Case Report

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Features of the Clinical Course, Diagnosis and Intensive Care of Acute Barium Poisoning

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ABSTRACT Acute barium poisoning is a rare but extremely dangerous type of poisoning in the structure of chemical injuries. The article provides a brief overview of the pathogenesis, clinical course and principles of intensive therapy of acute intoxication with this metal, as well as clinical examples of severe poisoning with barium salts with a negative and positive outcome in victims who used counterfeit barium carbonate instead of barium sulfate.

Keywords: barium sulfate, barium chloride, barium carbonate, acute poisoning, hypokalemia, primary cardiotoxic effect, toxic hepatopathy, antidote therapy, intensive care

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ABB — acid-base balance
ALV — artificial lung ventilation
ARF — acute respiratory failure
ECG — electrocardiography
EMS — emergency medical services
GIT — gastrointestinal tract
HR — heart rate
i/v — intravenously
i/m — intramuscularly
SES — sanitary and epidemiological service

INTRODUCTION

Barium is an element of the 2nd group of the sixth period of the periodic table of chemical elements of D.I. Mendeleyev with atomic number 56 and molecular weight 137.34. Denoted by the symbol Ba (lat. Barium). Barium is a soft, malleable alkaline earth metal of silver-white color. It has high chemical activity [1, 2].

Mankind has known barium for more than 250 years, since it was discovered as an oxide in 1774 by chemists Carl Scheele and Johan Gan. The next well-known barium mineral was natural barium carbonate, discovered in 1782 by Withering and subsequently named witherite in his honor. Barium metal was first obtained by the Englishman Humphry Davy in 1808 by electrolysis of wet barium hydroxide with a mercury cathode and subsequent evaporation of mercury from a barium amalgam [1].

The use of metallic barium is very limited due to its high reactivity, but its compounds are used much more widely.

The most commonly used are: barium oxide (BaO) – mainly in the production of thermionic cathodes, which are an integral part of modern monitors, barium hydroxide ($Ba(OH)_2 \cdot 8H_2O$) is used to remove sulfate ions from vegetable and animal oils and industrial solutions, to obtain hydroxides of rubidium and cesium, as a component of lubricants, barium carbonate ($BaCO_3$) is introduced into the composition of enamels and glazes and, finally, barium sulfate ($BaSO_4$), which is actively used in the production of paints, high-quality paper and barite concrete [1]. It is well known that barium sulfate intensely absorbs x-rays and gamma rays. This property is widely used in medicine for the diagnosis of gastrointestinal diseases. To do this, the patient is offered to swallow a suspension of barium sulfate dissolved in water [2, 3].

All barium salts are highly toxic, with the exception of insoluble barium sulfate, which is not absorbed from the gastrointestinal tract (GIT) and is excreted unchanged. Barium salt poisoning occurs mainly as a result of erroneous ingestion of other, soluble salts instead of barium sulfate. The toxic dose of soluble barium compounds is 0.2–0.5 g, the lethal dose is 0.5–4 g [1, 3, 4].

All soluble salts are characterized by rapid absorption in the gastrointestinal tract. Excretion from the body occurs mainly with feces, traces of barium are found in the urine, and a significant part of it is deposited in the bones (65%). Its concentration in plasma is higher than in erythrocytes. Barium does not penetrate into the cytoplasm, but is absorbed by the cell membrane. It binds to plasma proteins, with the exception of globulins. In the blood serum, 4.6% of the introduced barium is found 1.5 hours after administration, 0.1% a day later . Two days later barium is not detected in the blood. Barium passes through the placental, and to a lesser extent through the blood-brain barrier and has a weak mutagenic effect [3, 4].

In metabolism, barium behaves like calcium and strontium. However, it is absorbed into the blood worse, and is excreted much faster. Tissues containing large amounts of calcium usually contain more barium, and tissues rich in magnesium contain little calcium and barium [3, 4].

The main mechanism of the toxic effect of barium salts is that Ba^{2+} ions, having the same radius as K^+ ions, compete with it in biochemical processes. As a result of this mutual compatibility, severe hypokalemia occurs. Barium is similar in physicochemical characteristics to calcium. Getting into the body in large quantities, it can replace calcium ions in bone tissue. It is as a result of the replacement of calcium in the bone tissue with strontium that “barium” rickets develops - increased bone fragility [4]. It should be noted that barium ions are calcium synergists in the activation of some enzymes. The formation of very strong and poorly soluble barium phosphate in bone tissue, nerve cells, and medulla causes the toxicity of barium ions [4].

