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Asystolic Cardiac Arrest of Unknown Duration in Profound Hypothermia and Polysubstance Overdose: A Case Report of Complete Recovery

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 20
Final Diagnosis: Asystolic cardiac arrest in profound hypothermia and poly-substance overdose
Symptoms: Cardiac arrest • cardiac arrhythmia
Medication: —
Clinical Procedure: Endotracheal intubation • hemodialysis
Specialty: Critical Care Medicine





Objective: Unusual clinical course
Background: Opioid addiction and overdose is a serious problem worldwide. Fatal overdoses from opioids are responsible for numerous deaths and are increasing, especially if taken in combination with other psychoactive substances. Combined with environmental exposure, opioid overdose can cause profound hypothermia. Opioid abuse and other drugs of abuse impair thermoregulation, leading to severe hypothermia. Both drug overdose and severe hypothermia can cause cardiac arrest.

Case Report: We report a case of 20-year-old man with history of polysubstance abuse presenting with severe hypothermia and asystole of unknown duration with return of spontaneous circulation (ROSC) achieved after 28 minutes of cardiopulmonary resuscitation (CPR). Urine toxicology was positive for cocaine, heroin, and benzodiazepine, along with positive blood alcohol level. The patient was rewarmed using non-invasive techniques. Hospital course was complicated by acute renal failure (ARF), severe rhabdomyolysis, severe hyperkalemia, ST-elevation myocardial infarction (STEMI), shock liver, coagulopathy, and aspiration pneumonia.

Conclusions: Survival with full cardiovascular and neurologic recovery after a cardiac arrest caused by drug overdose in the setting of severe hypothermia is still possible, even if the cardiac arrest is of unknown or prolonged duration. Patients with severe hypothermia experiencing cardiac arrest/hemodynamic instability can be rewarmed using non-invasive methods and may not necessarily need invasive rewarming techniques.

MeSH Keywords: Acute Kidney Injury • Heroin • Hypothermia • Rhabdomyolysis

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/893880>

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Background

In 2013, 9.4% of the total USA population aged 12 years or older (about 24.6 million) were illicit drug users. This rate of illicit drug use in 2013 was higher than in 2002–2009 and in 2011, but similar to 2010 and 2012. Illicit drugs include marijuana, cocaine, heroin, hallucinogens, inhalants, or prescription psychotherapeutic drugs used non-medically. In the USA in 2013 there were 1.5 million (0.6%) cocaine users and 681 000 heroin users aged 12 years or older [1]. Opioid addiction and overdose is a serious problem worldwide. Fatal overdoses from opioids are responsible for numerous deaths and are increasing, especially when taken in combination with other psychoactive substances.

Case Report

A 20-year-old man with past medical history of polysubstance abuse was found unresponsive by his girlfriend in a bathtub full of cold water at around 2 PM and she called emergency medical service (EMS) immediately. EMS on arrival found the patient unresponsive with no pulse and in asystole on initial rhythm strip. Cardiopulmonary resuscitation (CPR) as per advanced cardiac life support (ACLS) protocol was initiated and intubation was done in the field. The ROSC was achieved in 28 minutes and the patient was transported to the hospital emergency room (ER). The patient had been repeatedly snorting heroin at 2 AM on the day of presentation.

In the ER, physical examination revealed systolic blood pressure of 62, pulse 56, respiratory rate 16, and temperature of 21.6°C (70.9°F). The patient was unresponsive to both verbal and physical stimuli, and his pupils were 4 mm and reactive to light. Further physical examination yielded no abnormalities; in particular there were no stigmata of intravenous drug use. Patient was noted to be biting the intubation tube, so Ativan and Vecuronium were given for sedation.

The initial laboratory work-up showed venous blood gas (VBG) with pH of <6.9 (7.32–7.42), pCO₂ 137 mm/Hg (38–50 mm/Hg), HCO₃ 14.4 mmol/L (22–28 mmol/L), and lactate 11.2 mmol/L (0.5–2.2 mmol/L). Blood urea nitrogen (BUN) was 21 mg/dl (8–2 mg/dl) and creatinine was 2.9 mg/dl (0.4–1.6 mg/dl), which increased to 115 mg/dl and 12.71 mg/dl, respectively. Peak levels of Troponin and CPK were 50.67 ng/ml (≤0.04 ng/ml) and 45000 IU/L (26–189 IU/L), respectively. Liver function test showed AST 5051 IU/L (10–40 IU/L) peaking to 6808 IU/L, ALT 4261 IU/L (7–50 IU/L) with peak of 4966 IU/L, GGT 189 IU/L (9–50 IU/L), LDH >6000 IU/L (90–225 IU/L), total bilirubin 2.7 mg/dl (0.0–1.5 mg/dl), and conjugated bilirubin 1.54 mg/dl (0.0–0.3 mg/dl). Hepatitis panel was negative. Urine toxicology was positive for opiates, cocaine, and benzodiazepine.

