321						

* To convert the values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the values for creatinine to micromoles per liter, multiply by 88.4. To convert the values for glucose to millimoles per liter, multiply by 0.5551. To convert the values for calcium to millimoles per liter, multiply by 0.250. To convert the values for phosphorus to millimoles per liter, multiply by 0.3229. To convert the values for magnesium to millimoles per liter, multiply by 0.500.

Approximately 30 minutes after his arrival at this hospital, the patient had a generalized tonic-clonic seizure. Lorazepam was administered intravenously every two minutes in doses of 2 mg up to a total of 20 mg. After 10 minutes of continuous seizure activity, phenytoin (1 g) was administered intravenously at a rate of 50 mg per minute. Administration of phenobarbital was in preparation when the seizure activity ceased at 20 minutes. The results of laboratory tests are shown in Table 1. The anion gap was calculated at 25 mmol per liter. The measured serum osmolality was 321 mOsm per kilogram, and the patient's initial osmolal gap was calculated at 8 mOsm per kilogram. Calcium gluconate (2 g), potassium chloride (1 liter of a 40-mmol solution in normal saline), and magnesium sulfate (2 g) were given. Toxicologic screening of serum was negative for ethanol, acetaminophen, and salicylates, and toxicologic screening of urine was also negative. Numerous needle-shaped crystals were seen in the urinary sediment.

Fomepizole (1 g; 15 mg per kilogram of body weight) was administered intravenously, and the nephrology division was consulted for immediate hemodialysis. A double-lumen hemodialysis catheter was placed in the right femoral vein, and hemodialysis was initiated. Results of laboratory tests obtained five hours after admission are shown in Table 1. The patient was admitted to the medical intensive care unit for further care.

DIFFERENTIAL DIAGNOSIS

Dr. J. Kimo Takayesu: I was involved in the case of this 47-year-old man, who had an acute onset of altered mental status, acute renal failure, and a severe metabolic acidosis associated with an anion gap. These findings initially gave rise to a broad differential diagnosis, which was quickly narrowed by the additional finding of typical crystals of calcium oxalate in a urine specimen.

THE ANION GAP

The anion gap is the difference between the measured concentrations of serum sodium and the total of the main serum anions chloride and bicarbonate (anion gap = Na - Cl + HCO₃); a normal anion gap is caused by the presence of negatively charged proteins that are not measured by serum analyzers, mainly albumin, and ranges from 3 to 11 mmol per liter.^{1,2} An elevated anion gap is

caused by either an excess of unmeasured serum anions in addition to albumin or a paucity of unmeasured cations such as calcium or magnesium. This patient had a profoundly elevated anion gap of 25 mmol per liter, which was not explained by the degree of hypocalcemia and hypomagnesemia, and thus prompted a search for the presence of unmeasured serum anions.

CAUSES AND EFFECTS OF ANION-GAP METABOLIC ACIDOSIS

Anion-gap metabolic acidosis has four main causes: lactic acidosis, ketoacidosis, renal failure, and ingested toxins and their metabolites (Table 2). An anion gap of 25 mmol per liter correlates strongly with the presence of one of these conditions³ and is often due to multiple concurrent conditions. This patient's acidosis was probably caused by a combination of lactic acidosis from peripheral hypoperfusion and tissue hypoxia, acute renal failure, and an ingested toxin. The physiological effects of metabolic acidosis include a reflexive increase in respiratory drive, depressed cardiac contractility, peripheral arterial dilatation, increased susceptibility to cardiac arrhythmias, central venoconstriction, a predisposition to pulmonary edema, depression of the central nervous system, and glucose intolerance. This pa-

Table 2. Four Main Causes of Anion-Gap Metabolic Acidosis.

Lactate
Carbon monoxide
Cyanide
Isoniazid (>30 mg/kg)
Iron
Salicylates (cytochrome poisoning)
Metformin
Acute alcohol intoxication
Ketoacidosis
Diabetic ketoacidosis
Alcoholic ketoacidosis
Renal failure
Uremia, decreased secretion of ammonium, hydrogen sulfate, hydrogen phosphate
Toxins and metabolites
Toluene
Methanol, ethylene glycol, paraldehyde (metabolized to formate, oxalate, acetate)

tient was mechanically hyperventilated after intubation in an attempt to maximize respiratory compensation for his metabolic acidosis.⁴⁻⁶ His hyperglycemia was probably a result of the infusion of bicarbonate in 5 percent dextrose he was receiving at the time of transfer, as well as a stress response. The episode of ventricular tachycardia that was precipitated by the guidewire insertion during placement of the central venous line may have been a result of the acidosis and the electrolyte abnormalities.

