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MODERN SYNTHETIC OPIOIDS AND RABDOMYOLISIS: CLINICAL AND FORENSIC ASPECTS

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Abstract

Introduction. Every year, the list of chemical substances, including drugs, that can potentially cause rhabdomyolysis grows worldwide.
Aim. Analysis of etiological factors that caused the development of rhabdomyolysis of non-traumatic genesis in adult patients with acute poisoning.
Materials and methods. A total of 243 cases of acute poisoning among the adult population of Kyiv, complicated by non-traumatic rhabdomyolysis, were studied for the period 1999-2020.
Results. According to the toxicological center and the bureau of forensic medical experts of Kyiv, the spectrum of toxic substances that cause the development of non-traumatic rhabdomyolysis is constantly changing. During the period 1999-2010, rhabdomyolysis was associated with poisonings with carbon monoxide, ethyl alcohol and its surrogates, barbiturates, clonidine, TCA, neuroleptics, and heroin. After 2010, cases of rhabdomyolysis associated with neuroleptics, street drugs: psychostimulants, heroin, methadone, in particular the combination of methadone with ethyl alcohol, or with benzodiazepines, neuroleptics, tricyclic antidepressants, have become more frequent. Cases of severe poisonings with unidentified substances were increasingly observed (12.75%). In-hospital mortality rates among patients with rhabdomyolysis remained high (from 10.0 to 25.8%) and depend on the patient's comorbidities, among which diseases of the digestive system (39.1%), the nervous system (27.9%), hematopoietic organs (20.5%) and kidneys (16.9%) predominate. The clinical picture of poisoning complicated by rhabdomyolysis is characterized not only by acute kidney injury, but also by the rapid development of multiple organ failure, which, despite the timely use of renal replacement therapy, leads to a fatal outcome.
Conclusions. In recent years, there has been a growing trend of drug poisoning involving synthetic opioids (heroin, methadone in combination with ethanol and other surfactants), complicated by non-traumatic rhabdomyolysis. Key factors in the development of multiple organ failure are the combined consumption of several psychotropic and narcotic substances with alcohol and comorbid pathology.

Keywords: **poisoning in adults, drug toxicity, rhabdomyolysis**

INTRODUCTION

Rhabdomyolysis is a common occurrence in the practice of toxicologists and intensive care physicians. However, the progressive increase in the spectrum of substances that can potentially cause rhabdomyolysis prompts an in-depth study of the etiological factors of this phenomenon.

It is known that rhabdomyolysis occurs as a result of the rapid breakdown of skeletal muscle fibers and the leakage of potentially toxic cellular contents into the systemic circulation. Regardless of the cause of rhabdomyolysis, direct damage to the sarcolemma occurs due to an increase in intracellular calcium concentration, which leads to fiber necrosis [1].

Rhabdomyolysis can be triggered by fever or excessive physical exertion. These conditions are associated with two phenomena: an increase in temperature and high levels of circulating pro-inflammatory mediators, such as cytokines and chemokines [2, 3].

Toxic rhabdomyolysis is an acute and potentially fatal condition of skeletal muscle resulting from the toxic effects of xenobiotics that damage the muscle cell wall and cause the release of intracellular enzymes and myoglobin into the bloodstream. Myoglobin is also excreted in the urine (myoglobinuria). Myoglobin and intracellular components in high concentrations cause obstruction of renal structures, leading to acute tubular necrosis and renal failure [4-5].

The typical clinical picture, according to many studies [6, 7, 8], is muscle swelling, weakness and pain in the affected area, and the appearance of dark brown urine due to the excretion of myoglobin by the kidneys. The level of creatine phosphokinase (CPK) is a marker of rhabdomyolysis, but its levels can vary greatly. Other dangerous complications of rhabdomyolysis are described: electrolyte disorders (hyperkalemia), which cause cardiac arrhythmias, metabolic acidosis, hyperphosphatemia and hypocalcemia. According to various authors, the mortality rate in rhabdomyolysis can exceed 10%. However, the development of rhabdomyolysis depends on many factors, including: the etiological factor of the comatose state, the surface area of the affected muscles, age, gender, the nature of the comorbid pathology, etc.