Barium has an effect on smooth muscle tissue and the myocardium, in its effect reminiscent of the action of acetylcholine. Thus, barium is a synergist of acetylcholine. It causes hypokalemia; barium polysulfide derivatives inhibit cellular respiration like cyanides. In case of poisoning, vascular permeability increases, this leads to hemorrhages and edema. Damage to the nervous system is manifested by encephalopathy, paresis and paralysis [1, 4, 5].

It has been established that with prolonged intake of barium chloride at a dose of 0.5 mg/kg into the body, it causes disturbances in conditioned reflex activity, changes in the activity of aldolase, alkaline phosphatase, lactate dehydrogenase and aspartic transaminase in the blood serum, nucleic acids in the gonads and leukocytosis. In the liver, there are single small hemorrhages and hyperplasia of the elements of the reticuloendothelial system, a sharp decrease in the amount of glycogen. Protein dystrophy and karyolysis were noted in the kidneys. In the spleen, an overflow of its blood cells was revealed, an increase in the number of leukocytes occurs. In the brain, small areas of cytolysis are visualized in various layers of the cortex [4].

With barium poisoning through the mouth, salivation, burning in the mouth and esophagus, stomach pain, colic, nausea, vomiting, diarrhea, high blood pressure, hard irregular pulse, convulsions, profuse cold sweat, gait, vision and speech disorders, shortness of breath, dizziness, noise in ears. Consciousness is usually preserved. Possible paralysis of the lower extremities, then the trunk and upper extremities. In severe cases, death occurred in the first hours or days. There are three stages of barium poisoning [4, 5]:

Stage I - the initial effects of brain disorders, acute gastrointestinal manifestations, cardiovascular weakness, changes in the blood (leukocytosis, lymphopenia, shift of the leukocyte formula to the left, an increase in the erythrocyte sedimentation rate - ESR);

Stage II - severe disorders of brain activity, dysfunction of the cerebellar-vestibular apparatus, short-term loss of speech, weakening of vision and hearing, increased autonomic and emotional lability, general weakness, reactive depression, hysterical manifestations;

Stage III - persistent neuropsychic changes, hysterical reaction on the background of dystonia. Cardiotoxic effect - a violation of the rhythm and conductivity of the heart. Tachycardia, bigeminia, ventricular fibrillation, as well as depression of the *ST* segments and the *T* wave, similar to those changes that are detected with digitalis intoxication. Paralysis of the respiratory muscles [5].

Cases of severe poisoning with barium carbonate are described (toxic dose 0.2-0.5 g, lethal 2-4 g). This poisoning is characterized by acute gastroenteritis, loss of tendon reflexes, paresthesia, convulsions, paralysis of the limbs, a decrease in the content of potassium in the blood; phenomena resemble a picture of botulism. Acute barium poisoning after fluoroscopy of the stomach is manifested by pain in the epigastric region, weakness and dry mouth [3–5]. Cold sweat and nausea appear. Further, pallor of the skin, acrocyanosis, shortness of breath, tachycardia, an earth shade of skin color, cyanosis of the lips, fatigue, lethargy, slowness develop; the tongue is dry with a brown coating, the abdomen is swollen. Death from cardiac arrest occurs within 5-6 hours. Pathological and anatomical examination revealed focal hemorrhages, defects in the gastrointestinal mucosa, cerebral edema, hemorrhagic pulmonary edema, hemorrhagic erosions of the stomach, myocardial and renal stromal edema, multiple central liver necrosis, focal necronephrosis, and vascular plethora [6].

In a forensic chemical study, mineralization is carried out using nitric, sulfuric and other acids. Barium is detected in the sulfate precipitate by the green color of the flame and then by the shape of barium iodate and/or barium sulfate crystals. The reaction with sodium rhodizonate is carried out dropwise on filter paper. The pinkish-brown complex formed turns grey-red upon addition of hydrochloric acid. In the presence of significant amounts of soluble barium salts, a positive effect with chromates is observed in the object [4–6].

Principles of intensive care. Acute barium poisoning in the practice of a toxicologist and resuscitator is quite rare, all recommendations for the treatment of poisoning with this poison are based on a small number of cases, and therefore they are advisory in nature, taking into account the individual characteristics of each case [5, 7].

1. Gastric lavage through a probe with a 1% solution of sodium or magnesium sulfate to form insoluble barium sulfate, magnesium sulfate 30 g orally (100 ml of 30% solution), in an enema (1% solution). Conducting intestinal lavage upon admission according to the method of V.A. Matkevich [8]. Intravenous 10–20 ml of 25% magnesium sulfate solution. Forced diuresis. Early hemodialysis.

