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Cardiac arrhythmias, respiratory failure, and profound hypokalemia in a trauma patient

A 50-YEAR-OLD MAN, previously in excellent health, arrived at the emergency department via helicopter, having been injured at his job at a munitions factory. As he was placing 1 kilogram of propellant behind a 2-inch-thick transparent blast shield, it exploded, throwing him 40 feet across the room.

Initially he was able to walk from the scene, but soon required help from coworkers. The emergency medical services crew arrived promptly and measured his heart rate at 122 beats per minute, respiratory rate 28, and systolic blood pressure 88 mm Hg; the heart monitor indicated he was in sinus rhythm. He had burns on his face and abdomen, with a marked deformity of his right arm. He arrived at the emergency department within 15 minutes of the event on a backboard with a cervical collar in place.

Physical examination

On arrival, his blood pressure was 170/110 mm Hg, rectal temperature 29.5°C (85.1°F), and heart rate 110 beats per minute, with ventricular bigeminy on the monitor.

Head and neck examination revealed no scalp lacerations, but he had partial-thickness and full-thickness burns with tattooing, multiple superficial lacerations, and singed eyebrows. His pupils were 4 mm and sluggishly reactive to light. There were bilateral corneal burns. His right tympanic membrane was ruptured.

Thorax. His breath sounds were diminished on the left side. His heart rhythm was regular with no murmur, gallop, or rub.

Abdomen showed full-thickness and partial-thickness burns, with diminished bowel sounds. There was ecchymosis on his right flank.

Extremities. The patient could not move his right arm, but otherwise moved his limbs symmetrically.

Oxygen saturation by pulse oximetry was 92% on room air.

Past medical history was unremarkable.

Imaging studies

Computed tomographic (CT) scans of the head and abdomen and lateral cervical spine radiographs were normal. Radiographs of the right arm indicated a fractured radius, ulna, and humeral condyles. A chest roentgenogram revealed a small left apical pneumothorax, consistent with the physical examination.

Arterial blood values

- P_{CO_2} 35 mm Hg (normal 34–46)
- P_{O_2} (while receiving oxygen via a nonrebreather mask at nearly 100% F_{IO_2}) 213 mm Hg
- Bicarbonate 10 mmol/L (normal 22–26)
- pH 6.84 (normal 7.35–7.45)

Chemistry profile

- Sodium 141 mmol/L (normal 135–146)
- Potassium 3.5 mmol/L (normal 3.5–5.0)
- Chloride 107 mmol/L (normal 98–110)
- Blood urea nitrogen 14 mg/dL (normal 10–25)
- Lactate 21.8 mmol/L (normal 0.5–2.2)
- Anion gap 24 mmol/L (normal 8–16)

Complete blood count

- Hemoglobin 10.2 g/dL (normal 13.5–17.0)

His lab findings: pH 6.84 bicarbonate 10 lactate 21.8 potassium 3.5 and falling

- Hematocrit 32.2% (normal 40.0–52.0)
- White blood cell count 18.2 × 10⁹/L (normal 4.0–10.0)
- Platelet count 197 × 10⁹/L (normal 150–400).

The hypokalemia worsens, despite potassium replacement

Because the electrocardiogram exhibited ventricular ectopy with frequent premature ventricular contractions and nonsustained ventricular tachycardia, a lidocaine infusion was started, and the patient was given three 50-mmol ampules of sodium bicarbonate.

Ninety minutes later the patient's potassium level had fallen to 2.9 mmol/L. An infusion of 50 mmol of potassium was started, to run at 10 mmol/hour, along with two units of red blood cells to be transfused over 2 hours. One hour later, his blood pH was 7.36, PCO_2 18 mm Hg, and potassium level 2.3 mmol/L. The remaining 40 mmol of potassium was rapidly infused over 30 minutes. Despite this potassium bolus, the potassium level fell further to 1.5 mmol/L. Another 100 mmol of potassium was infused over 30 minutes, and his potassium rose to 4.2 mmol/L.