AIM

Analysis of the etiological factors that caused the development of rhabdomyolysis of non-traumatic genesis in adult patients with acute poisoning.

MATERIALS AND METHODS

A total of 243 cases of acute poisoning among the adult population of Kyiv, complicated by non-traumatic rhabdomyolysis, were studied for the period 1999-2020. Among the patients aged 18 to 72 years, there were: men – 180 (74.1%), women – 63 (25.9%). All patients received a comprehensive examination and treatment in accordance with the requirements of the clinical protocols «Acute poisoning», approved by the Ministry of Health of Ukraine. The clinical diagnosis was established based on the results of chemical and analytical studies. The concentration of alcohol in the blood was determined (gas chromatography); the toxicological panel for drugs (enzyme-linked immunosorbent assay) included: opioids, ketamine, methadone, amphetamines, cocaine, benzodiazepines, barbiturates, buprenorphine, etc. Analytical studies on the content of drugs were carried out using liquid chromatography and gas chromatography-mass spectrometry.

A total of 46 people died, the corpses were sent for forensic medical examinations. All autopsies were performed within four days after the establishment of death. All corpses were stored at -4 ◦C. In selected cases, a standard sample collection protocol was used. Tissue pieces of lungs, kidneys, myocardium, brain, liver, and spleen fixed in a sufficient amount of formalin were examined. After formalin fixation, alcohol fixation, and paraffin embedding, the sections were stained with hematoxylin and eosin. The preparations were examined using an Olympus CX 41 microscope in transmitted light, at 40, 100, and 400 times magnification. Subsequently, a retrospective analysis of the results of forensic histological examinations (microphotographs) of the internal organs of the corpses was performed.

In all cases, documentation from the investigation authorities and the prosecutor's office was available, which provided for conducting examinations for forensic medical purposes; therefore, approval from the ethics committee or institutional review board was not required. All procedures performed in this study were in accordance with the ethical standards of the institution and the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Materials from the clinical archive of the Kyiv City Clinical Hospital of Emergency Medical Care and analytical data from the Kyiv City Bureau of Forensic Medical Expertise were used; sources of scientific information from the PubMed, Medline, and Elsevier libraries on the topic of the study were analyzed. Methods of systematic and comparative analysis were used.

RESULTS

On average, about 2,500 adult patients with chemical poisonings are admitted to the toxicology center of the Kyiv City Clinical Hospital of Emergency Medical Care each year. The vast majority of them are admitted to the hospital in a coma and are hospitalized in the intensive care unit and extracorporeal detoxification.

Among the most common causes of toxic coma are overdoses of alcohol, drugs, and medications. Rhabdomyolysis, as a complication of coma, is observed on average in 3.5 cases per 1000 poisonings (3.5‰).

If, over the 30-year observation period, the frequency of rhabdomyolysis has not changed significantly, it has remained a relatively rare complication, then the causes of its occurrence have undergone significant changes. Thus, during the period 1999-2010, rhabdomyolysis was mainly associated with poisonings with carbon monoxide, ethyl alcohol and its surrogates, barbiturates, clonidine, tricyclic antidepressants (TCA), neuroleptics, and heroin.

During the period from 2010 to 2015, significant changes occurred in the structure of drug poisonings, the

share of synthetic opioids increased, the leaders among which were heroin and illegally manufactured methadone. Thus, by 2010 their share was 28.7%, and in the period 2015-2019 it increased significantly (52.2%) and began to change qualitatively and quantitatively. Over the past 10 years alone, the number of positive tests (performed during the year) for methadone content has increased almost 150 times, for morphine and tramadol – 100 times; for heroin – 6 times; on the other hand, for natural opium («shirka», a raw, unrefined form of opium) – it decreased 7 times. In general, the number of cases of detection of natural opium products in the biological material of patients decreased from 57% in 2000 to 2% in 2020.

The spread of illegal methadone among drug users has changed the structure of the causes of hospital mortality from drug overdoses: in 2009, 19 deaths from methadone poisoning were registered (0.04 per 100 thousand population), and in 2019-599 cases (11.19 per 100 thousand population); the number of forensic medical studies of deaths associated with methadone has also significantly increased (by 9 times). It is important to note that in almost half of the poisoning cases, along with methadone, chemical and analytical studies of patients' blood and urine revealed ethanol, other narcotic substances (amphetamine derivatives, morphine, cocaine, etc.), as well as various medicinal and psychotropic substances (barbituric acid derivatives, benzodiazepine, phenothiazine, tricyclic antidepressants, etc.).