Blood alcohol level was 38 mg/dl. EKG revealed ST elevation in leads V1 and V2 with reciprocal changes ST depression in multiple leads and large-amplitude T waves.

Central venous access was obtained and aggressive fluid resuscitation along with presser support was started. Blood pressure improved, with mean arterial pressure in 50s mmHg. The patient was rewarmed using noninvasive rewarming techniques according to hypothermia protocol. A Foley catheter was placed and orange-tinged urine was noted. After stabilization, the patient was transferred to the intensive care unit (ICU).

Hypothermia protocol was completed and the patient remained normothermic. On day 3 of the hospitalization, pressers were slowly tapered and discontinued. The patient awoke on day 3, started following simple commands and was extubated on day 6. He developed many complications, including acute kidney injury (AKI) requiring multiple sessions of hemodialysis (HD); septal STEMI likely due to cocaine, which was managed medically; and aspiration pneumonia.

On day 7 the patient was transferred from the ICU to the regular medicine floor and he continued to improve clinically. The lab results showed progressive improvement. After 7 days of ICU stay and 17 days of floor stay, the patient walked out of the hospital with full neurologic and cardiovascular recovery. The liver and renal function test results returned to normal on outpatient follow-up.

Discussion

In 1980 Frederick et al. reported a case of severe hypothermia presenting with asystole and no signs of life. He was intoxicated with alcohol and was found sleeping in the hallway with windows open and no heat when the outside temperature was –16°C. After more than 2 hours of continued cardiopulmonary resuscitation and rewarming, heart rhythm changed from asystole to junctional rhythm. Finally, the patient walked out of the hospital after 40 days [2]. Similar to the above case report, our patient had severe hypothermia and asystole of unknown duration, with ROSC achieved after 28 minutes of CPR. Along with positive blood alcohol level, urine toxicology was positive for cocaine, heroin, and benzodiazepine.

Hypothermia is defined as core body temperature below 35.0°C (95°F), at which normal body metabolism and functioning cannot be performed. Mild hypothermia is defined as temperature between 35°C (95°F) and 32.2°C (90°F), moderate hypothermia is <32.2°C (90°F) to 28°C (82.4°F), and severe hypothermia is <28°C (82.4°F). Severe hypothermia can lead to depression of cerebrovascular autoregulation, coma, decrease in blood pressure and heart rate, arrhythmias, asystole,

respiratory depression leading to apnea, oliguria, and peripheral areflexia [3]. The Swiss staging system is used to classify hypothermia clinically into 4 stages on the basis of vital signs. The Swiss staging system is preferred over the former staging when core body temperature readings are not available in the prehospital setting [4]. Severe hypothermia acts on Purkinje fiber and can cause asystole, possibly by decreasing resting membrane potential, slowing conduction velocity, prolonging absolute and refractory periods, and slowing the rate of spontaneous depolarization [3].

Patients can develop severe metabolic acidosis in hypothermia due to increased lactate production and decreased clearance. Lactate production is increased because of decreased tissue oxygenation due to left shift of the oxy-hemoglobin dissociation curve, vasoconstriction, and increased blood viscosity. Decreased clearance of lactate is due to impaired hepatic function and impaired acid excretion [3].

Cocaine has been associated with various cardiac arrhythmias, including ventricular fibrillation and asystole [5]. Cocaine is well known to cause myocardial infarction in the absence of coronary artery disease. Our patient had septal ST elevation myocardial infarction with peak troponin levels of 50 ng/ml (normal value <0.04 ng/ml).

Heroin is commonly known by street names of H, smack, boy, horse, brown, black, and tar. Heroin intoxication causes depressed mental status ranging from drowsiness to stupor and coma, respiratory depression, and, in the worse cases, respiratory failure. Heroin also has significant cardiovascular effects leading to hypotension, bradycardia, and cyanosis. It also causes impaired thermoregulation, sometimes leading to severe hypothermia [6], which is usually in combination with environmental exposure. In obtunded/comatose patients, even room temperature can cause profound hypothermia. Opioids have analgesic, euphoric (via elevating dopamine levels in the mesolimbic system), and anxiolytic effect (via noradrenergic neurons located at the locus ceruleus [7]).

