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# A CLINICAL CASE OF POISONING WITH POTASSIUM PERMANGANATE CRYSTALS IN A YOUNG CHILD

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**Abstract.** The analysis of the incidence of acute poisoning of chemical etiology (data from the annual reports of the FSBI NPTC of the FMBA of Russia) shows that 21.5 to 30.6 thousand children under the age of 18 seek specialized medical care in the Russian Federation. According to statistical data of the Department of Toxicology of the N.F. Filatov State Medical University of Moscow, an average of 104–116 children with a diagnosis of acute poisoning with cauterizing substances are treated annually in the hospital. Among them, the cause of poisoning in 5–7% of cases is ingestion of potassium permanganate crystals. Potassium permanganate ($KMnO_4$) belongs to the category of cauterizing poisons, causes severe chemical damage to tissues. It is a strong oxidizer, in the body it is converted to caustic alkali, atomic oxygen, manganese dioxide. In addition to the cauterizing effect, manganese also has a neurotoxic effect due to the fact that it freely penetrates the blood-brain barrier, has a high tropicity to the subcortical structures of the brain. The lethal dose of potassium permanganate for adults when ingested is 0.3–0.5 g/kg. No data on lethal doses in children found.

**Key words:** potassium permanganate; acute poisoning; cauterizing substances; children.

# КЛИНИЧЕСКИЙ СЛУЧАЙ ОТРАВЛЕНИЯ КРИСТАЛЛАМИ ПЕРМАНГАНАТА КАЛИЯ У РЕБЕНКА РАННЕГО ВОЗРАСТА

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**Резюме.** Анализ заболеваемости острыми отравлениями химической этиологии (данные годовых отчетов ФГБУ НПТЦ ФМБА России) показывает, что в течение года в РФ за специализированной медицинской помощью по причине острых отравлений обращаются от 21,5 до 30,6 тысяч детей в возрасте до 18 лет. По статистическим данным отделения токсикологии ГБУЗ ДГКБ им. Н.Ф. Филатова г. Москвы, ежегодно в стационаре проходят лечение в среднем 104–116 детей с диагнозом: острое отравление прижигающими веществами. Среди них причиной отравления в 5–7% случаев является прием внутрь кристаллов перманганата калия. Перманганат калия ($KMnO_4$) относится к категории прижигающих ядов, вызывает тяжелые химические повреждения тканей. Является сильным окислителем, в организме преобразуется до едкой щелочи, атомарного кислорода, марганцевой двуокиси. Помимо прижигающего действия марганец обладает и нейротоксическим эффектом в связи с тем, что свободно проникает через гематоэнцефалический барьер, обладает высокой тропностью к подкорковым структурам головного мозга. Летальная доза перманганата калия для взрослых при приеме внутрь — 0,3–0,5 г/кг. Данных о летальных дозах у детей не найдено.

**Ключевые слова:** перманганат калия; острое отравление; прижигающие вещества; дети.

## RELEVANCE

An analysis of the incidence of acute poisoning of chemical etiology shows that during the year in the Russian Federation, from 21.5 to 30.6 thousand children under the age of 18 seek specialised medical care due to acute poisoning. These statistics are confirmed by data from annual reports of the Federal State Budgetary Institution “Scientific and Practical Toxicological Center of the Federal Medical and Biological Agency of Russia”.

One of the reasons for seeking medical attention is poisoning with substances with a cauterizing type of action — organic and inorganic acids (acetic, oxalic, hydrochloric), alkalis (drain cleaners, grease removers based on caustic soda, ammonia) or oxidizers (perhydrol, potassium permanganate). According to statistical data of the Department of Toxicology of the Children’s City Clinical Hospital named after N.F. Filatov in Moscow, an average of 104-116 children with a diagnosis of acute poisoning with cauterizing substances are treated annually in the hospital. Among them, the cause of poisoning in 5–7% of cases is ingestion of potassium permanganate crystals [1, 2]. Similar data are also presented by foreign authors [3, 4, 5].