After 2010, cases of rhabdomyolysis associated with neuroleptics, street drugs: psychostimulants, heroin, methadone, in particular the combination of methadone with ethyl alcohol, or with benzodiazepines, neuroleptics, tricyclic antidepressants, have become more frequent. Cases of rhabdomyolysis are increasingly observed in severe poisonings with unidentified substances (12.75%).

Hospital mortality rates among patients with rhabdomyolysis (Table 1) remain high (from 10.0 to 25.8%). Thus, with an average hospital mortality rate for poisonings of chemical etiology, which is from 2.5 to 4.5%, mortality in poisonings complicated by rhabdomyolysis is from 10.0% to 25.8%. There are gender differences: in the group of poisonings with chemicals, men predominate (75.0%), in the group of poisonings with medicines – women (69.0%), in the group of drugs, psychoactive and unknown substances, men predominate (81.5%). The risk of rhabdomyolysis in men, among other things, may be due to the presence of a greater muscle mass than in women.

The development of rhabdomyolysis of non-traumatic genesis is influenced by the presence of concomitant pathology in the patient. Analysis of the medical data of patients revealed a wide range of concomitant pathology, both among men and women (Table 2). Among the diseases, diseases of the digestive system (39.1%), nervous system (27.9%), hematopoietic organs (20.5%) and kidneys (16.9%) predominate. The range of identified diseases determines a high risk of rapid development of multiple organ failure against the background of rhabdomyolysis, which may not be the only complication of a comatose state.

For example, among 58 patients with methadone poisoning complicated by prolonged soft tissue compression syndrome, the following pathological conditions were found: cerebral coma, brain edema – 54 cases (96.4%); respiratory failure – 33 cases (56.9%); liver failure – 11 cases (18.9%); renal failure, including anuria – 14 cases (24.1%); cardiopulmonary failure, including pulmonary edema – 9 cases (15.5%); presence of thrombophlebitis, purulent wounds, sepsis – 14 cases (24.1%).

According to the results of autopsies, various pathologies in the organs were found, covering not only the kidneys (Fig. 1) but also the liver (Fig. 2).

Table 1

Etiological factors of rhabdomyolysis in patients with poisoning (N=243), study period: 1990-2020

Substance	Total cases, abs. number, (%)	Men abs. number, (%)	Women abs. number, (%)	Treatment period, days	Lethality abs. number, (%)
Chemicals					
Carbon monoxide	24 (9,8)	18 (75)	6 (25)	11,4±3,5	5 (20,8)
Ethyl alcohol and surrogates	36 (14,8)	34 (94,4)	2 (5,6)	9,5±4,7	4 (11,1)
Medicines					
Barbiturates	12 (4,9)	2 (16,7)	10 (83,3)	15,7±7,7	2 (16,6)
Tricyclic antidepressants	16 (6,6)	4 (25)	12 (75)	12,8±4,4	3 (18,8)
Neuroleptics	10 (4,1)	4 (40)	6 (60)	10,3±3,2	1 (10,0)
Clonidine	4 (1,6)	3 (75)	1(25)	10,7±4,6	1 (25,0)
Narcotics and psychotropic substances					
Heroin	45 (18,5)	39 (86,7)	6 (13,3)	12,8±3,9	7 (15,6)
Methadone	58 (23,9)	51 (87,9)	7 (12,1)	11,4±2,6	14 (24,1)
Psychostimulants	7 (2,9)	4 (57,1)	3 (42,9)	13,6±5,1	1 (14,3)
Unidentified substances	31 (12,75)	21 (67,7)	10 (32,3)	10,5±4,3	8 (25,8)
TOTAL:	243	180 (74,1)	63 (25,9)		46 (18,9)

Table 2

Structure of comorbidities in patients with poisonings (N=243) complicated by rhabdomyolysis, study period: 1990-2020

