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Case report

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Hemlock (Conium Maculatum) Poisoning

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BACKGROUND Spotted hemlock (Conium maculatum) is one of the most poisonous plants. Hemlock poisoning is extremely rare, but can lead to serious consequences, including death.

MATERIAL AND METHODS A clinical observation of a 50-year-old patient with a diagnosis of hemlock poisoning is presented.

CONCLUSION The clinical observation of acute hemlock poisoning presented by us does not exclude its direct cardiotoxic effect and requires further research in this direction, which will allow optimizing the treatment of this group of patients.

Keywords: acute poisoning, plant poison, hemlock poisoning, conium maculatum

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AKI – acute kidney injury
BP – blood pressure
CPK – creatine phosphokinase
ECG – electrocardiography
GCS – Glasgow Coma Scale
HR – heart rate
ICU – intensive care unit
IV – intravenously
PHS – prehospital stage

INTRODUCTION

Spotted hemlock (Conium maculatum) is one of the most poisonous plants. This is a well-known plant poison, often used back in Ancient Greece to execute criminals. Socrates was one of the dissidents killed by hemlock [1]. Currently, poisoning by this plant is extremely rare, but in cases of untimely provision of medical care, it can lead to serious consequences, including death [2–4]. At the same time, in folk medicine, tinctures, ointments, homemade decoctions, and other remedies prepared on the basis of spotted hemlock are widely used to treat various diseases.

Hemlock contains alkaloids, mainly coniine, which have a toxic effect when taken orally. The alkaloids of this plant are similar to nicotine in chemical structure and physiological action. They are selective agonists of nicotinic-type acetylcholine receptors [4]. It is known that hemlock poison causes damage to the nervous and respiratory systems of the body, as well as the kidneys. Clinical manifestations of hemlock poisoning are as follows: bronchospasm, bronchorrhea, increased blood pressure (BP), tachycardia, tremor, nausea, vomiting, ataxia, mydriasis, decreased body temperature, increased respiratory rate, development of respiratory failure, paralysis of muscles, breathing, seizures, depression of consciousness to coma, rhabdomyolysis and acute kidney injury (AKI) [5].

There are isolated publications in the literature that describe individual clinical observations, including various symptoms, depending primarily on the severity of poisoning.

This does not allow one to obtain a holistic picture of the toxic effect of hemlock on the body, which complicates the diagnosis and choice of treatment tactics for this poisoning.

The **aim** of this work is to demonstrate clinical case of severe hemlock poisoning.

Clinical observation

Patient M., 50 years old, was admitted to the N.V. Sklifosovsky Research Institute for Emergency Medicine with a diagnosis of "hemlock poisoning". It is known from the anamnesis, that 3–4 hours before admission to the hospital she drank 50 ml of alcoholic tincture of spotted hemlock for the purpose of self-

medication. After 2 hours, the patient noted a deterioration in her condition: rapid heartbeat, difficulty breathing, nausea, abdominal pain, repeated vomiting, and later repeated loose stools. At the prehospital stage (PHS), the level of consciousness was reduced to stupor, the respiratory rate was 22 breaths per minute, blood pressure was 109/77 mmHg, and the heart rate was 160 beats per minute. The stomach was washed through a tube, an enterosorbent was introduced, and infusion therapy and oxygen inhalation were started.

The patient was hospitalized in the intensive care unit (ICU) at 15:35. At the time of admission, stupor was observed (Glasgow Coma Scale (GCS) score of 14), and respiratory distress in the form of tachypnea - 28 respirations per minute, increased blood pressure - 146/86 mmHg, and heart rate – 146 beats per minute; with oxygen insufflation in a volume of 6 l/min, blood saturation with oxygen (SpO_2) was 78%.

Due to increased respiratory failure after preliminary premedication, a solution of propofol, 1% - 20 ml, atropine, 1% - 1 ml, rocuronium bromide, 5% - 5 ml was administered, orotracheal intubation was performed, after which the patient was connected to a mechanical ventilator to provide adequate oxygenation and prevent aspiration complications.

An electrocardiography (ECG) revealed the following data: atrial fibrillation, tachysystole, right bundle branch block, transient block of its left anterior and posterior branches, heart rate of 160 beats per minute. This was the basis for antiarrhythmic therapy with amiodarone at a dose of 5 mg/kg intravenous (IV) bolus. Indicators of laboratory tests (biochemical analysis, general clinical, acid-base status) were within the reference values.

7 minutes (15:42) after admission to the ICU, against the background of intensive therapy, the patient experienced a sharp deterioration in her condition. The skin became pale and cyanotic. The cardiac monitor revealed large-wave ventricular fibrillation, and there was no blood pressure.

Resuscitation measures were carried out:

- indirect cardiac massage;
- electric pulse therapy - defibrillation, 200 J 3 times;
- sodium bicarbonate, 4% - 200.0 ml IV in a stream, once;
- epinephrine, 0.1% - 1.0 ml, IV bolus, 7 injections.