Thus, according to the American Association of Poison Control Centers, only in 2008, there were more than 1,6 million cases of poisoning in children, with alkali burns of the esophagus recorded in 18–46% of cases after consuming various household chemicals [6].

Potassium permanganate ($KMnO_4$) is classified as a cauterizing poison and causes severe chemical injury to tissues. It is a strong oxidizer and is converted in the body to caustic alkali, atomic oxygen, and manganese dioxide. In addition to its cauterizing effect, manganese also has a neurotoxic effect due to the fact that it freely penetrates the blood-brain barrier and high tropicity to the subcortical structures of the brain. The lethal dose of potassium permanganate for adults when ingested is 0.3–0.5 g/kg. No data on lethal doses in children have been found. Manganese poisoning is possible when it enters the body through the respiratory tract (in the form of dust) and gastrointestinal tract, or as an impurity when using drugs [7].

When cauterizing substances are taken orally, a burn disease (damage to the gastrointestinal tract) of chemical etiology develops as a result of the local destructive effect of substances on tissues, which is accompanied by severe exotoxic shock. The trigger for the development of exotoxic shock is usually a pain syndrome and state of relative or absolute hypovolemia. The second key point in pathogenesis of the disease is the formation of respiratory disorders (acute respiratory distress syndrome, pneumonia). The causes may be upper airway burn injury (laryngeal edema and stenosis), aspiration of gastric contents or the cauterizing substance itself, exotoxic shock (impaired blood rheology and microcirculation in the pulmonary circulation) [1, 8, 9].

According to a number of authors, the severity of damage to the gastrointestinal tract and upper respiratory tract when exposed to cauterizing substances largely depends on several factors. These include the nature of chemical agent, its concentration and duration of contact with mucous membranes. It is believed that the development of chemical burns is based on tissue hypoxia [1, 10, 11]. Complications of chemical burns of the upper gastrointestinal tract include the development of cicatricial stenosis of the esophagus and stomach, early and late bleeding, and esophageal perforation [12].

## CLINICAL CASE

A child, 2 years 11 months old, was treated at the State Budgetary Healthcare Institution Children’s City Clinical Hospital named after N.F. Filatov in Moscow with the diagnosis of severe poisoning with a cauterizing substance (potassium permanganate). This disease was accompanied by

the development of a number of complications: 2nd degree of exotoxic shock, type 1 and type 2 respiratory failure, 2nd–3rd degree of chemical burn of the pharynx, epiglottis, upper third segment of the esophagus, stress ulcer of the stomach, gastrointestinal bleeding, severe posthemorrhagic anemia.

The boy was urgently taken to the hospital. It is known from anamnesis that 1 hour before admission at home, the child ate a large amount of potassium permanganate crystals. At the prehospital stage, self-induced vomiting, refusal to eat or drink, and anxiety were noted. The ambulance crew administered 1,0 ml of fentanyl solution to the child and performed gastric lavage through a tube.

The boy's condition at the time of admission to the department was severe. Upon examination, the mucous membranes of the oral cavity, pharynx and tongue were covered with a dark brown coating of potassium permanganate crystals. Crystal deposits were also noted on lips and skin of the face around the mouth, neck and anterior chest surface (Fig. 1).

The voice is practically absent, expressed salivation is observed due to a disorder of the act of swallowing. The child is conscious, productive contact is difficult. When irritated during the examination, the patient shows pronounced psychomotor agitation. The child's position is passive, the facial expression is pained. The skin is pale. Breathing is spontaneous through the natural airways with the participation of accessory muscles. Harsh vesicular breathing with prolonged expiration is heard on auscultation, conducted to all sections, conductive wheezing. Tachypnea up to 36 per minute is noted. Oxygen saturation is 96 %.

Examination of the cardiovascular system revealed tachycardia up to 160 beats per minute, a decrease in mean arterial pressure to 60 mm Hg. A prolonged capillary refill is noted, i.e. slow filling of skin capillaries after compression. The skin of limbs is marbled, cool to the touch. The abdomen is accessible for deep palpation, there are no clinical manifestations of gastrointestinal bleeding.