This patient's obtundation and hypotension probably resulted in tissue hypoxia with lactate overproduction, causing lactic acidosis. The treatment for lactic acidosis relies on restoration of adequate tissue perfusion through intravenous volume repletion and supplemental oxygen, both of which this patient received. The use of vasopressors can also be beneficial if tissue hypoperfusion persists after intravenous volume repletion, but these agents were not required in this case.⁷ A sodium bicarbonate infusion was begun; bicarbonate infusions may help to improve cardiac function when the pH falls below 7.20.^{8,9} However, the infusion can also cause fluid overload and paradoxical tissue acidosis in patients with limited respiratory reserve, cardiac failure, or arrest. When the patient arrived here, the pH had corrected to 7.10, and the central venous pressure and blood pressure were normal.

Diabetic ketoacidosis often occurs in patients with insulin-dependent diabetes in whom an acute physiological stressor results in the accumulation of acetoacetate and β -hydroxybutyrate from fatty-acid metabolism. This patient had no history of insulin dependence, making this an unlikely cause of his acidosis. He did have a remote history of alcohol abuse, making alcoholic ketoacidosis a possible contributing diagnosis. An abrupt discontinuance of alcohol consumption by a long-time alcohol user results in a starvation state that is worsened by subsequent vomiting and dehydration. The ketosis in this condition is predominantly from β -hydroxybutyrate, which is not detected by the nitroprusside ketone reaction used to detect acetoacetate. This patient received appropriate treatment for alcoholic ketoacidosis, including intravenous hydration with a dextrose solution and electrolyte replacement; however, given the acute onset of profound acidosis and the history of recent sobriety, other diagnoses had to be considered.

An important feature of this case is the new onset of renal failure. Acidosis in renal failure is principally due to an accumulation of acids and a reduction of ammonium production due to decreased nephron mass. Acute renal failure typically presents with a combination of hyperchloremic acidosis and anion-gap metabolic acidosis. Bicarbonate levels usually remain greater than 15 mmol per liter, and the anion gap usually does not exceed 20 mmol per liter. This patient had a large anion gap, severe acidemia with a relatively low creatinine level, and no history of previous renal disease. Thus, although renal failure probably contributed to this patient's severe acidosis, it was unlikely to be the primary cause.

Many toxins and drugs can induce acidosis. Carbon monoxide poisoning causing lactic acidosis was ruled out by the other hospital. Salicylates increase lactate production by poisoning mitochondrial cytochromes. However, in this patient toxicologic screening did not show the presence of salicylates. Toxic alcohols, such as methanol and ethylene glycol, would be a primary consideration in this case of anion-gap acidosis. They are converted by alcohol dehydrogenase to toxic anionic acids that increase the anion gap. In addition, these alcohols are osmotically active in their native forms before they are metabolized, and they cause an increase in the measured serum osmolality. This osmolal increase is estimated by the difference between the measured osmolality and the calculated osmolality, determined as follows: $(2 \times \text{the serum sodium level}) + (\text{the blood urea nitrogen level} \div 2.8) + (\text{the blood glucose level} \div 18)$ — known as an osmolal gap. When the osmolal gap exceeds 10 mmol per liter, the presence of a toxic alcohol must be considered. This patient had an osmolal gap of only 8 mmol per liter, which could argue against the presence of a toxic alcohol. However, by the time he came to medical attention, it is probable that most of the osmotically active alcohol would have already been converted to an anionic metabolite, which would not contribute to the serum osmolality. The needle-shaped crystals found in the urine on his arrival in the emergency department of this hospital are characteristic of calcium oxalate, a metabolite of ethylene glycol; this finding, together with the presence of anion-gap acidosis, altered mental status, and acute renal tubular injury, strongly supports the diagnosis of ethylene glycol intoxication.

DR. J. KIMO TAKAYESU'S DIAGNOSIS

Ethylene glycol intoxication.