A chest tube was inserted to treat his pneumothorax, and he was taken to the operating room for irrigation, debridement, and fasciotomies of his right upper extremity. He recovered uneventfully from his injuries. His acid-base status stabilized and he required no further treatment for acidosis.

■ **WHAT CAN WE LEARN FROM THE ELECTROCARDIOGRAM?**

The patient's electrocardiogram showed a rate of 81 with sinus rhythm. The QT interval (406 ms) and QTc (470 ms) were prolonged—at a rate of 81 beats per minute a QT interval of no more than 350 ms would be expected. The electrocardiogram also showed ST depression in leads V_1 through V_5 and T-wave flattening.

1 What caused the prolonged QT interval in this patient?

- ❏ Hypothermia
- ❏ Hypokalemia
- ❏ A central nervous system disease
- ❏ Hypomagnesemia and hypocalcemia
- ❏ Toxic exposure

Causes of QT interval prolongation include all of the above; however, we can exclude some of them in this patient.

Hypothermia. This patient was hypothermic, presumably because of his trauma. His rectal temperature of 29.5°C could certainly account for QT prolongation.

Hypokalemia. We know the patient had hypokalemia. The long QT interval associated with hypokalemia is probably due to a U wave, which may be masked by ST-T abnormalities. T and U wave fusion may explain a prolonged QT interval.

Central nervous system diseases that can cause QT prolongation include subarachnoid hemorrhage. Although this patient's head injury may have caused some blood to be present in the subarachnoid space, there is no evidence that he suffered a primary subarachnoid bleed, and his head CT was normal.

Hypomagnesemia and hypocalcemia were not present: the patient's magnesium level was 2.0 mg/dL and his calcium level was 9.8 mg/dL.

Toxic exposure that can cause QT prolongation can be from drugs such as phenothiazines, tricyclic antidepressants, and other psychotropic drugs; class IA antiarrhythmics (quinidine, disopyramide, procainamide); and class III antiarrhythmics (sotalol, amiodarone, ibutilide, dofetilide). The patient had been taking none of these. It can also be due to ethylene glycol or barium. He was not exposed to ethylene glycol; we will consider barium below.

■ **WHAT ABOUT HIS ACIDOSIS?**

2 What are possible causes of the acidosis in this patient?

- ❏ Renal failure
- ❏ Diabetic or alcoholic ketoacidosis
- ❏ Lactic acidosis
- ❏ Toxic exposure

All of the above are possible causes of metabolic acidosis with a high anion gap, but we can rule out most of them in this patient. He

There was no sign that potassium actually left his body

TABLE 1

Categories of lactic acidosis

Type A
Due to hypoperfusion, hypoxemia
Causes: Cyanide, carbon monoxide, shock, pulmonary edema, methemoglobinemia, sulfhemoglobinemia, asphyxia, status asthmaticus

Type B
1. Due to systemic illness (diabetes mellitus, malignancy, pancreatitis, leukemia, sepsis, liver disease)
2. Due to toxins (phenformin, ethanol, fructose, iron, isoniazid, strychnine)
3. Due to congenital errors of metabolism or enzyme deficiencies

was not taking any medications and he had not ingested any poisons. Since there is also no evidence that he has renal insufficiency or any state that can lead to ketoacidosis, the acidosis must have been due to lactic acidosis or to a toxic substance to which he was exposed during the explosion.[1,2] One substance used in the explosive industry that can cause metabolic acidosis is toluene.

With a lactate level of 28.8 mmol/L, the patient clearly had lactic acidosis. **TABLE 1** lists the general categories of lactic acidosis,[3] and **TABLE 2** lists substances that can induce it. Since this patient does not have any history of systemic illness, specific toxin ingestion, or a congenital error of metabolism, his lactic acidosis must be of type A, ie, due to hypoperfusion or hypoxemia. Of the various causes of hypoperfusion or hypoxemia, cyanide and carbon monoxide poisoning are in the differential diagnosis for anyone sustaining a blast or inhalation injury.