According to laboratory tests, metabolic acidosis was present: base excess (BE) — 5.7 mmol/l (norm 2.5 to — 2.5), pH 7.31 (norm 7.35–7.45). The presence of tissue hypoperfusion was indicated by an elevated lactate level of up to 2.9 mmol/l (norm 0.5–1.6). Also, at the time of admission, the child had relative stress-induced hyperglycemia of up to 8.9 mmol/l (normal 3.9–5.8) [13]. When studying the parameters of coagulation hemostasis, a tendency towards hypercoagulation was noted, which is usually observed in patients with poisoning by cauterizing substances in the first 48 hours of the disease [14].

Thus, the severity of the child's condition upon admission was due to manifestations of exotoxic shock and respiratory failure against the background of a burn disease of chemical etiology.

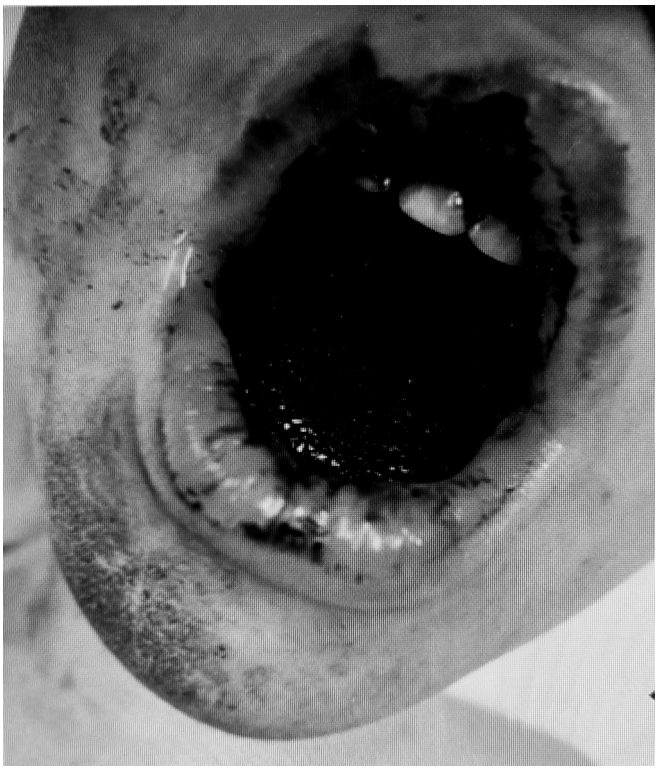

**Fig. 1. The appearance of the child upon admission**
**Рис. 1. Внешний вид ребенка при поступлении**

At the time of admission, the child had leukocytosis up to 15.8 × 109/l (4.0–9.0 × 109 cells/l), with a shift in the leukocyte formula of the blood due to an increase in the level of the relative number of granulocytes to 67 % (the norm is 35–45 %). The level of hemoglobin and red blood cells remained normal.

In the department, after provisional analgesia and sedation therapy (tramadol at the rate of 2 mg/kg, sibazon 0.5 mcg/kg of body weight), the child's accessible mucous membranes, tongue, lips and skin were treated with an ascorbic acid 5 % solution.

In acute poisoning with cauterizing poisons, exotoxic shock is hypovolemic in nature with disturbances in the patient's metabolic status from the point of view of pathophysiology. Therefore, infusion of balanced salt solutions was started immediately in order to maintain adequate water balance and prevent capillary blood flow disorders. The child was also prescribed the standard treatment, including antibacterial and hormone, analgesic therapy, antacids and proton pump inhibitors [1, 9, 14, 15].

Despite the treatment, 3 hours after admission, the child showed negative dynamics in the form of increasing respiratory failure. Tachypnea up to 50 beats per minute, pronounced participation of accessory muscles in the act of breathing, cyanosis of the nasolabial triangle, desaturation up

to 80% were observed. In this regard, a decision was made to perform emergency intubation of the patient and transfer to mechanical ventilation. During direct laryngoscopy, the boy had potassium permanganate crystals in the laryngopharynx. This required additional washing of the crystals with a 5 % solution of ascorbic acid. Crystals of potassium permanganate were also obtained during sanitation of the tracheobronchial tree.

