Cyanide Poisoning and Lactate in Smoke Inhalation

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ABSTRACT

House fires can lead to cyanide poisoning and an associated elevated serum lactate level. Because of delays in obtaining serum cyanide levels, clinical symptoms and serum lactate are often used to guide clinical decision making and antidote administration. However, as this case report identifies, lower levels of serum lactate may in fact correlate with higher levels of serum cyanide that could benefit from treatment with an antidote.

KEYWORDS: cyanide; house fire; lactate; antidote; toxicology; emergency medicine

INTRODUCTION

House fires lead to the burning of both natural and synthetic materials, which can result in the creation and release of hydrogen cyanide.[1] Cyanide exposure can occur in several ways, including dermally, parenterally, and by ingestion.[1] In house fires, cyanide is released as a gas that leads to clinical asphyxiation, and leads to acute elevations in serum lactate.[1] Here, we present a case of smoke inhalation with a markedly elevated serum cyanide but a discordantly low level of serum lactate.

CASE REPORT

The patient was a 55-year-old female with a prior medical history of anxiety, depression, gastroesophageal reflux, and anemia, who presented to the emergency department by emergency medical services (EMS) after a house fire. She reported that she had fallen asleep after throwing a cigarette into the wastebasket, and was awoken to large flames and a smoke-filled room. EMS was contacted, and at scene arrival the patient had a reported oxygen saturation of 85% by pulse oximetry. She improved to 92% after she was placed on oxygen delivered by a non-rebreather (NRB) mask. The patient was a long-term tobacco user who currently smoked about 1 pack per day.

On arrival at the emergency department (ED), the patient was alert and oriented to person, place and time, speaking comfortably in full sentences. She was on the non-rebreather, breathing 15 times per minute (reference: 12–18 times per minute), and saturating 98% on pulse oximetry (reference: ≥95%) on arrival. Her blood pressure was 111/56 mmHg (reference: 90–140/60–90 mmHg), and her heart rate was 110 beats per minute (reference: 60–90 beats per minute). Her face, including her lips and nares, and upper arms were covered in soot. She had soot on the anterior aspect of her tongue but there was none noted in her posterior oropharynx. No burns were noted throughout the face, nose, or oropharynx. There was no stridor, and on auscultation, the patients' lungs were clear bilaterally with no wheezing. Cardiac exam revealed a tachycardic, regular rhythm. Pulse co-oximetry, which measures carbon monoxide bound to hemoglobin, was performed in the ED and showed a level of 33.The patient was intermittently sleeping between interactions, but easily aroused to voice and could hold a full conversation with her eyes open.

A chest radiograph demonstrated bibasilar opacities thought to be secondary to atelectasis. An electrocardiogram showed sinus tachycardia, at a rate of 142 (and a PR of 136, QRS of 78, and QTC of 406). The patient's initial venous blood gas revealed a pH of 7.23, and a whole blood lactate was 6.2 mEq/L. Other blood gas and lab values are listed in **Figures 1**, **2**. Repeat lactate after 2 hours on NRB mask and two liters of intravenous crystalloid was 1.2 mEq/L.

Initial venous carboxyhemoglobin level was 13.5%, and when repeated about 12 hours later had decreased to 3%. A cyanide level was sent on arrival and resulted the next day at >100 ug/dL (reference range 0–20 ug/dL).

The case was discussed with the regional poison center who recommended against hydroxocobalamin or

Figure 1. Initial venous blood gas obtained on arrival

Test	Patient Value
pH	7.23
PCO2	60
PO2	50
Venous O2	67
Glucose	169

Figure 2. Additional laboratory findings obtained in the ED

Test	Patient Value
Initial Lactate	6.2 mEq/L
Repeat Lactate (2 hours)	1.2 mEq/L
WBC	9.6 x 109/L
Hgb	16.3 g/dL
Hct	49.4 g/dl
Platelets	305 x 109/L
Sodium	139 mEq/L
Potassium	3.6 mEq/L
Chloride	102 mEq/L
CO2	22 mEq/L
Ethanol	Not detected
HS troponin	19 ng/L

transfer for hyperbaric oxygen (as this was not available at the medical center she presented to) based on the patient's clinical presentation and the available laboratory results (particularly the lactate and carboxyhemoglobin levels). The patient was maintained on 100% oxygen by NRB mask and was admitted to the hospital for observation, showing improvement in her carboxyhemoglobin level after approximately 12 hours. She remained persistently hypoxic on room air over the next several days, but was weaned to room air and was subsequently discharged to home without any oxygen supplementation.