This patient was not hypotensive for very long. He did not have pulmonary edema or status asthmaticus. The possibility of methemoglobinemia, especially following exposure to explosives, is very strong.[4]

The acidemia resolved rapidly when the patient was given sodium bicarbonate, and remained normalized as his vital signs stabilized. The anion gap resolved quickly as well. It appears that this laboratory abnormality can be attributed to a period of hypoxemia or hypoperfusion. There is no evidence that he was inappropriately ventilating for the degree of metabolic acidosis, as his PCO_2 was 35 mm Hg, and his first arterial blood gas measurements did not indicate any degree of hypoxemia.

Can methemoglobinemia explain the acidosis?

The munitions industry uses a number of nitrogenous compounds, including aromatic nitrates and ammonium nitrate, which carry the hazard of acute methemoglobinemia. **TABLE 2** lists other toxins shown to cause methemoglobinemia.

Symptoms and findings of methemoglobinemia parallel those of carbon monoxide poisoning to a certain extent. Methemoglobin levels of 10% to 15% can cause cyanosis; levels of 20% to 30% can cause headache, fatigue, and nausea; and levels of 50% to 60% can cause metabolic lactic acidosis and dysrhythmias. Methemoglobinemia can be diagnosed by a chocolate-brown color of blood after it has dried on filter paper.[5] The classic slate-gray cyanosis may be masked by burns. If methemoglobinemia is suspected, it is important to measure the methemoglobin level because this condition has specific therapy.[6]

Patients with methemoglobinemia may benefit from administration of methylene blue, exchange transfusion, or hyperbaric oxygen. Patients exposed to toxins that induce methemoglobinemia should be decontaminated, including removing contaminated clothing and flushing the skin.

This diagnosis was not looked for initially in this patient. Presumptive therapy with methylene blue may induce central nervous system effects such as tremor, nausea, vomiting, dizziness, and confusion—potential confounding effects. Our patient's acidosis resolved with bicarbonate replacement and treatment of his injuries and did not require specific therapy. His prompt recovery with supportive care essentially rules out significant cyanide or carbon monoxide exposure.

■ WHAT IS THE DANGER FROM HYPOKALEMIA?

Under experimental conditions, for every decline of 0.1 in the pH, the measured serum potassium level rises by 0.6 mmol/L, although

TABLE 2

Substances that can induce lactic acidosis

Substances that can induce hypotension, seizures, respiratory failure (Po_2 < 35 mm Hg), hepatic failure, or renal failure

Acetaminophen	Ethylene glycol	Papaverine
Aspirin	Fructose	Phenformin
Biguanides	Iron	Povidone-iodine ointment
Carbon monoxide*	Isoniazid	Ritodrine
Chloramphenicol	Metformin	Sorbitol/xylitol
Cocaine	Methanol	Streptozotocin
Cyanide*	Nalidixic acid	Strychnine
Epinephrine	Nitroprusside	
Ethanol	Norepinephrine	

Substances that can cause methemoglobinemia*

Nitrites (rapidly oxidize heme iron because of their high oxidation potential)
  Bismuth subnitrate, other nitrates (can be converted to nitrites by gut or skin bacteria and absorbed)
  Butyl, amyl, isobutyl nitrites (recreational agents)
  Nitroglycerin
  Silver nitrate
Anilines (easily penetrate skin)
  Acetanilid
  Toluidine, nitrobenzenes (plastics, fungicides, dyes, paints, rubber)
Potassium and sodium chlorates (matches, weed killers, mouthwash)
Therapeutic agents
  Antimalarial agents
  Benzocaine
  Dapsone
  Metoclopramide
  Nitroglycerin
  Phenacetin
  Sulfonamides
Contaminated well water (nitrates in fertilizers)

*Classified as type A lactic acidosis

Hypokalemia may be spurious or due to either depletion or redistribution

this may not be seen with organic acidosis or acidemia. This patient, who had acidosis at the same time that his potassium level was 2.3 or 1.5 mmol/L, had life-threatening hypokalemia.

The clinical effects of hypokalemia are a direct consequence of membrane hyperpolarization, ie, the membrane potential is made more negative so that there is a shift in the membrane potential away from the threshold level required to produce an action potential.[7]

The major clinical effects of hypokalemia are in myocardial tissue, skeletal muscle, and the gastrointestinal system.