During the first day of the disease, the child had hyperthermia, high lactate blood levels from 2.5 to 3.3 mmol/l remained, leukocytosis increased to $18.7 \times 10^9$/l, the hemoglobin level and color index remained normal. Hyperglycemia was kept within 9.2 mmol/l. The boy also had lymphopenia up to 10 % (normal 26–60 %). According to X-ray data, pulmonary plethora is determined and interstitial tissue edema is pronounced.

During esophagogastroduodenoscopy, the base of the epiglottis, tonsils, posterior pharyngeal wall, entrance of the esophagus and upper third of the esophagus are visualised, completely covered with a black coating (Fig. 2).

From the upper to the middle third of the esophagus, deposits in the form of spots and stripes are determined, further there is no deposit of crystals. The arytenoid cartilages are glassy edematous, the epiglottis is swollen,

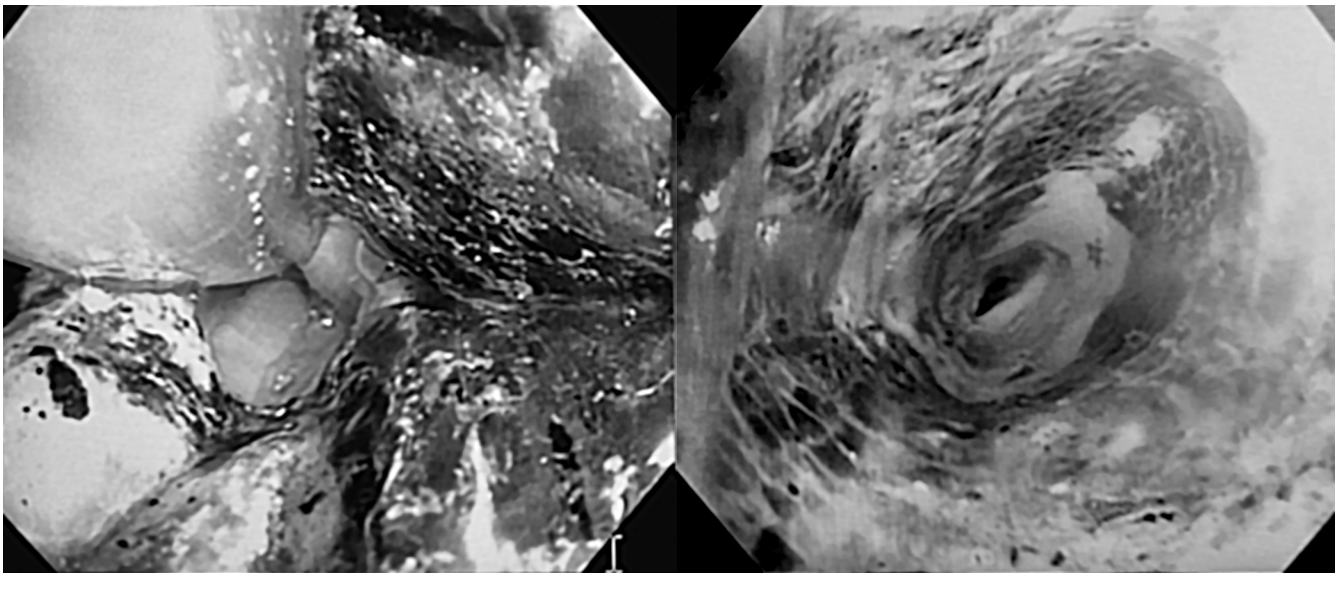

**Fig. 2. Esophagogastroduodenoscopy before mucosal treatment with ascorbic acid solution**
**Рис. 2. Эзофагогастродуоденоскопия до обработки слизистых оболочек раствором аскорбиновой кислоты**