Obtundation caused by illicit drugs and alcohol leads to prolonged immobilization, causing pressure-related ischemic muscle injury due to vascular occlusion, leading to rhabdomyolysis. Psychoactive substances such as cocaine, heroin, and alcohol also have direct muscle toxicity. In addition, the sympathomimetic action of cocaine causes vasoconstriction potentiating further ischemic muscle damage. The decreased ATP production due to hypoxia leads to increased calcium influx, causing cell lysis and release of creatinine kinase (CK). Myoglobin is then released from injured myocytes, giving a dark red/brown color to urine. Myoglobin generates free radicals, which are nephrotoxic and also causes tubular obstruction when combined with Tamm-Horsfall protein, forming pigment casts leading to

acute renal insufficiency. Rhabdomyolysis accounts for 10% of the cases of AKI [8].

Management of such patients is complicated and needs a multidisciplinary approach. The literature strongly recommends that invasive rewarming should be reserved for patients having severe hypothermia accompanied with cardiac arrest and/or hemodynamic instability [9]. However, our patient had severe accidental hypothermia accompanied by cardiac arrest, and was rewarmed using noninvasive methods with good clinical outcome.

Patients with multidrug overdose present with a complicated clinical picture that does not fit any toxidrome. In the management of overdose, it is important to obtain a history and physical examination, but the patient's "ABC" should first be stabilized. If intubation is needed, a non-depolarizing agent (Vecuronium) should be used, especially if hyperkalemia is suspected. The "coma cocktail" includes dextrose, naloxone, and thiamine (NDT) [10]. However, naloxone administration in polydrug abuse due to opioid and sympathomimetic drugs like cocaine should be avoided because naloxone antagonizes opioid CNS depressant effects and causes extreme sympathomimetic toxicity. Naloxone itself also has arrhythmogenic potential [11]. Hypoglycemia must be ruled out in a patient with altered mental status (AMS). Patient with AMS should be given intravenous thiamine; however, holding dextrose administration before thiamine administration should not be a priority in hypoglycemic patients because dextrose is readily taken up by cells compared to thiamine and there is no point of waiting for thiamine administration [12,13].

Complications like aspiration pneumonia, rhabdomyolysis, AKI, and electrolyte disturbance should be managed appropriately. The mainstay of treatment of rhabdomyolysis is prompt and aggressive fluid replacement with a goal of urine output of 200–300 ml/h. Use of bicarbonate and mannitol have little or no benefit in the treatment of AKI due to rhabdomyolysis. Loop diuretics may be helpful, especially in patients with hyperkalemia. Renal replacement therapy is the last resort if noninvasive measures to treat AKI fail. If rhabdomyolysis is associated with AKI, chances of mortality and hospital stay increase [8].

A prior study done on heroin overdose and cardiac arrest concluded that survival after cardiac arrest caused by heroin overdose is possible if the arrest is EMS-witnessed or if EMS is called before the cardiac arrest occurs [14], but our case shows that full recovery is still possible even if the cardiac arrest is of unknown duration or is unwitnessed. Another study on drug overdose and cardiac arrest was done for age, etiology, rhythm, and outcome. The total number of subject was 8054, out of which 252 were 18–35 years old. In this age group, 24% of the cardiac arrests were caused by drug overdose and the

outcome was worse in patients who had asystole (4% survival) than in patients who had ventricular fibrillation or tachycardia (28% survival), indicating that the prognosis depends on rhythm rather than age [15].

Conclusions

Survival after a cardiac arrest caused by drug overdose in the setting of severe hypothermia is still possible even if the arrest is of unknown or prolonged duration. Patients with severe hypothermia having cardiac arrest/hemodynamic instability can be rewarmed using non-invasive methods and do not necessarily need invasive rewarming, as is shown by our case. So

that rewarming can be initiated in these patients with non-invasive means without waiting for invasive rewarming techniques, Naloxone can antagonize opioid CNS depressant effect, causing extreme sympathomimetic toxicity, and it should be given with caution in suspected polysubstance abuse. In patients with altered mental status, glucose administration may not have to wait for thiamine administration, especially in hypoglycemic patients, since uptake of thiamine by cells is slow even if it is given first.

Statement

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