Disease groups	Pathology frequency, %
Diseases of the digestive system	95 (39,1)
Liver disease	58 (23,8)
Diseases of the pancreas	29 (11,9)
Diseases of the nervous system	68 (27,9)
Mental and behavioral disorders	65 (26,7)
Viral hepatitis	62 (25,5)
Diseases of the blood and blood-forming organs (anemia)	50 (20,5)
Renal tubulointerstitial diseases	41 (16,9)
Some infectious and parasitic diseases	36 (14,8)
Respiratory diseases (pneumonia)	29 (11,9)
Diseases accompanied by high blood pressure	26 (10,7)
Diseases caused by human immunodeficiency virus (HIV)	21 (8,6)
Diseases of the endocrine system	15 (6,1)
Diabetes mellitus, type II	5 (2,1)

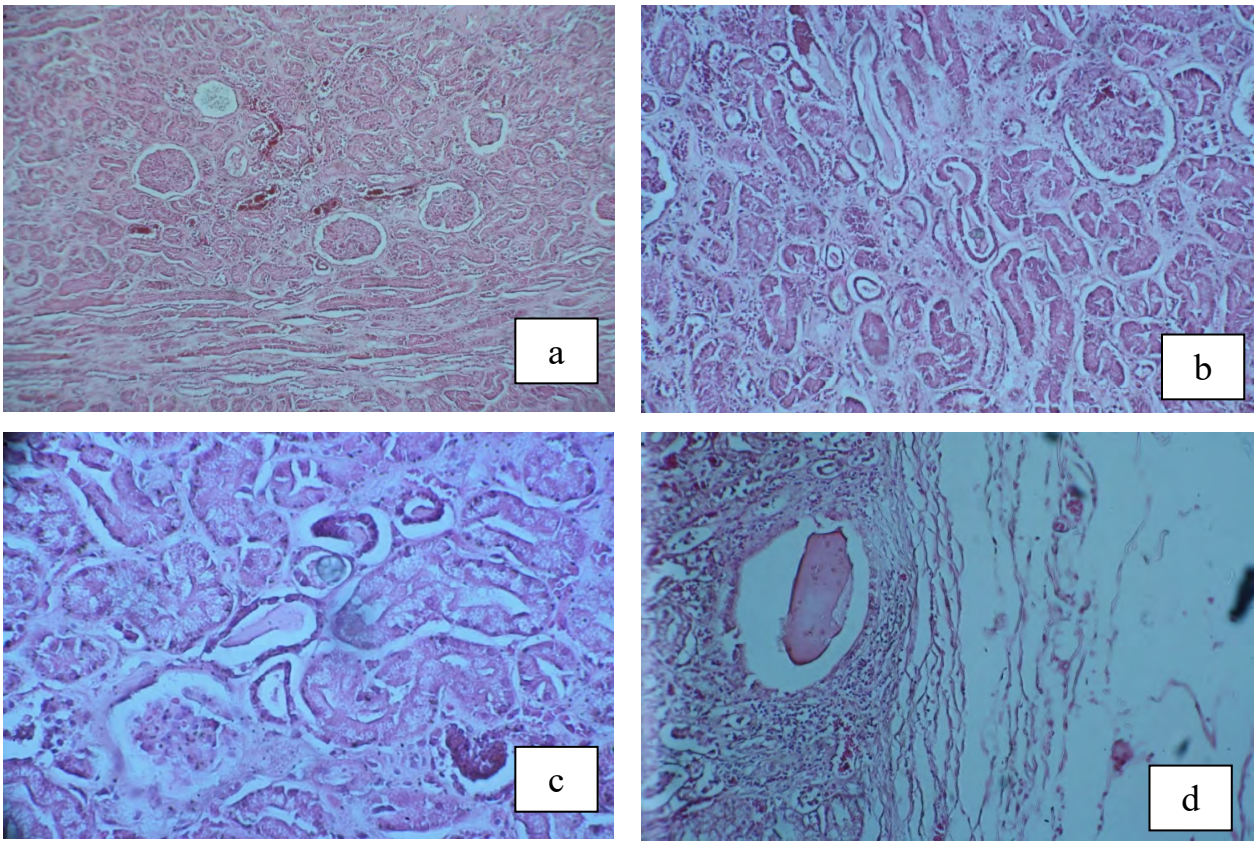


Figure 1. Micrographs of a kidney preparation (GxE, 100, 400) of a male who died from opioid drug poisoning complicated by non-traumatic rhabdomyolysis.