DISCUSSION

Patients with smoke inhalation can exhibit an array of neurologic symptoms, ranging from asymptomatic to agitation/confusion to somnolence to coma. Other complications can include myocardial ischemia, dysrhythmias, and metabolic acidosis due to impaired oxygen transfer and utilization in tissues. Smoke inhalation can lead to a complex set of chemical exposures and a variety of physiological impacts, but rapid assessment and initiation of interventions to reverse tissue hypoxia and promote oxygen delivery is essential.[1] Toxic combustion products from fires include simple asphyxiants such as carbon monoxide and cyanide, both of which have antidotes. For cyanide poisoning, several antidotes exist, but currently hydroxocobalamin is used most frequently. It is generally well tolerated and works by chelating cyanide to form cyanocobalamin which can then be excreted.[2]

The relationship between elevations in serum lactate in cyanide poisoning, due to inhibition of cellular cytochrome c oxidase and the electron transport chain, has been well elucidated.[3–5] Serum lactate measurement is of particular value and interest to emergency providers given this test is widely available, portable at the point-of-care, and results rapidly.[6,7] This is in contrast to the time delay in obtaining serum cyanide levels, which are often either unavailable or whose results are significantly delayed, such that they are of low clinical relevance and cannot serve to guide real time decision making.

Several case reports and retrospective case series have examined serum lactate levels in the setting of smoke exposure. Data from the Paris Fire Brigade (a unit of the French army that serves as the fire and rescue service for Paris) suggested that a lactate level of 10 mmol/L or higher was a sensitive indicator of cyanide toxicity.[8] Of 39 victims of smoke inhalation, 23 had blood cyanide levels >40 umol/L (100ug/dL), revealing cyanide toxicity. Of the 23 patients, only 3 had plasma lactate concentrations below 10 mmol/L.[8] Recent work in smoke-exposed porcine animal models has also shown that there is a significant correlation between cyanide toxicity and serum lactate levels. However, this study also found that low or minimally elevated lactate levels cannot exclude lethal cyanide intoxication.[9]

In our case, the lactate level was 6.2 mg/L. While elevated, it fell below the threshold for hydroxocobalamin administration suggested by the existing literature and standard of care. However, this lactate concentration correlated with a cyanide concentration of >100 ug/dL (>1 mg/L). It is possible that the patient's tachycardia and sleepiness were clinical indicators of cyanide toxicity. This patient's serum cyanide level was certainly elevated, with serum levels of 1–2 mg/L considered moderate, 2–3 mg/L as severe, and >3 mg/L as lethal.[7] Unfortunately, the resulted lab value in this case was simply reported as >100 ug/dL, and not a more specific value. Further, the patient's history of smoking may serve as a confounder, as smoking can lead to a baseline presence of cyanide in the serum.[10] Serum cyanide levels are noted to be difficult to interpret, and as with some other poisons, the test result must be clinically correlated. Given the delays in getting the results of a serum cyanide level, clinicians must make decisions based on the patient's clinical exam and other rapid diagnostics that can serve as corollaries.

The clinical symptoms seen in the Paris Fire Brigade data showed that cardiovascular instability (hypotension) and significantly depressed Glasgow Coma Scale scores can be useful clinical indicators in evaluating the risks and benefits of hydroxocobalamin administration.[8] Further, in this case, EMS had applied oxygen on scene and thus it is possible that this intervention lowered the lactate that was drawn on ED arrival, given that elevated lactate in smoke inhalation is often multifactorial. As EMS pre-hospital care evolves, on scene point-of-care measurements of lactate by EMS could help more rapidly assess lactate level and guide early field-based administration of hydroxocobalamin.

The existing literature centers around elevated lactate levels as a corollary for elevated cyanide levels, though little data exists regarding patients with a lower range lactate and concurrent potentially lethal levels of cyanide poisoning. We report one such case here. Further evaluation through additional research may be beneficial, as it may have implications for revising protocols to be more sensitive and lead to the early administration of useful antidotes.

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Acknowledgment

Thank you Dr. Nathan Charlton at the University of Virginia, for his insights and suggestions in the drafting of the manuscript.

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