Restraint associated profound lactic acidosis in a cocaine intoxicated patient: Early recognition and treatment are life-saving.

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Short title: Lactic acidosis in restrained cocaine intoxicated patients

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Abstract:

Severe lactic acidosis has been reported in patients struggling against restraints, especially in association with the use of stimulant drugs, such as cocaine. Profound acidosis occurring under these conditions can lead to cardiac arrhythmias, autonomic instability and cardiac arrest, a syndrome known as restraint associated asphyxia. Early recognition of this condition and removing the stimulus for lactic acid production (excessive muscle activity) by aggressive sedation and ventilatory assistance coupled with fluid administration to improve tissue perfusion and lactate metabolism can be life-saving.

The current report describes a case of restraint associated severe lactic acidosis in a cocaine intoxicated patient that was successfully treated by sedation, muscular paralysis and mechanical ventilation. Public safety personnel must be aware of this potentially life threatening complication. Avoiding hobble and prone restraint position may eliminate some of the problems that contribute to the pathophysiology of this condition.

Keywords: Prone position, physical restraints, cocaine intoxication, lactic acidosis, ventilation.
**Introduction:**

When patients present with agitated delirium resulting in unusually aggressive and hyperactive behavior, physical restraints are often necessary for avoidance of self-inflicted injury and to permit adequate evaluation. Consequently, such patients may become physically exhausted secondary to the continuing struggle against restraints. Profound lactic acidosis has been reported to occur under these conditions, especially in the presence of stimulant drugs such as cocaine (1-2). These patients may also be unable to appropriately hyperventilate in response to metabolic acidosis due to restrictive positioning in a prone or hobble positions. They are also at risk for the development of a hyper catabolic state secondary to the release of catecholamines and subsequent depletion of energy stores. All of these consequences may contribute to the development of lactic acidosis, respiratory muscle fatigue, cardiac arrhythmias and cardiac arrest. This overall syndrome has been termed “restrained associated asphyxia”.

Aggressive sedation, fluid administration and mechanical ventilation may improve survival in restrained agitated patients who have profound lactic acidosis by countering the development of lactic acidosis secondary to muscle contractions and respiratory acidosis secondary to muscle fatigue (2,3). The current report describes a patient who developed profound lactic acidosis due to struggling against maximal restraint in the prone position, which was required for the treatment of agitated delirium related to acute cocaine intoxication. Aggressive sedation, intubation with mechanical ventilation and intravenous fluid
administration rapidly corrected the metabolic derangements.

**Case Report:**

A 24 year old non diabetic male presented with agitated delirium and hypertensive urgency after cocaine abuse. He required restraint in the prone position and was hand cuffed behind his back for self protection. Due to continued combativeness, the patient required sedation, muscular paralysis and mechanical ventilation.

Vital signs before intubation were: Temperature: 36.5 °C, Pulse rate 180 beats per minute and blood pressure 224/120 mmHg. Respiratory rate (RR): 28 per minute. Physical examination was otherwise unremarkable.

Laboratory evaluation immediately after intubation included a white blood cell count of 19.3×10⁹/L, hematocrit 43%, platelet count 352×10⁹/L. The serum creatinine concentration was 1.9 mg/dl and blood urea nitrogen (BUN) concentration was 9 mg/dl. Serum electrolyte concentrations (in meq/l): sodium 138, potassium 4.1, chloride 102, and bicarbonate 7. The plasma anion gap was 29 meq/l, plasma lactate level 20 mg/dl, blood glucose 327 mg/dl, albumin 4.1 gm/dl, calcium concentration 9.6 mg/dl, and creatine phosphokinase (CPK) 231 U/l. Hepatic transaminases, alkaline phosphatase, and prothrombin time were normal. Blood alcohol level 104 mg/dl. The plasma osmolality was 315 mosm/kg H₂O. The plasma osmolar gap after correction for ethanol was 5 mosm/kg H₂O. Tests for serum and urinary ketones were negative. Arterial blood gas studies showed a pH of 7.10, pCO₂ 44 mmHg, PaO₂ 327 mmHg (FIO₂ 100%). Salicylate and acetaminophen levels were undetectable. Urine drug screen was
positive for cocaine. Electrocardiogram showed sinus tachycardia with heart rate of 175 bpm. Computerized tomography of the abdomen was negative for any acute intraabdominal process. Cardiac ischemia was ruled out by serial normal EKGs and negative plasma troponins.