2. Specific treatment. Unithiol 5% solution intravenously (IV) drip 2–3 times a day or sodium thiosulfate, 30% solution in/in drip 2–3 times a day for 4–5 days [3, 5, 7].

3. Symptomatic therapy. With bradycardia - 0.1% solution of atropine, 1 ml IV. In case of rhythm disturbances - potassium chloride in large doses in the form of a polarizing mixture IV drip, if necessary, repeatedly, depending on the level of potassium in the blood. Cardiovascular agents. Vitamins B_1 and $_6$ intramuscularly (IM) - not at the same time. Antihistamines. Oxygen therapy. In order to prevent the growth of rhythm and conduction disorders of the heart - hydrocortisone 250 mg 3-4 times a day, an oil solution of vitamin E - 1 ml 2-3 times a day IM. D defibrillation indicated. Mechanical ventilation for paralysis of the respiratory muscles [3, 5, 7].

In Uzbekistan, clinical toxicologists for the first time in the history of domestic medicine encountered barium salt poisoning in February 2020. In a short period from February 14 to February 27, 2020, the Department of Toxicology of the Republican Scientific Center for Emergency Medical Aid (RSCEMA) received 4 patients with severe poisoning with barium salts, resulting from a counterfeit batch containing barium

carbonate entering the pharmacy network of the city of Tashkent. Of the total number of patients, 3 were discharged with recovery, one patient died.

Let us consider how the clinical picture of the disease developed in the case of negative and positive outcomes of poisoning.

clinical observation 1.

Patient T., 23 years old, was taken to the emergency department by gravity on February 18, 2020, at about 11:50 pm and hospitalized in the toxicological resuscitation department of the RSCEMA, case history No. 8721/498.

Complaints upon admission: severe weakness, numbness of the muscles of the upper and lower extremities, impaired movements in the legs, difficulty in swallowing, speech, drowsiness.

From the anamnesis: according to the patient's relatives, for an X-ray examination of the stomach and intestines, 3 hours before admission, he drank about 200 ml of barium sulfate, which he bought in a pharmacy in a plastic cup without marking and indicating the manufacturer's company. The patient had the above complaints within an hour after that, and therefore he was taken by gravity to the toxicological emergency department and, after gastric lavage, was hospitalized in the toxicological resuscitation department. The police and the sanitary and epidemiological service (SES) were informed as well.

Objectively upon admission: the patient's condition is severe, conscious, but lethargic, drowsy, there was a sharp weakness, numbness and impaired movement in the lower and upper extremities. Tendon reflexes are sharply reduced. The skin is pale, dry to the touch; there are no visible bodily injuries. Body temperature 36.6ºC. Breathing is free, shallow; the respiratory rate is 20 times per minute, vesicular breathing is auscultated in the lungs, single dry rales. Heart tones are muffled, rhythmic, pulse 74 per minute, blood pressure (BP) 110/60 mm Hg. The abdomen is soft and painless.

Tests: hypokalemia upon admission and in dynamics - 2.5 mmol / l. In dynamics 2 hours later - 1.8 mmol / l. Leukocytosis was noted - $18.0 \cdot 10^{9}$ U/l, without a shift of the leukoformula to the left. Other clinical and biochemical parameters were within the normal range. Acid-base state (CBS) - compensated metabolic acidosis: pH 7.35, pO_2 - 88, pCO_2 - 49, BE - -4.2.

ECG upon admission: signs of hypokalemia in the form of a trough-shaped depression of the *ST segment*.

According to the Bureau of Forensic Medical Examination, barium carbonate was detected in the blood and urine.

The patient in the toxicological resuscitation department underwent a set of measures: gastric lavage through a tube with the introduction of 10 grams of a suspension of magnesium sulfate orally, infusion-detoxification therapy, forced diuresis, alkalization of the blood, antidote therapy - sodium thiosulfate 30 ml of 30% solution intravenously drip , magnesium sulfate 25% - 20.0 ml IV drip, potassium preparations (120 ml 4% potassium chloride in the form of constant IV drip infusions of a polarizing mixture consisting of 10% glucose, 400.0 ml, 4% p- ra potassium chloride, 40.0 ml, and insulin, 8 units), hormone therapy, enterosorption (polysorb 10 grams). To prevent the primary cardiotoxic effect, intramuscular hydrocortisone, 250 mg and α – tocopherol, 2 ml of 10% oil solution were prescribed. The patient was promptly installed a double-lumen catheter in the right subclavian vein, but hemodialysis was not performed due to unstable hemodynamics.