Histopathological examination of kidney preparations revealed hemodynamic disturbances, tubular, glomerular and interstitial changes. Tissue samples showed dilatation and engorgement of vessels, including glomerular capillaries, signs of sludge phenomenon (Fig. 1 – b, c, d). In the cortical section, obstruction of part of the distal tubules with proteinaceous material occupying part of the lumen; in the proximal tubules, degeneration of epithelial cells, tubules somewhat dilated, epitheliocytes flat with pyknotic nuclei; detachment of the epithelium from the basement membrane; cellular detritus; crystals. In single glomeruli, in capillaries and Bowman's space, poorly stained, probably proteinaceous substance, atrophy of glomerular bundles. Focal proliferation and mononuclear infiltration of the stromal tissue (Fig. 1 – a, b, c, d).

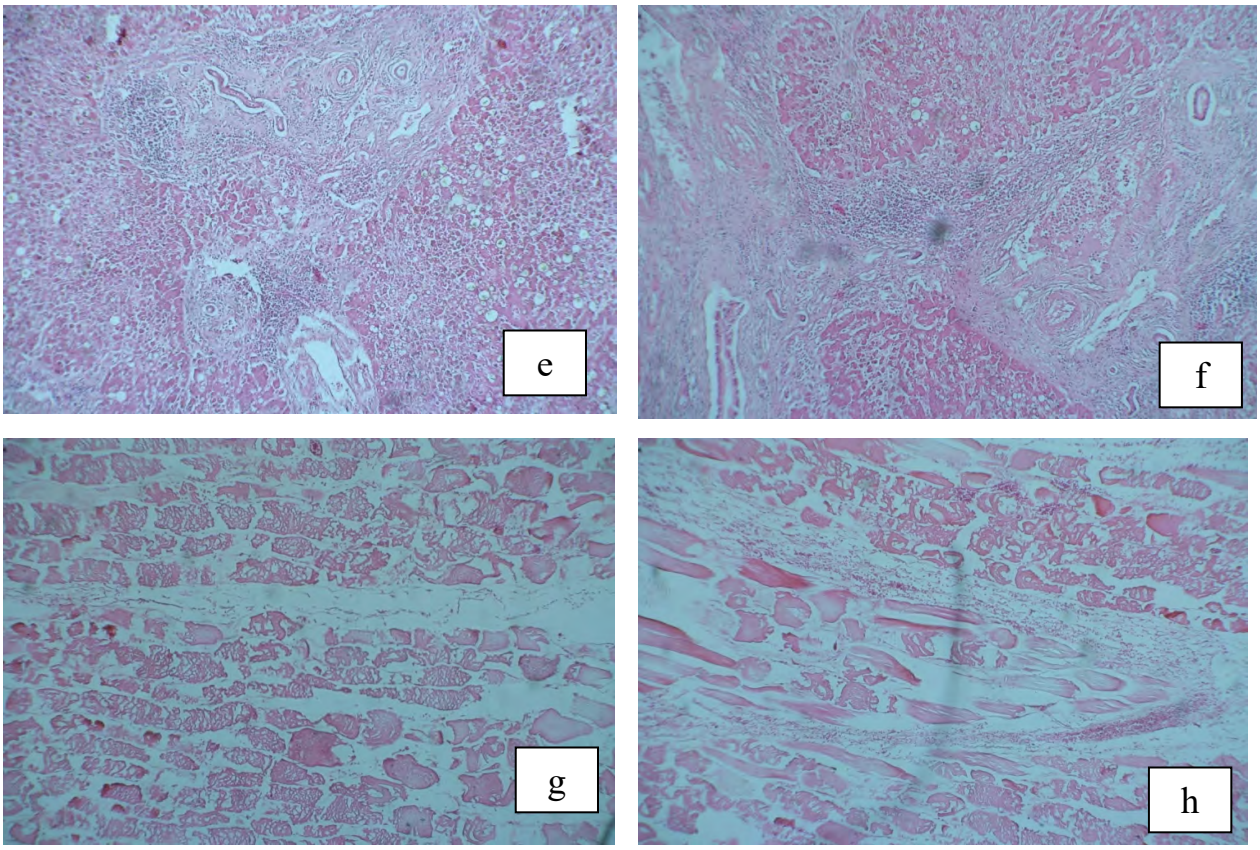


Figure 2. Micrographs of liver (e, f) and skeletal muscle (g, h) preparations of a male subject who died from opioid drug poisoning complicated by non-traumatic rhabdomyolysis (GxE, 100, 400).