Case follow up:

Following intubation, mechanical ventilation, and continued sedation the patient's vital signs rapidly normalized. After three hours of mechanical ventilation and administration of 4 L of normal saline, repeat laboratory studies showed a plasma lactate level of 2.0 mg/dl, serum creatinine 1.1 mg/dl, bicarbonate 23 meq/l; plasma anion gap 4 meq/l. Results of arterial blood gas studies were: pH 7.31, pCO₂ 34 mmHg, PaO₂ 174 mmHg. CPK increased to 2000 U/l. Test for urine myoglobin was negative. Blood glucose was 70 mg/dl. After 24 hours all laboratory results were normal with complete resolution of lactic acidosis. CPK decreased to 117 U/l and the patient was weaned from sedation and extubated with no complications.

Discussion:

During intense exercise, anaerobic glycolysis in skeletal muscle is the source of energy for the synthesis of adenosine tri-phosphate (ATP) accompanied by lactate and hydrogen ion production (4). Lactic acid is rapidly buffered resulting in increased plasma lactate concentration and decreased plasma bicarbonate concentrations. Lactate is avidly conserved by the kidney and subsequently metabolized to carbon dioxide and water, primarily in the liver (4). This process
results in the regeneration of bicarbonate lost in the initial buffering of lactic acid. Plasma lactate levels may transiently be as high as 15 meq/L during a grand mal seizure and can reach 20 to 25 meq/L with maximal exercise (5). Plasma lactate concentrations peak about 5 minutes after the cessation of intense exercise and return to pre-exercise levels within an hour post-exercise after cessation of the muscle activity (6).

Profound lactic acidosis has been reported to occur in agitated, combative patients for whom maximal restraints have been required, especially in the presence of cocaine use (2). Lactic acidosis is generated by the vigorous muscle exertion in the setting of acute agitation/delirium. The additional generalized sympathetic nervous system activity induces vasoconstriction, which is augmented by the presence of cocaine leading to impaired hepatic clearance of lactate; i.e., to impaired regeneration of bicarbonate (4). Maximal restraint, which is often required during treatment, may also limit maximal compensatory hyperventilation (2,7).

Patients with agitated delirium, particularly when associated with drug abuse, often have impaired pain sensation (8), and may continue to resist and to struggle against restraints beyond normal physiologic limits. This excessive physical activity can result in a severe lactic acidosis. The excessive sympathetic drive also contributes to a hypercatabolic state. The net result is depletion of body energy stores (ATP) and impaired tissue oxygen delivery, which can lead to generalized muscle fatigue, loss of vascular tone, and sudden cardiovascular collapse (2,3,9).
Cocaine inhibits presynaptic reuptake of noradrenaline and dopamine, leading to accumulation of these neurotransmitters at the postsynaptic receptor sites. These changes lead to generalized vasoconstriction, which can contribute to tissue hypoperfusion. (10,11). The generalized vasoconstriction may contribute both to skeletal muscle ischemia and associated lactic acid generation and to impaired hepatic perfusion leading to delayed hepatic metabolism of lactate (4).

Lactic acidosis in restrained agitated cocaine intoxicated patients tends to be progressive leading to progressively impaired tissue perfusion resulting in impaired cardiac contractility, cardiac arrhythmias, and cardiac arrest, which is typically not responsive to aggressive advanced cardiac life support (ACLS) measures (2,12). Early intervention with sedation and paralytic agents to halt the vigorous muscle activity to prevent ongoing lactic acid production, mechanical ventilation to increase minute ventilation, and fluid administration to improve tissue perfusion to optimize metabolism of accumulated lactate has been associated with markedly improved patient survival (2).

Compensatory hyperventilation in response to metabolic acidosis may be limited by the restrained prone or hobble positions (2,7). Cross over studies comparing oxygen saturation, pO2 and pCO2 in restrained versus unrestrained healthy volunteers after vigorous exercise have shown no significant difference in these variables despite a 20% lower maximal ventilatory volume in the restrained position (13,14). Although volunteers in these studies were restrained
in a prone position, they did not continue to struggle during continued restraint, which differs from the real life situation when an intoxicated agitated patient is forcefully prone-restrained and continues to resist against restraints. In the setting of profound metabolic acidosis, a 20% reduction in maximal ventilatory volume may be sufficient to impair maximal compensatory hyperventilation. Bicarbonate administration in lactic acidosis is controversial (15), and has been recommended primarily for partial correction of severe academia (pH <7.1) to allow for time to correct the underlying etiology of lactic acid generation.

Conclusion: Public safety personnel must be aware of this potentially life threatening complication. Avoiding hobble and prone restraint position may eliminate some of the problems that contribute to the pathophysiology of this condition. Early intervention including sedation, muscle paralysis, fluid administration, and mechanical ventilation can be life saving. (2,7).

References:


