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Acute Methadone and Cocaine Poisoning Complicated by Cardiac Arrest: Case Report

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Summary

Objective: to evaluate the effects of correcting metabolic disorders in the post-resuscitation period in a clinical case report.

Materials and methods. Management and dynamic monitoring of acute poisoning in a 29-year-old patient after concomitant use of methadone and cocaine complicated by cardiac arrest and prehospital biochemical and acid-base balance alterations.

Results. The bradypneic and comatose patient developing out-of-hospital cardiac arrest (OHCA) was hospitalized after effective resuscitation by emergency team. Comprehensive lab examination revealed the presence of methadone and cocaine, decompensated metabolic lactic acidosis and hyperkalemia. Patient's condition improved after intense correction of metabolic alterations with sodium hydrocarbonate, a glucose-insulin mixture, and a pharmaceutical containing inosine + nicotinamide + riboflavin + succinate. A positive trend including recovered consciousness, switch from ventilator support to spontaneous breathing, and stable hemodynamics was documented after 3 days of treatment. However, emerging complications such as hospital-acquired pneumonia and acute kidney injury had to be managed. The patient improved significantly by the 17th day of treatment, and was discharged on day 21.

Conclusion. Intensive care to promptly address decompensated metabolic lactic acidosis (sodium hydrocarbonate, a multi-component drug containing inosine + nicotinamide + riboflavin + succinate) and hyperkalemia (glucose-insulin solution), reduced the severity of metabolic alterations after cardiac arrest due to acute methadone and cocaine poisoning, favoring patient's outcome.

Keywords: acute poisoning; methadone; cocaine; cardiac arrest; sodium hydrocarbonate, succinate; Cytoflavin

Conflict of interest. The authors declare no conflict of interest. NTFF POLYSAN LLC did not initiate the study and had no influence on the study design, analysis of the obtained data, interpretation of the results and writing the manuscript.

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Introduction

Currently, cardiac arrest assignable to various causes remains an urgent problem in resuscitation due to high rate of disability and fatal outcomes [1, 2]. According to published data, the survival rate in out-of-hospital cardiac arrest (OHCA) does not exceed 7–10% [3, 4]. Acute poisoning with the opioid drug methadone is the most common cause of death among all poisoning — associated OHCA cases. According to the Center for acute poisoning at the I. I. Dzhanelidze Research Institute of Emergency Medicine (St. Petersburg), in 2022, acute

methadone poisoning accounted for 38.5% of all poisoning-related deaths, in 2023, it was 42.4%, and in 2024 — already for 40%. In addition to the specific mechanisms of toxicant damage, emerging severe metabolic disorders significantly exacerbate the clinical course of poisoning [5], eventually resulting in fatal outcomes of chemical injury [6]. Polydrug poisoning following simultaneous use of narcotic substances with conflicting mechanisms of action, for example, with depressing and stimulating effects (pendulums) should be mentioned as very dangerous and unfavorable in terms of outcomes. According to our data, recently there has been an increase in acute poisoning with mixtures of methadone and other psychostimulant narcotic substances, however, in available literature we failed to find info on specific clinical features of such poisonings.

Methadone (6-dimethylamino-4,4-diphenyl-3-heptanone) is a synthetic substitute for opioid alkaloids (a synthetic opioid) [6]. Cocaine belongs to the group of psychoactive and sympathomimetic stimulants of the central nervous system [7, 8].

Presented clinical case could be of interest for specific intensive care management of acute severe poisoning by a mixture of narcotic substances with conflicting mechanisms of action (depressant toxicant methadone and stimulant toxicant cocaine), complicated by the development of OHCA.

Timely initiated extended cardiopulmonary resuscitation in the ambulance and continued in the hospital included chest compressions, mechanical ventilation, intravenous administration of epinephrine, infusion therapy, and syndrome-specific therapy. Measures were also taken to reduce mixed decompensated metabolic acidosis, hyperkalemia, and provide pharmacological neuroprotection using a multicomponent pharmaceutical containing inosine + nicotinamide + riboflavin + succinate (Cytoflavin, POLISAN LLC).