At 16:12, a positive effect was noted. On the cardiac monitor – arrhythmia in the form of atrial fibrillation. Blood pressure - 82/43 mm Hg, heart rate – 179 beats per minute. Level of consciousness - coma (GCS score - 6).

To maintain effective blood circulation, norepinephrine was administered at a dose of 0.3 mcg/kg/min. Infusion therapy and administration of amiodarone at a dose of 50 mg/h were continued. A repeat ECG (20:20) revealed the following: tachysystolic form of atrial fibrillation, a prolonged QT interval, incomplete right bundle branch block. After 6 hours: sinus rhythm was restored, alternans of QRS complexes were observed.

Results of laboratory blood tests: leukocyte content - 17.5×10^9/l, aspartate aminotransferase (AST) - 465 U/l, alanine aminotransferase (ALT) - 262 U/l, creatinine - 110 μmol/l, urea - 4.14 mmol/ l, lactate dehydrogenase (LDH) - 1328 U/l. The other indicators were within the reference values.

After 13 hours from the moment of hospitalization, the administration of sedatives to the patient was stopped. 14 hours 30 minutes after hospitalization, the patient was in clear consciousness (GCS score – 15), hemodynamics were stable; the patient was extubated and breathing spontaneously. A repeat ECG showed sinus rhythm, heart rate – 85 beats per minute. Positive dynamics of laboratory parameters were noted. Noteworthy was a short-term increase in the level of creatine phosphokinase (CPK) to 2147 U/l on the 3rd day of hospital stay. Three days after hospitalization, the patient was transferred from the ICU to the acute poisoning department. The duration of hospitalization was 5 days.

DISCUSSION

Hemlock poisoning usually occurs when it is consumed orally. In most cases of poisoning described in the literature, they occur due to the ingestion of plant leaves mistaken for parsley. In our observation, the patient took hemlock tincture in a dose of 50 ml for the purpose of self-medication.

Early symptoms of hemlock poisoning, according to the literature, are as follows: nausea, vomiting, sweating, tachypnea, salivation and urination, muscle weakness, ataxia, paralysis of the legs and arms, tremor, blurred vision, tachycardia/bradycardia, increased BP, headache and mydriasis [4, 6]. In one observation, a 6-year-old girl who took hemlock orally had the following complaints: burning in the mouth, hypersalivation, tremor, ataxia [7]. According to the literature, increasing muscle paralysis, including respiratory muscles with shortness of breath/apnea, convulsions and depression of the level of consciousness to coma are noted later [4]. In the present case, the patient complained of nausea, vomiting, abdominal pain, diarrhea, as well as rapid heartbeat and difficulty breathing; fluctuations in blood pressure (146/86 mm Hg) and heart rate (146 beats per minute) were observed. In the literature, there are indications of the development of disturbances in the acid-base status and electrolyte composition of the blood due to repeated vomiting and diarrhea in case of hemlock poisoning [4]. However, in our case, these disorders were absent.

Foreign authors described the development of rhabdomyolysis during hemlock poisoning, leading to AKI [5]. It should be noted that patient M. had a short-term increase in the level of CPK in the blood to 2147 U/l in the absence of AKI. In the early period of intoxication in the ICU, she had tachyarrhythmia in combination with cardiac conduction disturbances, which required the administration of the antiarrhythmic drug; and then ventricular fibrillation and cardiac arrest developed. A similar situation was described by D. Brtalik in a 30-year-old patient, when after intravenous administration of hemlock poison, depression of consciousness to the point of coma, rhythm disturbance, and cardiac arrest were observed [8]. A.U. Ferah et al. demonstrated a case of cardiac arrest at the PHS in a 49-year-old woman with hemlock poisoning, that notwithstanding, resuscitation efforts were successful. However, the patient died in the hospital on the 9th day [9]. The authors conclude that patients with hemlock poisoning should be hospitalized in the ICU, and their connection to mechanical ventilation before the development of complete paralysis of the respiratory muscles can lead to a decrease in mortality [9]. Based on the above observation and literature data, it can be assumed that disturbances in heart rhythm and conduction with subsequent cardiac arrest in this pathology are associated with the direct toxic effect of hemlock poison on the heart.

Currently, there is no antidote for hemlock poisoning. In the presence of bronchorrhea, administration of atropine before its relief is indicated [10]. The presented clinical example shows that in case of oral hemlock poisoning, the stomach should be washed out as soon as possible, followed by enterosorption, and according to indications, infusion detoxification and symptomatic therapy should be carried out (oxygen inhalation, tracheal intubation, mechanical ventilation, vasopressor support, etc.).

CONCLUSION

Our clinical observation of acute hemlock poisoning does not exclude its direct cardiotoxic effect and requires further research in this direction, which will optimize the treatment of this patient population.

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