However, despite the ongoing intensive therapy, the patient's condition worsened over time and about 4 h 30 min. in the morning it suddenly progressively worsened significantly, there was an increase in respiratory failure, weakness, a sharp violation of hemodynamics, against which the patient was transferred to a ventilator, vasopressors were initiated (dopamine 10 μg / kg body weight per minute, then 15-20 μg / kg body weight per minute). In the dynamics of the patient on February 19, 2020 at 7:25 a.m. ventricular fibrillation, which led to cardiac arrest, was noted. The resuscitation measures carried out were unsuccessful. At 8:00 a.m. biological death was called, the corpse was transferred to the forensic medical examination.

Post-mortem diagnosis: Acute poisoning with severe barium salts. Toxic cardiomyopathy. Toxic polyneuropathy. Toxic myopathy. Hypokalemia. Acute respiratory failure - ARF. Acute cardiovascular failure - ACVF.

Clinical observation 1.

Patient M., 70 years old, was delivered to the admission department of toxicology of the RSCEMA on February 27, 2020 at 3:30 via emergency medical services (EMS) by transfer from the admission department of the 4th city hospital with complaints on admission of nausea, vomiting, loose stools, sharp weakness, numbness of the muscles of the face, upper and lower extremities, lack of movement in the legs and arms, difficulty swallowing, impaired speech.

Anamnesis of the disease: according to relatives, the patient, 16 hours before admission (02/26/2020 at about 11:00) went to the 4th city hospital, where she drank about 300 ml of mixed with suspension of barium with water. After that, already at home, in an hour, nausea developed, as well as vomiting and loose stools. In 3 hours, facial numbness and severe muscle weakness developed. The EMS team was called, the doctor stated an increase in blood pressure and conducted antihypertensive therapy. The patient was left at home, in the period from 18 to 19 hours, against the background of continuing frequent vomiting and loose stools, the numbness of the extremities increased. According to relatives, at about 20:00 - 20:30 the patient stopped walking. In this regard, the ambulance crew was called again and an increase in blood pressure to 180/90 mm Hg was again stated. Conducted antihypertensive therapy and the patient left at home. Movement disorders in the hands, difficulty in swallowing and speech impairment appeared in the dynamics, in connection with which the resuscitators (MDS-service) and the patient were called at about 23:30. The patient was taken to the emergency department of the 4th city hospital, where she stayed until 03:10, while a nasogastric tube was installed, ECG and chest X-ray was performed. The patient was examined by a surgeon, therapist, neuropathologist, and resuscitator. According to their recommendations, an ambulance was called and with a referral diagnosis "Acute poisoning?" The patient was transferred to the emergency department of toxicology of the RSCEMA, where she was hospitalized in the department of toxicological resuscitation. The police and SES were informed as well.

The general condition of the patient upon admission is severe, consciousness is clear, answers questions to the point, but speech is impaired, slurred, barely legible, movements in the arms and legs are absent, pupils are equal in size, *OD=OS* , photoreaction is preserved. The skin and visible mucous membranes are pale, dry, and warm to the touch; there are no traces of violence on the body. Body temperature 36.6ºC. Tendon reflexes are sharply reduced, muscle strength in the arms and legs is 0 points. Pathological reflexes and meningeal symptoms are negative. Breathing is rapid, superficial, with the participation of auxiliary muscles; the frequency of respiratory movements is 28 per minute. Vesicular breathing, single dry rales were auscultated over the lungs. Heart tones are muffled, the pulse is rhythmic 80 bpm, weak filling and tension, blood pressure 150/90 mm Hg. The tongue is moist, lined with white coating, there is a nasogastric tube. The abdomen is swollen on palpation, soft, slightly painful in the epigastrium, intestinal motility is reduced, there was no stool. The kidneys are not palpable on both sides, the effleurage symptom is positive on both sides. The potassium level upon admission was 1.3 mmol/L. Other clinical and biochemical parameters were within the normal range, except for a slight increase in the level of liver enzymes - ALT - 87.6, AST - 74.5. The CBS indicators corresponded to moderate metabolic acidosis: pH 7.36, pO_2 - 94, pCO_2 - 51, BE - -4.8. Leukocytosis was noted - $15.0 \cdot 10^9$ U/l, a shift of the leukoformula to the left to metamyelocytes. On the ECG: signs of severe hypokalemia in the form of: a) sinus tachycardia without extrasystole (heart rate (HR) 138 bpm); b) trough-shaped depression in the *ST segment*; c) negative *T wave*. These ECG changes were consistent with the detected potassium level and descriptions in the literature.

According to the Bureau of Forensic Medical Examination, barium carbonate was detected in the blood and urine. The preliminary diagnosis was made: Acute poisoning with severe barium salts. Toxic gastroenteritis. Toxic cardiomyopathy. Toxic myopathy. Hypokalemia.