Objective: to evaluate the effects of correcting metabolic disorders in the post-resuscitation period in a clinical case report.

Results

Patient K., 29 years old, was admitted to reanimatology and intensive care department No. 11 (toxicology) (ICU No. 11) of the I. I. Dzhanelidze Research Institute of Emergency Medicine on February 14, 2023, at 02:48, in a critical condition.

Medical history: according to the data provided by the ambulance doctor, the patient was found at home with impaired (very close to coma) consciousness, severe bilateral non-reactive miosis and severe respiratory depression with 2–3 breaths per minute and a $SPO2_2$ level of 20% at the time of initial medical examination. Based on these findings the emergency medical team doctor diagnosed «opioid syndrome» (as depressed consciousness, bradypnea, and miosis are specific characteristics of acute opioid drug poisoning). The presence of an insulin syringe near the patient and a post-injection mark on the left upper limb were additional confirmation of the diagnosis of acute opioid poisoning. Antidote naloxone was not administered by the ambulance team.

Five minutes after tracheal intubation and initiation of mechanical ventilation, the patient experienced a cardiac arrest, and the ECG showed asystole. The ambulance team performed extended cardiopulmonary resuscitation (CPR), including chest compressions, continuous mandatory ventilation (CMV mode; FiO_2 — 100%, PEEP — 5 cm of water), administration of epinephrine 1 mg bolus each 3 minutes No. 3 and 12 mg dexamethasone, infusion of glucose-insulin mixture and 10 ml of multicomponent drug containing inosine + nicotinamide + riboflavin + succinic acid. The sinus rhythm was restored after 10 minutes of resuscitation, and dopamine was administered at a rate of 10 μg/kg/min.

An extremely severe condition on admission to ICU No. 11 resulted from severe toxic-hypoxic encephalopathy, clinically manifested by deep atonic coma (3 scores on the Glasgow Coma Scale (GCS), and acute respiratory failure (ARF) necessitating mechanical ventilation, with acute circulatory failure, which was treated with dopamine at a dose of 10 μg/kg/min. Inotropic and chronotropic effects of dopamine were acting on cardiotoxic effects of methadone, manifested as bradycardia and hypotension. Chemical and toxicological examination using gas chromatography-mass spectrometry revealed methadone and cocaine in the patient's urine.

Laboratory tests showed significant increase in the white blood cell (WBC) count (23.94×10^9/l, reference range $4.0–9.0\times10^9$/l). Biochemistry panel revealed an increase in creatine kinase to 1794 units/L (reference 7.0–190.0 units/L) and creatinine to 275 μmol/L (reference 60.0–120.0 μmol/L), indicating systemic rhabdomyolysis and acute kidney injury. Upon admission, serum concentration of myoglobin was 84 μg/L (reference 23.0–72.0 μg/L). Analysis of blood plasma electrolytes revealed severe hyperkalemia, reaching up to 6.14 mmol/L (reference 3.5–5.0 mmol/L). The arterial blood gas (ABG) measurements showed altered acid-base balance (ABB) due to hypoxemia and decompensated metabolic lactate acidosis: pH — 6.98; pCO_2 — 37.6 mmHg, pO_2 — 69 mmHg (with a fraction of inhaled oxygen (FiO_2) 80%), SO_2 — 86.1%, HCO_3 — 8.6 mmol/l, BE (base excess) — –23 mmol/l, arterial blood lactate — 19.8 mmol/l (reference 0.6–1.4 mmol/l). A low value of the Horowitz index — 86.3 mmHg confirmed impaired

transpulmonary oxygen transport and inadequate gas exchange function.

Chest X-ray showed enhanced pulmonary pattern on admission.

The patient's diagnosis on admission was as follows: «Severe acute poisoning with a mixture of narcotic substances (methadone, cocaine). Toxic-hypoxic encephalopathy. Coma stage 3. Complications: Acute respiratory failure. Acute circulatory failure. Cardiac arrest on February 14, 2023. Systemic rhabdomyolysis. Acute kidney injury».