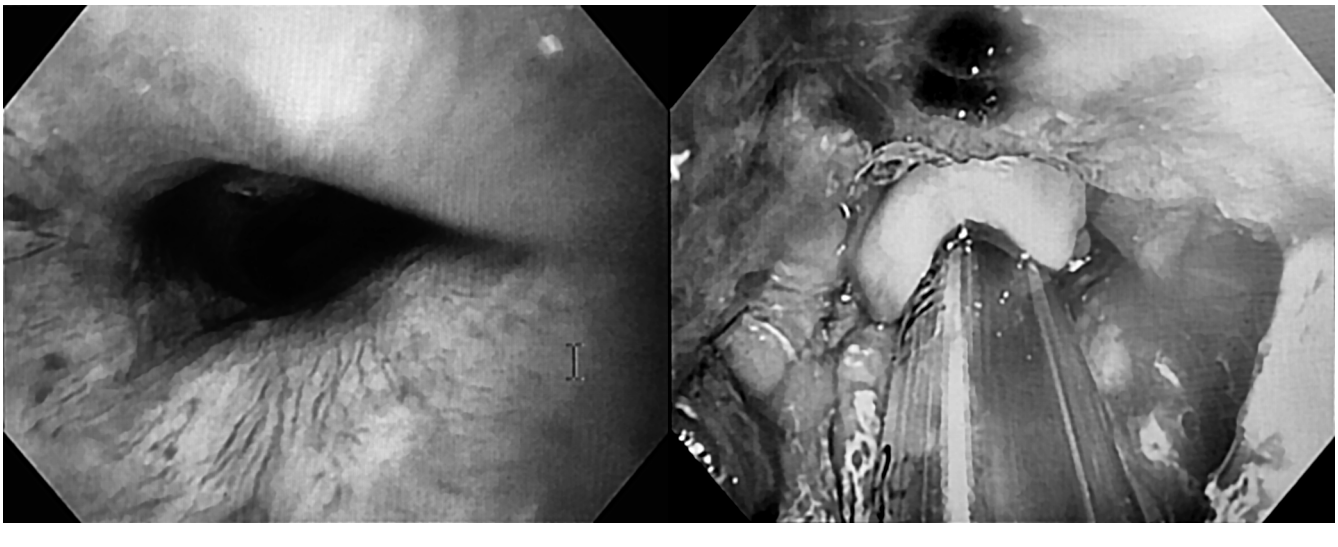

**Fig. 3. Esophagogastroduodenoscopy after mucosal treatment with ascorbic acid solution**
**Рис. 3. Эзофагогастродуоденоскопия после обработки слизистых оболочек раствором аскорбиновой кислоты**

the mucous membrane except for the base is unchanged. Manually under the control of laryngopharyngoscopy, the mucous membrane was cleaned of crystals with an ascorbic acid 5% solution. During the control examination, the mucous membrane of the tongue, pharynx, esophagus is without deposits of crystals, with multiple superficial erosions (Fig. 3). There is no bleeding.

After 48 hours, the patient's condition showed sharp negative dynamics in the form of gastrointestinal bleeding (gastric contents mixed with coffee grounds, black stool, hemodynamic destabilisation in the form of arterial hypotension with the need for vasopressor support). Progressive reduction of hemoglobin level to 50 g/l (normal 115–125 g/l) was noted. According to the coagulogram, INR increased to 1.68, and the tendency toward hypercoagulability persisted on other indicators. Hemostatic therapy, transfusion of red blood cells and fresh frozen plasma were initiated.

An emergency repeat esophagogastroduodenoscopy was performed. Delicate fibrin deposits on the back wall of the pharynx were found during the procedure. Circular shallow erosions covered with fibrin upper third of the esophagus were also observed. Along the lesser curvature of the stomach, an erosion covered with a blood clot was noted. A fixed blood clot, covering an area of 1.5 × 1.0 cm, was determined on the wall in the middle third of the duodenum.

Thus, the available clinical, laboratory and instrumental data indicate the development of complications of the disease in the child. Stress ulcer of the stomach and 2nd–3rd degree of chemical burn of the pharynx, epiglottis, upper third segment of the esophagus were detected. This subsequently creates high risks for the development of cicatricial stenosis of the esophagus and, accordingly, long-term surgical treatment [16–20].