Accompanying diseases: Hypertension, 2nd stage.

Complications: ARF 1st degree.

Treatment. The patient was put on a ventilator, while there was no resistance to the introduction of an endotracheal tube, infusion-detoxification therapy and alkalization of the blood were carried out, potassium preparations were introduced (360 ml of 4% potassium chloride in the form of constant intravenous drip infusions of a polarizing mixture consisting of 10% glucose 400.0 ml, 4% potassium chloride solution 40.0 ml and insulin 8 U), antidote therapy - sodium thiosulfate 30% - 30-40.0 ml IV, magnesium sulfate 25% - 20.0 ml orally through a tube , Panangin orally through a tube, hormone therapy - prednisolone 90 mg IV 3 times a day, enterosorption - Polysorb through a probe 20 grams, lactulose 20.0 ml orally, antibiotic therapy - ceftriaxone at a dose of 1 gram IV. To prevent the primary cardiotoxic effect, hydrocortisone 250 mg IM and α – tocopherol 2 ml of a 10% oil solution IM were prescribed. In order to accelerate the removal of barium from the body, 8 hours after admission, a session of bicarbonate hemodialysis was performed for 4 hours.

After the therapy, the patient's condition in dynamics gradually improved, she regained consciousness, there was a regression of peripheral paralysis - already on the 3rd day, the patient's movements in the limbs were completely restored, blood pressure and heart rate stabilized, diuresis was adequate - 2000-2200-2400 ml / day . On March 29, 2020, the patient was extubated. On March 3, 2020, she was transferred to the Department of Toxicology. The dynamics of some laboratory parameters is presented in the table.

Table

Changes in biochemical parameters in patient M. in dynamics

Tests	Date						
	27.02 18:00	28.02 6:00	28.02 18:00	29.02 6:00	29.02 18:00	2.03 6:00	3.03 6:00
Potassium, mmol/l	0.37	0.87	1.3	1.6	2.52	3.7	4.6
Blood leukocytes, 10^9 U/l	15.2		12.4		11.6	7.8	6.7
ALT, U/l	66.5			114.2		45.6	39.5
AST, U/l	71.8			138.6		48.8	41.4

Notes: ALT, alanine aminotransferase; AST - aspartate aminotransferase

As can be seen from the table, the patient had a critical decrease in the level of potassium in the blood for 3 days, which couldn't even be managed by introducing ultra-high doses of a glucose-potassium mixture.

On the ECG dynamics, a complete recovery of parameters was observed - a decrease in heart rate to 80 per minute, the return of the *ST segment* to the isoline and the appearance of a positive T wave.

The Department of Toxicology continued infusion therapy (glucose-potassium mixture, Ringer's solution), hormone therapy, as well as the introduction of an antibiotic (ceftriaxone), heparin therapy and antisecretory therapy (omeprazole). The patient's condition in dynamics improved significantly, the patient was activated and prepared for discharge.

At the time of discharge, the patient's condition was significantly improved, there were no complaints. The skin is of normal color, body temperature is 36.8ºC. Vesicular breathing in the lungs, no wheezing. No respiratory or hemodynamic disturbances were observed. Heart sounds are clear, rhythmic. BP 120/80 mm Hg, pulse - 84 per minute. Acute poisoning resolved. The patient was discharged on March 9, 2020 in a satisfactory condition, without any neurological deficit.

When studying both clinical cases, a reasonable question arises: why did the young victim die, despite all the therapeutic measures we carried out, while the elderly patient survived and recovered? In our opinion, the leading role in the development of a fatal outcome for patient T. was played by the dose of the consumed poison, since he drank a full bottle of counterfeit barium with a volume of 200 ml, which led to a direct cardiotoxic effect of barium on the myocardium and the development of ventricular fibrillation, while the patient M. took a suspension of barium strongly diluted with water.

In the remaining 2 patients, clinical symptoms, laboratory parameters, the course and outcome of poisoning proceeded almost the same as in patient M., and therefore we do not see the need to describe their data in this article.

CONCLUSIONS

1. Acute barium poisoning is an extremely rare, but extremely dangerous pathology in the structure of chemical injuries. For successful treatment of poisoning with this agent, an integrated approach is required, including oral and parenteral administration of antidotes (magnesium sulfate and sodium thiosulfate), intestinal lavage, early hemodialysis, prevention of primary cardiotoxic effect and adequate correction of electrolyte imbalance.

2. The prevention of acute barium poisoning requires increased control over the supply of this radiopaque substance to prevent counterfeit consignments containing water-soluble barium salts from entering the country, as well as a ban on its free sale in the pharmacy network.

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