PATHOLOGICAL DISCUSSION

Dr. Michael Linshaw: The original sample of urinary sediment from this patient was not available to photograph. However, the morphologic structure of urinary crystals can be helpful in establishing a diagnosis. Calcium oxalate crystals are of two types (Fig. 1): calcium oxalate dihydrate crystals, which are typically octahedrons, and calcium oxalate monohydrate crystals, which are needle-shaped. Calcium oxalate monohydrate crystals are not often seen in urinary sediment, but they are typical, and therefore very suggestive, of ethylene glycol ingestion. Calcium oxalate dihydrate crystals may be seen in other conditions and are therefore less specific for the diagnosis. If calcium oxalate crystals are seen within a cast or are associated with dysmorphic red cells (defined as those showing marked variation in size and shape), this finding suggests the possibility of crystal nephropathy.

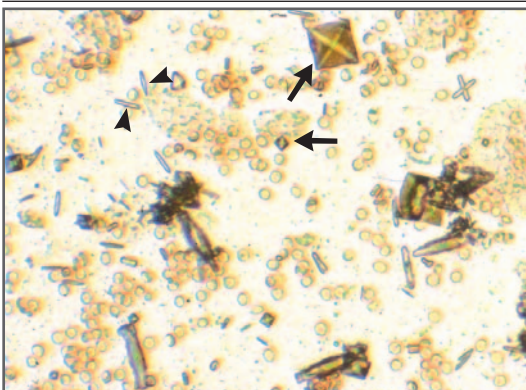


Figure 1. Urinary Sediment Obtained from Another Patient.

This specimen from another patient, who ingested ethylene glycol, contains calcium oxalate crystals of two types. The crystals shaped like envelopes with diagonally crossing lines are octahedrons of calcium oxalate dihydrate (arrows). Calcium oxalate dihydrate crystals can also resemble an hourglass, a dumbbell, or oval shapes about the size of a red cell. The needle-shaped crystals are calcium oxalate monohydrate (arrowheads). These are often slightly thicker in the center of the needle than at the ends. Calcium oxalate monohydrate crystals are rarely seen in the urinary sediment but are typical of ethylene glycol ingestion, and therefore when seen, they strongly suggest the diagnosis.

DISCUSSION OF MANAGEMENT

Dr. Hasan Bazari: This man presented with the triad of depression of the central nervous system with seizures, acute renal failure, and anion-gap metabolic acidosis. An evaluation in the emergency department led to the diagnosis of ethylene glycol intoxication, and the nephrology division was consulted regarding management.

The two alcohols that typically cause both a severe anion-gap metabolic acidosis and an osmolal gap are methanol and ethylene glycol.¹⁰ The osmolal gap is a useful way to detect the presence of alcohols, but it has several pitfalls. The serum osmolality can be measured by determining either the freezing-point depression or the boiling-point elevation (the vapor-pressure method). The latter is unreliable in the presence of volatile substances such as ethanol and methanol,¹¹ but not with ethylene glycol because it has a high boiling point.¹² Once the osmolal gap is calculated, if the ethanol concentration is known, the presence of a second unmeasured osmole can be deduced. However, ethylene glycol, with a relatively high molecular weight of 62, contributes fewer osmoles at a given serum concentration than alcohols of lower molecular weight. Hence, at a concentration of 21 mg per deciliter, the contribution of ethylene glycol to the osmolality may be only 4 mOsm per kilogram and at a lethal dose of 50 mg per deciliter, the contribution to osmolality may be only 8 mOsm per kilogram. Excessive reliance on the osmolal gap can lead one to omit consideration of ethylene glycol intoxication if the gap is below that considered to be normal, as happened in this case. Finally, the time that has elapsed since the ingestion is also important. Alcohols that are metabolized, such as ethylene glycol, will contribute less to the osmolal gap and more to the anion gap as they are metabolized, which is what probably occurred in this patient.¹³

Unfortunately, the routinely available tests for intoxication include the major alcohols in the screening, such as ethanol, methanol, and isopropyl alcohol — but not ethylene glycol. The clinician is forced to rely on other clues to the diagnosis of ethylene glycol intoxication and often has to treat presumptively. Many products that contain ethylene glycol have fluorescein added to them, which causes fluorescence of the urine when visualized under a Wood's lamp.^{14,15} The detec-

tion of fluorescence in the urine is an appropriate test, but the sensitivity, specificity, and accuracy of the findings may be limited.¹⁶