According to the results of forensic histological examination, portal cirrhosis with a characteristic massive proliferation of connective tissue with abundant lymphoid cell infiltrate, along with admixtures of plasmatic and histiocyte elements was detected in liver samples. The predominant mass of hepatocytes was in a state of small and medium droplet fatty dystrophy and necrosis (Fig. 2 – e, f). Skeletal muscles with massive destructive-dystrophic changes (vacuolization, disintegration, necrosis). Vascular congestion, edema and stromal infiltration were noted (Fig. 2 – g, h).

DISCUSSION

Alcohol and drug use, leading to prolonged sleep in a fixed position, is the most common cause of toxic rhabdomyolysis. Our experience demonstrates that acute and/or chronic exposure to alcohol and illicit drugs is the most common trigger for rhabdomyolysis: most hospitalized patients with elevated CPK levels had a history of alcohol and substance abuse.

According to many authors, the leading drugs causing rhabdomyolysis are illegal methadone, heroin, cocaine, methamphetamine, ecstasy, and LSD; stimulants have been implicated in rhabdomyolysis with hyperthermia and muscle rigidity.

Given the prevalence of rhabdomyolysis, obtaining information about substance use is an important part of clinical diagnosis.

Because patients with rhabdomyolysis may be subdural and comatose due to toxic and metabolic effects, clinicians should anticipate and screen for rhabdomyolysis [2, 4].

In addition to alcohol and drugs, some prescription drugs can also cause rhabdomyolysis. For example, antipsychotics that cause neuroleptic malignant syndrome are a prominent cause of rhabdomyolysis in adults and children. Among psychiatric drugs, antidepressants are also frequently associated with rhabdomyolysis.

Statins used in hypothyroidism increase the risk of rhabdomyolysis, especially those metabolized by CYP3A4 (simvastatin, atorvastatin, and pravastatin). The risk of rhabdomyolysis is increased with concomitant use of other drugs metabolized by CYP450, including calcium channel blockers, amiodarone, macrolide antibiotics, and HIV protease inhibitors, among others [1, 10].

Rhabdomyolysis can occur as a result of electrolyte disturbances (hypokalemia, hypophosphatemia, hyponatremia, and hypernatremia) in the setting of endocrine diseases such as diabetes, thyroid dysfunction, primary hyperaldosteronism, diabetes insipidus, and

pituitary dysfunction, etc. Cases of rhabdomyolysis are also associated with electrolyte disturbances (mainly hypokalemia and hyponatremia) in the setting of laxative and diuretic abuse [3, 11].

Biological toxins and pesticides [12] are relatively rare causes of rhabdomyolysis; however, certain snake toxins can cause rhabdomyolysis through direct myotoxicity and/or muscle ischemia associated with coagulopathy. Toxins from wasps and spiders, as well as certain species of inedible mushrooms, can also cause rhabdomyolysis [13].

The diagnosis of rhabdomyolysis is based primarily on the presence of myoglobin in the urine or a significant increase in serum CPK levels. After muscle injury, plasma myoglobin levels rise rapidly and then fall rapidly due to renal excretion and recover within 24 hours. In contrast, serum CPK levels rise 2-12 hours after the onset of muscle injury, peak 3-5 days after injury, and decline over the next 6-10 days. Given that not all patients are hospitalized within 24 hours of muscle injury, measurement of CPK levels may be the most reliable biochemical marker of the severity of rhabdomyolysis [14].

The characteristic clinical features of rhabdomyolysis include myalgia, muscle weakness, and muscle edema that develop over hours to days.

Laboratory diagnosis of rhabdomyolysis indicates an increase in serum CPK levels, usually three to five times the upper limit of normal, from 100 to 400 IU/L (approximately 1000 IU/L). Although less than 10% of patients have classic symptoms, most have mild laboratory abnormalities.