Myocardial effects of hypokalemia include re-entrant and automatic atrial and ventricular tachycardias, AV dissociation, and ultimately ventricular fibrillation. If the patient is in sinus rhythm, the electrocardiogram may show T-wave flattening, ST depression, and U waves.

Skeletal muscle effects include paresthesia, muscle cramps, respiratory muscle paralysis, weakness, and lassitude. At potassium levels below 2.5 mmol/L, muscle necrosis may develop, and at levels below 2.0 mmol/L, ascending paralysis may occur, although this is unusual.[8]

Gastrointestinal effects commonly include nausea, vomiting, and paralytic ileus. Conscious patients may complain of constipation.

■ HYPOKALEMIA: A DIAGNOSTIC APPROACH

3 What is the probable cause of this patient's low potassium measurements?

❑ Spurious hypokalemia
❑ True potassium depletion
❑ Potassium redistribution

Spurious hypokalemia. In some cases the patient actually has a normal serum potassium level, but the level in the blood sample declines by the time the laboratory measures it. This occurs in two specific instances.
• If the patient has received insulin within the past 15 to 20 minutes, the insulin may transiently drive down the serum potassium, representing redistribution.
• In leukemic patients with white blood counts of 100 to 250×10^9/L, the white blood cells may metabolize the potassium in the serum.

Neither of these situations existed in this case.

(Pseudohyperkalemia, representing an artificially elevated plasma potassium from hemolysis, prolonged use of a tourniquet, or marked leukocytosis or thrombocytosis is much more common than pseudohypokalemia.)

True potassium depletion can develop from either chronic extrarenal or renal losses. Extrarenal loss of potassium associated with acidosis can be caused by prolonged fasting, diarrhea, GI fistulas, and villous adenomas of the rectum. Renal causes of potassium loss include certain primary proximal types of renal tubular acidosis, diabetic ketoacidosis, ureterosigmoidostomy, loop diuretics, and carbonic anhydrase inhibitors.

We can dismiss these conditions, as the patient has not been chronically ill, is not taking a diuretic, and has no known history of renal disease or diabetes. He has no history of anything that would cause gastrointestinal potassium loss, and he has no history of surgical procedures such as ureterosigmoidostomy. Finally, he simply has not had time to lose potassium—the hypokalemia developed acutely and with no evidence that potassium actually left his body.

Potassium redistribution is clearly what is going on in this patient. The major causes of potassium redistribution are:
• Alkalemia with increased plasma bicarbonate. This can be dismissed as the patient is not alkalemic and does not have an elevated plasma bicarbonate level.
• Insulin excess (endogenous or exogenous). This does not apply to this patient either.
• Beta-adrenergic antagonists, which this patient does not use.
• Drugs and toxins such as theophylline, barium, toluene, and calcium channel blockers. The patient is not taking theophylline or calcium channel blockers; however, barium and toluene have to be strongly considered.

How do drugs and toxic substances induce hypokalemia?

Some drugs and substances can induce hypokalemia by causing actual potassium loss. Others, however, cause potassium redistribution (**TABLE 3**). In the latter group, substances such as theophylline, beta-2 agonists, and thyroid hormone drive potassium into cells via the sodium-potassium ATPase pump. Others, such as barium and chloroquine, lower serum potassium by blocking potassium channels and preventing potassium from getting out of the cells via osmosis. In these cases, although potassium cannot exit the cell, it continues to be pumped in via the ATP-dependent sodium-potassium pump, and although the total body store of potassium remains normal, the extracellular potassium level may drop precipitously.[9]

The two toxic substances that lower serum potassium and warrant serious consideration are toluene and barium, both of which are used in the explosives industry.[10–12]

■ CAN TOLUENE ACCOUNT FOR HIS HYPOKALEMIA?

Toluene is used in the explosives industry, especially in the early phases of manufacturing. Specifically, it is used with sulfuric acid or nitric acid at 100°C to produce the aromatic nitrates dinitrotoluene and trinitrotoluene (TNT).