Ethylene glycol is found predominantly in antifreeze but is also present in air conditioner coolant, shoe polish, window cleaner, and fire extinguisher fluid; rare cases of poisoning have been reported with contamination of the water supply.^{17,18} There are about 5000 cases of ethylene glycol ingestion in the United States each year, with an associated mortality of about 1 percent.¹⁹ The lethal dose may be as low as 100 ml. The toxic effects of ethylene glycol are due to the conversion of ethylene glycol to glycoaldehyde and subsequently glycolate by alcohol dehydrogenase. Glycolate is converted to glyoxalate and then to oxalate; glycolate is the major cause of the anion-gap metabolic acidosis.^{12,20}

There are four stages of the toxic effects of ethylene glycol. Stage 1 is characterized by depression of the central nervous system by the native alcohol and lasts from a few hours to as long as 12 hours.²¹ This patient showed the earliest stages of intoxication when he returned with lethargy from working on his car and later when his wife found him incapable of being aroused from sleep. Stage 2 is dominated by the appearance of anion-gap metabolic acidosis, hyperventilation, seizures, pulmonary infiltrates, and hypotension, which were present when the patient was admitted to the other emergency department and progressed on transfer to this hospital.²² Stage 3 is characterized by acute oliguric to anuric renal failure that begins 24 to 48 hours after the ingestion of ethylene glycol. The renal dysfunction, although reversible when treated, may lead to both end-stage renal disease and chronic renal failure. Oxalic acid does not contribute to the metabolic acidosis, but it causes organ damage by perivascular deposition of calcium oxalate crystals in tissues.²³ Consumption of calcium in the formation of calcium oxalate is also assumed to be the cause of the hypocalcemia, which in turn contributes to the occurrence of seizures. A fourth stage characterized by delayed onset of cranial neuropathies can occur from 8 to 15 days after the ingestion and resolves slowly.^{24,25} There is a published case of colonic perforation from oxalate crystals many days later.¹⁷

When we saw this patient, he was transitioning from stage 2 to stage 3. The therapy with so-

dium bicarbonate at the other hospital may have contributed to a precipitous drop in the level of ionized calcium, because the correction of the acidosis leads to a drop in the level of ionized calcium and, at the same time, the production of oxalate and consumption of calcium increase. A lesson from this case that applies to most situations in which there is coexisting metabolic acidosis and hypocalcemia is that calcium needs to be replaced adequately while the acidosis is corrected.

The treatment for ethylene glycol intoxication has a rational and physiologic basis. Hydration and the use of bicarbonate have a number of benefits, including promotion of the excretion of ethylene glycol, correction of the metabolic acidosis, and promotion of the excretion of glycolic acid by alkalization of the urine. Historically, the administration of ethyl alcohol was common,²⁶ drawing on the ability of ethanol to compete with ethylene glycol for metabolism by alcohol dehydrogenase. However, before 1966, most cases involving the ingestion of large amounts of ethylene glycol were fatal.

Since 1966, hemodialysis has been a cornerstone of treatment of intoxication with both methanol and ethylene glycol. The parent compounds as well as the metabolites of the two alcohols are effectively removed with standard hemodialysis with use of a bicarbonate bath.²⁷ The other major advance occurred with the introduction in 1988 of fomepizole (4-methylpyrazole) as a competitive inhibitor of alcohol dehydrogenase, without the side effects of inebriation and risk of aspiration associated with the use of ethanol.²⁸⁻³⁰ Currently, fomepizole is recommended for all cases of ingestion of methanol or ethylene glycol, with hemodialysis reserved for those with very high serum levels, severe metabolic acidosis, and evidence of end-organ damage such as renal failure, which this patient had. Thiamine is used, in addition, to promote the metabolism of glycolic acid to a nontoxic metabolite, alpha-hydroxy-beta-keto-adipate, and also pyridoxine to help metabolize glyoxalate to glycine.³¹

This case raises a number of important points about ethylene glycol toxicity. First, the osmolal gap may be low even with a high level of toxicity. Second, the presence of marked anion-gap acidosis and acute renal failure should suggest the toxic effects of ethylene glycol, especially if there is no other cause identified for the acidosis. Third, nee-

dle-shaped crystals of calcium oxalate monohydrate in the urine are characteristic of ethylene glycol intoxication. Fourth, hypocalcemia may be exacerbated by the correction of the acidosis, causing a precipitous fall in the ionized calcium level. Fifth, the toxic effects of ethylene glycol should be treated immediately when sufficient clues exist to make the diagnosis, even without conclusive proof, since timely therapy can lead to a good outcome but delaying therapy can be fatal.