Subsequently, the child continued complex drug therapy aimed at treating the chemical burn, which included a course of antibacterial therapy for up to 10 days, hormone therapy (dexamethasone 0.5 mg/kg of body weight), and gastroprotective therapy. For the purpose of gastroprotection, we used antacids (aluminum phosphate gel 20 %, 5 grams 3 times a day 40–50 minutes after meals and 1 time before bedtime), which have acid-neutralising and enveloping effect. The child was also prescribed proton pump inhibitors (omeprazole infusion 10 mg 2 times a day as intravenous infusion). These drugs inhibit the $H^+/K^+$-ATPase enzyme (proton pump) in the parietal cells of the stomach and thereby block the final stage of hydrochloric acid synthesis.

Against the background of the treatment, the child showed positive dynamics in the form of complete regeneration of the mucous membranes of the oral cavity and pharynx, epiglottis, an upper third segment of the esophagus, normalisation of laboratory parameters (Table 1).

On the 14th day of the disease, the boy was discharged under outpatient observation of a gastroenterologist and pediatrician.

## CONCLUSION

The presented clinical observation demonstrates the multifaceted nature of the pathogenesis of the disease in acute poisoning with potassium permanganate crystals. The response to chemical trauma is caused not only by the direct destructive action of the poison, but also by the development of dysfunction of cardiovascular and respiratory systems against. It is necessary to note the tension in the work of stress-realizing body systems, which is indirectly pointed by the indicators of glycemia, lymphopenia in our patient and the development of a stress ulcer. So, it becomes obvious that in order to provide

*Таблица 1*

**Мониторинг лабораторных показателей у ребенка с отравление перманганатом калия**

*Table 1*

**Monitoring of laboratory parameters in a child with potassium permanganate poisoning**

| Лабораторный показатель/ Laboratory indicator | Нормальные значения / Normal values | Поступление / Entrance | 1-е сутки / 1 day | 2-е сутки / 2 day | 3-и сутки / 3 day | 7-е сутки / 7 day | 14-е сутки / 14 day |
|---|---|---|---|---|---|---|---|
| С-реактивный белок в сыворотке крови / C-reactive proteinin blood serum | 0–5 г/л | 6 | 18 | 48 | 36 | 12 | 4 |
| Глюкоза в сыворотке крови / Glucose in blood serum | 3,4–6,1 ммоль/л | 8,9 | 9,1 | 7,6 | 6,5 | 5,6 | 5,4 |
| Гемоглобин / Hemoglobin | 110–140 г/л | 118 | 98 | 50 | 95 | 112 | 116 |
| Эритроциты / Red bloodcells | 3,5–4,5 × $10^{12}$кл/л | 4,1 | 3,4 | 2,9 | 3,2 | 3,9 | 4,1 |
| Лейкоциты / White blood cells | 4,0–9,0 × $10^{9}$кл/л | 15,8 | 18,7 | 16,8 | 13,1 | 12,0 | 9,5 |
| Нейтрофилы / Neutrophils | До 70% | 69 | 83 | 81 | 76 | 71 | 63 |
| Лимфоциты / Lymphocytes | 26–60% | 31 | 20 | 10 | 11 | 21 | 34 |

highly effective care for children with poisoning by cauterizing substances, hospitalisation in a multidisciplinary hospital which has experience in treating this pathology is necessary, with the possibility of emergency endoscopy. It should also be noted that thorough cleansing of the affected skin and accessible mucous membranes with weak acid solutions (ascorbic acid) is required when providing specialised medical care at the prehospital stage in case of poisoning with potassium permanganate crystals. This significantly reduces the destructive effect of the crystals on tissues and thereby lowers the risk of developing complications of the disease.

## ADDITIONAL INFORMATION

**Author contribution.** Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

**Competing interests.** The authors declare that they have no competing interests.

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**Consent for publication.** Written consent was obtained from the patient for publication the legal representative for the publication of medical dat.

## ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

**Вклад авторов.** Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

**Конфликт интересов.** Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

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