Potassium replacement may be ineffective if magnesium is low

Toluene is also a major ingredient of airplane glue and may be an additive in unleaded gasoline. Toluene toxicity has been mainly described in persons who sniff glue or spray paint to produce euphoria.

Diagnosis of toluene intoxication

Symptoms of toluene intoxication include muscle weakness, quadriparesis, and a variety of gastrointestinal complaints including abdominal pain, nausea, vomiting, and hematemesis.[13] Neuropsychiatric signs may predominate and include altered mental status, cerebellar signs, peripheral neuropathy, headache, dizziness, and syncope. Confusion and coma have been reported, as have dysrhythmias and myocardial infarction.[14]

Factors pointing to toluene intoxication in this patient include hypokalemia, high anion gap metabolic acidosis, and possible exposure in his work. However, other factors argue against toluene intoxication in this patient:

• The muscle weakness in toluene intoxication generally spares respiratory and central muscles.

• Toluene intoxication is generally associated with chronic, not acute, exposure or abuse.

• Toluene is used at an earlier stage of manufacturing than this patient was exposed to.

• Toluene tends not to cause as acute a drop in serum potassium levels as in this patient, since it causes potassium wasting through a mechanism of renal tubular acidosis, which entails more of a potassium loss rather than a potassium redistribution.

This patient's presentation is therefore not consistent with acute toluene intoxication.

■ CAN BARIUM PRODUCE THIS PICTURE?

Barium is a divalent alkaline earth metal with a molecular weight of 137. With such a high molecular weight, it is not readily penetrated by x-rays, and so it is used as a contrast agent for radiographic studies in the form of barium sulfate, which is insoluble.

Barium poisoning results from ingestion or inhalation of the acid salts,[15] most often rodenticides such as barium carbonate or barium hydroxide. Of note: barium chlorate is

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TABLE 3

Substances that lower serum potassium levels

Substances that can cause spurious hypokalemia
Insulin

Substances that can cause true potassium depletion
Acetazolamide
Amphotericin B
Antibiotics in high doses
   Ampicillin
   Carbenicillin
   Nafcillin
   Penicillin
Caffeine
Carbonic anhydrase inhibitors
Cathartics
Dextrose
Licorice, carbenoxalone, gossypol
Loop diuretics
Oral hypoglycemics
Osmotic diuretics
Salicylates
Sodium polystyrene sulfonate
Steroids
Strychnine
Sympathomimetics
Thiazide diuretics
Vitamin B_{12}

Substances that can cause potassium redistribution
Barium (soluble salts)
Beta-adrenergic agonists
Bicarbonate
Chloroquine
Insulin (possibly)
Theophylline (in overdose)
Thyroid hormone
Toluene
Verapamil (in overdose)

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used in fireworks and in explosives, and barium nitrate is commonly used in manufacturing TNT. A chronic form of barium poisoning called baritosis has also been reported and represents a chronic and benign pneumoconiosis.

The hypokalemia of barium poisoning, as discussed above, entails potassium redistribution. Barium reduces potassium efflux from muscle cells by blocking potassium channels and has no proven activity on the sodium-potassium ATP-dependent pump. Therefore, the continued activity of the ion pump com-

bined with blocked potassium efflux results in extracellular hypokalemia and intracellular potassium accumulation. Since muscle accounts for 44% of body mass, the serum potassium level can drop quite precipitously, and as it does, so does the transmembrane ionic diffusion potential, to less than 60 mV. At this membrane potential the muscle is unexcitable and paralysis ensues.[16]

Diagnosis of barium intoxication

Laboratory abnormalities with barium intoxication are rhabdomyolysis, hypophosphatemia, and potentially severe hypokalemia. Potassium levels as low as 0.3 to 0.8 mmol/L have been reported.[17]

Other manifestations of barium poisoning include hypertension, cardiac dysrhythmias, and gastrointestinal toxicity. Perioral paresthesia, tingling in the face and hands, abdominal cramps, diarrhea, and intractable vomiting may develop within minutes. Central nervous system signs of barium poisoning include mydriasis, anxiety, headache, confusion, myoclonus, trismus, and seizures. Barium can cause ventricular tachydysrhythmias, QT prolongation, congestive heart failure, and asystole. Sensation is preserved, as is level of consciousness. The weakness may not correlate with the degree of hypokalemia documented by the laboratory.