Dr. Nancy Lee Harris (Pathology): Are there any questions for Dr. Bazari?

Dr. Ron M. Walls (Emergency Medicine, Brigham and Women's Hospital): Reference is made to bicarbonate administration for a pH below 7.10, when the pH value is higher. I am not aware of any reliable evidence that bicarbonate administration improves outcome in cases of anion-gap metabolic acidosis. Studies of the use of bicarbonate in diabetic ketoacidosis have concluded that it is of no benefit and may even be harmful.^{32,33} This relative proscription has been extended to other causes of anion-gap metabolic acidosis.³⁴ Despite the apparent lack of any supporting evidence, however, a tendency persists to recommend bicarbonate therapy when metabolic acidosis is severe.⁸ Can you identify any studies that have demonstrated an improved outcome when patients with anion-gap metabolic acidosis have been treated with bicarbonate?

Dr. Bazari: The management of an anion-gap metabolic acidosis depends on its cause. There is no evidence that correction of the acidosis in diabetic ketoacidosis has a beneficial effect on the outcome. Similarly, correcting the acidosis may actually be detrimental in the management of cases of lactic acidosis. However, in cases of the ingestion of certain toxic substances, the pathophysiology of the anion-gap metabolic acidosis is very different, and I do not have a problem with the use of sodium bicarbonate. In patients with a metabolic acidosis that is not associated with the anion gap — especially those with chronic kidney disease and a limited ability to respond to an acid load — replacement of bicarbonate can expedite

normalization of the serum bicarbonate level and clinical well-being.

Dr. Harris: Dr. Kehoe took over this patient's primary care while he was in this hospital.

Dr. Laura G. Kehoe (Internal Medicine): The patient's mental status improved over the course of the first hospital night, and the trachea was extubated without complications. When the patient awakened, he told the nurse that he had ingested antifreeze in an attempt to injure himself. Suicide precautions were instituted with a one-to-one sitter in the room. When I questioned him directly, he admitted to an oxycodone (OxyContin) habit (120 mg daily) and said he had been recently cut off by his supplier for lack of payment. Thus, he was probably in opiate withdrawal, which presumably contributed to the malaise that precipitated his ingestion of ethylene glycol.

The department of psychiatry was consulted, and the patient was seen daily. He had several episodes of nausea, vomiting, and anxiety with respiratory distress; quetiapine was given, which relieved his symptoms. On the second hospital day, an additional dose of fomepizole was administered; hemodialysis was repeated on that day and again on days 8 and 9 because of the rising level of serum creatinine (Table 1). A chest radiograph continued to show consolidation of the left lower lobe of the lung. Intravenous antibiotics were discontinued, and oral levofloxacin was administered (a 14-day course). His renal function gradually stabilized. Gabapentin was prescribed for relief of his chronic back pain. He was discharged on the 20th hospital day. At the time of discharge, he was not believed to be at further risk for suicide, and outpatient psychiatric care was scheduled.

He is now enrolled in weekly psychiatric care and in a methadone program; he takes 120 mg of methadone per day and is doing well.

FINAL DIAGNOSIS

Ethylene glycol intoxication.

No potential conflict of interest relevant to this article was reported.

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CORRECTION

Case 7-2006: A 47-Year-Old Man with Altered Mental Status and Acute Renal Failure

Case 7-2006: A 47-Year-Old Man with Altered Mental Status and Acute Renal Failure . On page 1067, in the left-hand column, the formula in line 4 under the heading "The Anion Gap" should have read " $\text{Na}-(\text{Cl}+\text{HCO}_3)$," rather than " $\text{Na}-\text{Cl}+\text{HCO}_3$," as printed. We regret the error.