SEVERE ACUTE α-PVP POISONING COMPLICATED BY SYSTEMIC RHABDOMYOLYSIS SYNDROME: CASE REPORT

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Today, acute poisoning with modern psychoactive substances and narcotic drugs is among the major causes of emergency admissions to toxicology units. There is a clear upward trend in the number of life-threatening complications of poisoning with psychoactive substances, one of which is rhabdomyolysis. Here we report a clinical case of severe acute α-PVP poisoning complicated by systemic rhabdomyolysis, describe the features of the clinical course and intensive care for this disorder. The early diagnosis, timely and effective treatment by conducting aggressive detoxification infusion therapy made it possible to prevent progression to the anuric phase of acute kidney injury, as well as to avoid invasive procedures of extracorporeal detoxification methods and probably avoid the adverse outcome of the acute α-PVP poisoning complicated by systemic rhabdomyolysis.

Keywords: cathinones, alpha-PVP, α-PVP, α-pyrrolidinovalerophenone, acute poisoning signs and symptoms, diagnosis, treatment, rhabdomyolysis

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КЛИНИЧЕСКИЙ СЛУЧАЙ ОСТРОГО ТЯЖЕЛОГО ОТРАВЛЕНИЯ α-PVP, ОСЛОЖНЕННЫЙ РАЗВИТИЕМ СИНДРОМА СИСТЕМНОГО РАБДОМИОЛИЗА

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В настоящее время острые отравления современными психоактивными веществами и наркотическими веществами входят в число основных причин экстренной госпитализации лиц в отделения токсикологии. Имеется четкая тенденция к росту числа жизнеугрожающих осложнений при отравлениях психоактивными веществами, одним из которых является рабдомиолиз. Представлен клинический случай острого тяжелого отравления альфа-PVP, осложненного развитием системного рабдомиолиза, описаны особенности клинического течения и интенсивной терапии при данной патологии. Ранняя диагностика, своевременное и эффективное лечение путем проведения агрессивной инфузионно-детоксикационной терапии позволили избежать развития анурической стадии острого повреждения почек, а также отказаться от проведения инвазивных процедур экстракорпоральных методов детоксикации и, вероятно, избежать неблагоприятного исхода острого отравления α-PVP, осложненного развитием системного рабдомиолиза.

Ключевые слова: катиноны, альфа-PVP, α-PVP, α-пирролидиновалерофенон, клиника острого отравления, диагностика, лечение, рабдомиолиз

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Today, acute poisoning with narcotic drugs occupy leading positions in the structure of the patients' admission to toxicology units; there is no trend towards the decrease in the number of such patients, which determines high relevance of the issue.

Among the most numerous are cases of acute poisoning with synthetic cathinones ("designer drugs" in slang) showing strong psycho-stimulating activity and the high rate of life-threatening conditions affecting the CNS, respiratory and cardiovascular system [1]. Thus, based on the epidemiological situation in Saint Petersburg, a total of 2596 patients poisoned with the synthetic cathinone α-pyrrolidinovalerophenone (α-PVP) were delivered to the Acute Poisoning Center of the Dzhanelidze Research Institute of Emergency Care in 2024, which far exceeded the number of cases of acute poisoning

with other drugs, such as methadone (a total of 1422 patients were admitted in 2024), cocaine (a total of 68 patients were delivered within the same period). All the above emphasizes predominance of acute α-PVP poisoning in the structure of acute poisoning at the moment.

The α-PVP properties were first reported in 1963 by researchers of the Boehringer Ingelheim pharma company (Germany) [2]. However, later successful therapeutic use of α-PVP was not confirmed. Since early 2010s, the α-PVP chemical compound appeared on the illegal drug market in European countries, where it was distributed under the guise of "bath salts" (that is why it is referred to as "salt"), then it was noted to be distributed in Moscow, Saint Petersburg, and Russia [3]. Considering social dangerousness of this toxicant, the α-PVP turnover is currently controlled in 16 countries of the European Union and prohibited in the Russian Federation [4].

The recreation effects of synthetic cathinones, including α-PVP, are associated with the strong stimulating effect on the CNS and cardiovascular system. In individuals with the extremely severe poisoning, along with the toxic damage to the nervous and cardiovascular systems, the condition severity is aggravated by the early development of severe metabolic disorders in the form of decompensated metabolic acidosis, coagulopathy [5, 6]. Systemic rhabdomyolysis that is usually accompanied by acute kidney injury represents one of the most severe complications of severe acute α-PVP poisoning.