Rhabdomyolysis often leads to acute renal failure. Intensive therapy should be aimed at dehydration and plasma alkalinization to reduce myoglobin precipitation in the renal tubules, and renal replacement therapy should be used in the presence of anuria.

CONCLUSIONS

The trend of recent years is an increase in cases of drug poisoning involving synthetic opioids (heroin, methadone in combination with ethanol and other surfactants), complicated by non-traumatic rhabdomyolysis. The key factors in the development of multiple organ failure are the combined consumption of several psychotropic and narcotic substances with alcohol and comorbid pathology.

Perspectives for further research. The specific toxicity of new synthetic opioids and psychotropic substances remains an area that requires additional research. In particular, a promising direction of scientific research is the study of the combined effect of narcotic substances with alcohol, drugs, as factors in the occurrence of rhabdomyolysis and other organotoxic effects.

COMPLIANCE WITH ETHICAL REQUIREMENTS

In this study, the authors adhered to the Ethical Principles of Medical Research Involving Humans as set forth in the Declaration of Helsinki of the World Medical Association (WMA, 1964) and current Ukrainian regulatory documents. The study protocol was approved by the Commission on Ethics of Medical and Biological Research of the State Enterprise «Scientific Center for Preventive Toxicology, Food and Chemical Safety named after Academician L. I. Medved of the Ministry of Health of Ukraine». Animal testing was not used for this study.

FUNDING AND CONFLICT OF INTEREST

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Резюме

СУЧАСНІ СИНТЕТИЧНІ ОПІОЇДИ ТА РАБДОМІОЛІЗ: КЛІНІЧНІ ТА СУДОВО-ЕКСПЕРТИЗНІ АСПЕКТИ

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Вступ. У світі щороку збільшується перелік хімічних речовин, в тому числі наркотиків, що потенційно можуть бути причиною рабдоміолізу.
Мета. Аналіз етіологічних чинників, що обумовили розвиток рабдоміолізу нетравматичного генезу у дорослих пацієнтів з гострими отруєннями.
Матеріали та методи. Досліджені 243 випадки гострих отруєнь серед дорослого населення м. Києва, ускладнених нетравматичним рабдоміолізом, за період 1999-2020 рр.
Результати. За даними токсикологічного центру і бюро судово-медичних експертиз м. Києва, спектр отруйних речовин, що обумовлює розвиток нетравматичного рабдоміолізу, постійно змінюється. У період 1999-2010 рр. рабдоміоліз був асоційований з отруєннями моноксидом вуглецю, етиловим алкоголем та його сурогатами, барбітуратами, клофеліном, ТЦА, нейролептиками, героїном. Після 2010 р. почастішали випадки рабдоміолізу, асоційованого з нейролептиками, вуличними наркотиками: психостимуляторами, героїном, метадоном, зокрема поєднанням метадону з етиловим алкоголем, або з бензодіазепінами, нейролептиками, трициклічними антидепресантами. Все частіше спостерігаються випадки тяжких отруєнь невстановленими речовинами (12,75%). Показники госпітальної летальності серед пацієнтів з рабдоміолізом залишаються високими (від 10,0 до 25,8%) і залежать від наявної у пацієнта супутньої патології, серед якої переважають хвороби органів травлення (39,1%), нервової системи (27,9%), кровотворних органів (20,5%) та нирок (16,9%). Клінічна картина отруєння, ускладненого рабдоміолізом, характеризується не лише гострим ураженням нирок, а й стрімким розвитком поліорганної недостатності, що, незважаючи на своєчасне застосування методів замісної ниркової терапії, призводить до летального результату.
Висновки. Тенденцією останніх років є збільшення випадків наркотичних отруєнь за участі синтетичних опіоїдів (героїну, метадону у поєднанні з етанолом та іншими ПАР), що ускладняються нетравматичним рабдоміолізом. Ключовими чинниками розвитку поліорганної недостатності є комбіноване споживанні декількох психотропних і наркотичних речовин з алкоголем та коморбідна патологія.

Ключові слова: **отруєння у дорослих, токсична дія наркотиків, рабдоміоліз**

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