The differential diagnosis of barium poisoning includes various diseases that may induce paralysis, including familial hypokalemic periodic paralysis, thyrotoxic periodic paralysis, Guillain-Barré syndrome, gastroenteritis, botulism, diphtheria, paralytic shellfish poisoning, ciguatera fish poisoning, and neurotoxic shellfish poisoning.

This patient's clinical presentation and exposure in the explosive industry are consistent with and characteristic of barium intoxication. The most likely barium salt that he would have been exposed to is either barium nitrate or barium chlorate.

Treatment of barium poisoning

Treatment involves aggressive potassium replacement, since paralysis is due to hypokalemia, not to the barium per se.[18] Patients may need as much as 400 mmol of potassium over the first 24 hours. There is a theoretic risk of potassium overshoot, since the total-body store of potassium is normal, but potassium replacement seems to be well tolerated. The serum potassium level must rise high enough to displace barium from the potassium channels. With potassium replacement, symptoms generally abate by 24 hours and patients are usually ambulatory by 48 hours.

If hypertension, paralysis, or QT prolongation exist, cardiac monitoring is indicated. Dysrhythmias secondary to barium poisoning should be treated with potassium—not with lidocaine or procainamide. This patient was given lidocaine, but it was not known at the time what the etiology of his ventricular dysrhythmias was. Lidocaine was discontinued with potassium replacement. The diagnosis of barium poisoning was not made until his hypokalemia was addressed. Hypertension should be treated with nitroglycerin in general. Intravenous magnesium sulfate poses a risk if precipitating barium sulfate in the renal calyces.

Gastric lavage with sodium sulfate or potassium sulfate may have a role if the mechanism was ingestion of barium, since the sulfate ions bind to the barium ions, which precipitate as nontoxic barium sulfate. Activated charcoal is ineffective in adsorbing barium. Hemodialysis may shorten the half-life of barium but is generally unnecessary.

■ WHICH LABORATORY TESTS CAN CONFIRM THE DIAGNOSIS?

4 What further laboratory tests might be indicated for this patient?

❑ Serum barium levels
❑ Tests for methemoglobinemia
❑ Tests for toluene toxicity
❑ Thyroid function tests

Barium levels can confirm the diagnosis of barium toxicity. The normal range for serum barium is 3 to 29 µg/dL. This patient had a serum barium level of 133 µg/dL. Since he was treated appropriately with potassium replenishment, his high barium level did not change his course of therapy. Abdominal roentgenograms may reveal ingested barium,

Both toluene and barium are used in explosives

although this patient's exposure appears to have been by inhalation and through skin absorption via his burns.

Tests for methemoglobinemia. This patient did not sustain clinically significant methemoglobinemia, although testing in this situation may be warranted.[19]

Urinary DNAT. Another test to consider in anyone who works in the munitions industry is urine screening for 2,6-dinitroamino-toluene (DNAT). By industry standards this is the screen most often used routinely for ongoing monitoring of toluene toxicity, although in this patient's case it would be of little emergency use.

Serum magnesium is generally important to check because the patient may be refractory to potassium replacement if he is hypomagnesemic.

Thyroid function tests. The patient had hypothermia, paralysis, and hypokalemia, all of which are indications for checking thyroid function at some point. Hypothyroidism is always in the differential diagnosis of hypothermia. Hyperthyroidism can be associated with periodic paralysis and as noted earlier, development of hypokalemia via the sodium-potassium ATPase pump.

■ DIAGNOSIS

- Barium poisoning with acute hypokalemia due to potassium redistribution
- Type A lactic acidosis due to transient hypoxemia or hypoperfusion
- QT prolongation due to hypothermia, to barium exposure, and perhaps also to hypokalemia.

In barium poisoning, treat arrhythmias with potassium, not lidocaine or procainamide

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