Rhabdomyolysis is a syndrome characterized by muscle necrosis and the release of the muscle intracellular components into bloodstream [1]. The development of systemic rhabdomyolysis being one of the most severe complications of acute poisoning, including poisoning with narcotic drugs, is currently poorly understood [3]. The study aimed to present a clinical case of acute α-PVP poisoning complicated by systemic rhabdomyolysis with identification of early diagnostic criteria and the efficacy of the conservative therapy applied.

Clinical case

Patient M., male, 23 years old, was admitted to the Acute Poisoning Center of the Dzhanelidze Research Institute of Emergency Care with the following referral diagnosis: "Moderate acute poisoning with the unknown neurotropic substance". The patient examination revealed mental confusion, psychomotor agitation, disorientation in space, time and own personality. Conciousness scored +3 on the RASS scale. To jugulate psychomotor agitation, the patient was intramuscularly administered Elzepam 2.0 mg. Hemodynamics were stable, heart rate (HR) was 125 bpm, the rhythm was sinus tachycardia, blood pressure was 150 and 80 mmHg.

The clinical laboratory testing revealed leucocytosis up to 26.58×10^{12}/L with the neutrophil right shift in complete blood counts, along with the increase in creatinine levels to 227 μmol/L and urea levels to 19.7 mmol/L in the blood biochemistry panel. Urinalysis revealed proteinuria (with the increase in protein levels to 0.75 g/L), hematuria (with the increase in red blood cell counts to 2.5×10^2/μL). Chemical toxicology testing by gas chromatography–mass spectrometry (GC–MS) detected α-PVP in the urine.

Within 12 h after admission the patient was transferred to the intensive care unit (ICU) No. 11 due to deterioration in his condition. The patient's overall condition was assessed as severe at admission to the ICU. Pupil size D = S, mydriasis. Lively papillary light response. Normal muscle tone. Normal reflexes. No abnormal reflexes. Respiratory rate 16 breaths per minute. Oxygen saturation 98%. Stable nemodynamics, HR was 89 bpm, blood pressure was 120 and 70 mmHg, central venous pressure was (+2) cm H_2O. A total of 100 mL of urine were collected by bladder catheterization. The urine color was brown. The following diagnosis was established: "Severe acute α-PVP poisoning. Toxic encephalopathy. Complication: Systemic rhabdomyolysis. Acute kidney injury".

On day 2 after admission the decrease in leucocyte counts to 14.9×10^{12}/L was reported; as for blood biochemistry parameters, worsening in the form of the increase in creatinine levels to 235 μmol/L and urea levels to 25.1 mmol/L was noted, along with the increase in cytolytic enzyme levels (ALT to 462.6 U/L, AST to 1782 U/L), creatine kinase levels to 88,980 U/L and hyperkalemia to 5.4 mmol/L. Along with negative values of blood biochemistry parameters, the urine output decrease to 100 mL/day was reported, which corresponded to stage 2 acute kidney injury according to the KDIGO classification [5].

Furthermore, when assessing the patient's mixed venous blood gas and acid-base state, we observed the development of subcompensated metabolic acidosis with the electrolyte balance disturbances: pH — 7.285, HCO_3^- — 16 mmol/L, BEb — (–9.3) mmol/L, BEect — (–10.6) mmol/L, Na^+ — 133 mmol/L, K^+ — 4.49 mmol/L, Ca^{2+} — 1.018 mmol/L, blood osmolality — 266 mOsm/L.

Improvement of a number of parameters was reported within 6 h after the beginning of the intensive metabolic acidosis correction: pH — 7.523, HCO_3^- — 27.9 mmol/L, BEb — 5.1 mmol/L, BEect — 5.1 mmol/L, K^+ — 2.87 mmol/L.