In the hospital, the patient was managed on mechanical ventilation in backup modes under continuous ABG and ABB monitoring and dopamine support at a dose of 10 μg/kg/min. Decompensated metabolic acidosis was corrected by administering 4% sodium bicarbonate. The amount of required bicarbonate was calculated using the Mellengaard–Astrup equation [9]. According to calculation, the amount of required hydrocarbonate was 552 mmol, and 2000 ml/day of glucose-insulin mixture (12 IU of insulin added to each 500 ml of 10% glucose) was infused to correct hyperkalemia. A multi-component pharmaceutical Citoflavin (inosine, nicotinamide, riboflavin, and succinic acid) at a dose of 10 ml per each 500 ml was added to the glucose-insulin solution to provide neurometabolic support and decrease the lactate level. The therapeutic regimen also included proton pump inhibitors (omeprazole lyophilisate at a dose of 40 mg dissolved in 100 ml of 5% glucose solution, administered intravenously over a period of 30 minutes once daily), thiamine 50 mg administered intramuscularly once daily, and continuous intravenous infusion of heparin Na at a rate of 10 IU/kg/h during patient's management in the ICU. To interrupt enterohepatic circulation of methadone, the patient was subjected to gastric lavage and cleansing enema, administered enterosorption and laxatives (Duphalac 50.0 ml).

In view of deterioration caused by progression of circulatory failure 3 hours after admission, the patient's dose of dopamine was increased to 15 micrograms/kg/min. However patient's toxic-hypoxic encephalopathy and impaired consciousness persisted, assessed as grade 3 coma (GCS 3). Continuous infusion therapy improved baseline metabolic alterations, allowing de-escalation of the dopamine support dose to 7 μg/kg/min after 4 hours, and to 5 μg/kg/min after 12 hours of infusion.

Stabilization of systemic hemodynamic parameters was achieved 24 hours after the initiation of treatment, and dopamine support was discontinued. Toxic-hypoxic encephalopathy has also diminished allowing patient's consciousness to improve to the level of stupor (GCS 10 scores). A simultaneous decrease in WBC count to 11×10^9/l was also documented. Positive shifts in ABG and ABB were also evident, based on lab tests data, including: pH = 7.341, pCO_2 = 60.2 mmHg, pO_2 = 110.9 mmHg, SO_2 = 97.9%, HCO_3^- = 31.8 mmol/L, BE = 4.7 mmol/L, with a fraction of inhaled oxygen (FiO_2) of 40%, and a decrease of potassium to 3.7 mmol/L. There was also a decrease of arterial blood lactate to 3.2 mmol/L and an improvement in pulmonary gas exchange function (an increase in the Horowitz index to 275 mmHg). However, biochemistry panel indicated to deterioration of kidney function, i.e. progression of impaired nitrogen excretion, resulting in increases of creatinine to 337 mmol/l (reference 60.0–120.0 mmol/l), and urea — to 20.3 mmol/l (reference 0–8.3 mmol/l); worsening of cytolysis syndrome evidenced by aspartate aminotransferase increase to 6844.5 u/l (reference is up to 31 u/l) and systemic rhabdomyolysis (an increase of creatine kinase to 10545 u/l (reference is 7.0–190.0 u/l) and myoglobin to 7856 mcg/l).

A relative decrease in daily urine output to 1000 ml/day was documented along with above-mentioned lab findings compared to daily infused volume of 3200 ml, which was associated with the progression of acute kidney injury due to escalating systemic rhabdomyolysis. To remove circulating myoglobin, a hemodialysis was administered using membranes with a high cutoff point and the following parameters: perfusion rate of 300 ml/min, ultrafiltration rate of 1.0 L/h, dialysate flow of 500 ml/min, sodium conductivity of 140 mmol/l, perfusion time of 240 min, 72 L of blood were processed, 4.0 L of fluid were removed, and the fluid deficit was 3.0 L. Lab test performed 6 hours after hemodialysis showed decrease of creatine kinase to 3348 U/L and of myoglobin — to 3153 μg/L. On day 6 of intensive care including 3 hemodialysis sessions the concentrations of creatine kinase decreased to 263 U/L and of myoglobin — to 81 μg/L. Symptoms of encephalopathy resolved by the third day, the patient recovered spontaneous breathing and was extubated. However, new complications emerged on the same day, including in-hospital ventilator-associated pneumonia requiring administration of antibacterial therapy, and deterioration of acute kidney injury, necessitating five sessions of hemodialysis, after which renal function recovered on the 17th day, as evidenced by a decrease of creatinine to 194 μmol/L and urea to 8.9 mmol/L.

The patient was discharged from the hospital on the 21st day in a satisfactory condition.

Therefore, effective resuscitation measures provided by the emergency medical team during prehospital care alongside with effective correction of metabolic disorders (decompensated metabolic lactic acidosis, hyperkalemia, and hyperlactatemia), and the use of hemodialysis in the ICU setting resulted in favorable outcome for the patient experiencing acute poisoning with a mixture of narcotic

substances (methadone, cocaine), complicated by cardiac arrest.

Discussion

The available published data on acute poisoning with a mixture of narcotic substances containing opioids and cocaine only allows to conclude that such a combination is extremely unfavorable, and clinical course of concomitant poisoning is understudied [10–12]. Therefore, this clinical case is interesting in terms of pathogenesis of both — the critical state, including acute respiratory failure, cardiac arrest and coma due to acute methadone and cocaine poisoning, and the recovery from it owing to adequate intensive care in the ICU setting.

One of major causes of cardiac arrest was unfavorable combination of narcotic substances with conflicting mechanisms of action: the methadone depressant effect on the respiratory center and cardiovascular system leading to bradypnea and bradycardia, and the stimulating effect of cocaine, which provokes tachycardia, arterial hypertension, and an increase in myocardial oxygen demand. In our opinion, this pathophysiological «conflict» was one of the leading causes of OHCA.

Systemic rhabdomyolysis due to severe hypoxia caused by methadone-induced respiratory depression, which further led to severe mixed respiratory and metabolic acidosis could also contribute to circulatory failure [13], enhanced by psychostimulant effect of cocaine, which increased tissue oxygen demand due to the direct cytotoxic effect of cocaine on skeletal muscles [14].

Moreover, massive damage to muscular tissue was another factor that exacerbated decompensated metabolic acidosis and hyperkalemia, which were also key factors leading to cardiac arrest. After successful resuscitation outside of a medical facility, the patient developed acute kidney injury, which had both a pre-renal component (due to circulatory arrest), and a renal component caused by myoglobinuria due to systemic rhabdomyolysis [13, 14]. This determined the need for renal replacement therapy as an integral part of the patient's management leading to recovery.

It should be noted that the medical interventions not only saved the patient's life but also minimized the consequences of the circulatory arrest. Firstly, the timely and effective resuscitation measures outside the hospital laid the foundation for further successful treatment. Secondly, rapid correction of metabolic disorders such as decompensated metabolic acidosis (using sodium bicarbonate), hyperkalemia (using a glucose-insulin mixture), and lactatemia (using a multi-component drug: inosine + nicotinamide + riboflavin + succinic acid).

It should be especially noted that the routine use of sodium hydrocarbonate is not recommended currently, according to guidance of the European Resuscitation Council from 2021 [15] and the American Heart Association from 2021 [16]. However, the exceptions to recommended algorithm in notes to item 25 in «Cardiac Arrest» (Adult Patients) [17] clinical guidelines include cardiac arrest in acute opioid drug poisoning, so the use of sodium hydrocarbonate was one of the basic components of treatment. In our opinion, the use of a multicomponent drug (inosine + nicotinamide + riboflavin + succinic acid) in the acute stage significantly reduced the energy deficiency of brain cells, which had a positive effect on brain functions recovery in the post-resuscitation period [18, 19].

Conclusion

This clinical case demonstrated the effectiveness of a comprehensive approach to the intensive care of a patient with combined acute poisoning by drugs with conflicting mechanism of action (methadone, cocaine), complicated by cardiac arrest.

The main factors contributing to a favorable outcome were timely and high-quality advanced resuscitation measures implemented at the prehospital stage and in the ICU setting; correction of life-threatening metabolic disorders (lactic acidosis, hyperkalemia); active detoxification; and pathogenetic therapy aimed at correcting energy metabolism.

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