The infusion therapy prescribed included the use of infusion solutions to adjust metabolic acidosis (Sol. Natrii hydrocarbonatis 4% — 200.0 No. 3). Sodium hydrocarbonate was quantified using the Mellengaard-Astrup formula: the required amount of sodium hydrocarbonate (in mmol) is equal to the product of body weight in kilograms by 0.3 by BE (base excess) [7]. According to calculations, the required hydrocarbonate quantity was 297 mmol, which was 623.9 when recalculated for the 4% sodium hydrocarbonate solution. Hyperkalemia was adjusted using the glucose-insulin solution (Sol. Glucosae 10% — 500.0 + Sol. Magnii sulf. 25% — 10.0 + Insulini 2 ED No. 3/day). Lactic acidosis was adjusted via infusion of Sol. Reamberini 500.0 No. 2/day. Sol. Natrii chloridi 0.9% — 500.0 No. 2/day was prescribed to adjust the volemic status. The infusion therapy volume was calculated based on the urine output, which was 1 mL/kg/h (the daily volume reached 2160 mL), perspiration losses calculated as 0.5 mL/kg/h and hyperthermia over 38 °C (0.5 mL/kg/h — 1580.0). After the first 24 h of hospital stay the daily urine output was 2900 mL/day. Furthermore, the urine color changed from black to straw-yellow. In response to the infusion therapy applied, the daily urine output became 3900 mL with the central venous pressure of (+4) cm H_2O on day 2 after the beginning of treatment.

On day 4 of intensive care, improvement was noted in the form of the decrease in creatine kinase levels to 35,000 U/L, cytolytic enzyme levels (ALT to 372.7 U/L, AST to 760 U/L). The complete blood count test revealed the decrease in leucocyte counts to 6.4×10^{12} L. The daily urine output was 5200 mL with the central venous pressure of (+8) cm H_2O.

On day 5 of treatment, the blood biochemistry test showed improvement in the form of the decrease in creatinine levels to 183 μmol/L, urea levels to 10.9 mmol/L, creatine kinase levels to 14,220 U/L, K^+ levels of 3.35 mmol/L. The daily urine output was 6200 mL, and the central venous pressure was (+8) cm H_2O.

On day 8, the patient was transferred to the toxicology unit and discharged home in a satisfactory condition.

Thus, the early diagnosis and timely, effective intensive care made it possible to avoid progression to the anuric phase of acute kidney injury and probably avoid the adverse outcome of the acute α-PVP poisoning complicated by systemic rhabdomyolysis.

Clinical case discussion

This case report demonstrates the development of systemic rhabdomyolysis in acute α-PVP poisoning, resulting from the combination of the direct and indirect factors causing the early development of this life-threatening condition. Myoglobin is the main endotoxin associated with systemic rhabdomyolysis [8–10]. Normally, myoglobin is filtered freely in the renal glomeruli, but under conditions of metabolic acidosis and hypovolemia it precipitates to form insoluble compartments that obturate the tubule lumen, thereby causing myoglobinuric acute kidney injury (AKI), as observed in the patient (urine color change to brown, oliguria, increase in creatinine and urea levels). The increase in creatine kinase levels is a diagnostic marker of damage to the skeletal muscles. In this case, these increased to 98,780 U/L, which was a diagnostic criterion of systemic rhabdomyolysis.

Given the above, in this clinical case, treatment was based on the aggressive detoxification infusion therapy aimed to ensure the rapid and effective metabolic acidosis adjustment by using sodium hydrocarbonate and then Reamberin, the infusion medication with the base reserve, massive infusion therapy with strict monitoring of the central venous pressure and urine output. The total infusion volume was calculated considering the volume deficit, perspiratory losses, and the need to maintain sufficient urine output (> 1 mL/kg/h) [11]. This made it possible to "cleanse" the renal tubules and prevent obstruction with the myoglobin cylinders. The urine output preservation and urine color change from brown to straw-yellow represent a clinical criterion of the applied therapy efficacy. Rapid hyperkalemia adjustment by using the glucose-insulin mixture was used to eliminate hyperkalemia resulting from massive cellular decay. Furthermore, vitamin therapy, gastroprotective therapy, anticoagulant therapy represented the important comprehensive treatment components [12–14].

CONCLUSION

Thus, this case report shows that acute α-PVP poisoning can lead to the early development of severe systemic rhabdomyolysis with the high risk of acute kidney injury, metabolic acidosis and toxic CNS damage. The key factors of the beneficial outcome are the early systemic rhabdomyolysis disgnosis and the rapid, effective intensive infusion therapy aimed at the early effective adjustment of metabolic disorders and maintenance of adequate renal blood flow for prevention of the irreversible kidney damage.

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