UDC 616-099
DOI: 10.56871/RBR.2025.79.48.011

CLINICAL CASE OF HOUSEHOLD CHLORINE POISONING

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For citation: Ananenko VA, Popov AV, Fursova EN, Trofimov MA, Vinogradov AA. Clinical case of household chlorine poisoning. Russian Biomedical Research. 2025;10(2):104–113. DOI: https://doi.org/10.56871/RBR.2025.79.48.011

Received: 28.03.2025 **Revised: 15.05.2025** **Accepted: 24.06.2025**

Abstract. Despite the fact that for most people chlorine, from a historical point of view, is primarily associated with chemical weapons, chlorine and its compounds are widely used in various spheres of human life, from rubber production and metalworking to cleaning products. The last are accessible to every person, which increases the risk of poisoning and makes it relevant to study every such case and also the pathological processes in the human body resulting from poisoning. The following clinical case is presented to highlight this issue: N., a 22-year-old man, was admitted to the Intensive care Unit (ICU) of the Military Field Therapy Clinic of Military Medical Academy named after S.M. Kirov in a state of moderate severity with impaired respiratory functions due to household chlorine poisoning. The patient underwent a comprehensive clinical examination, which included general blood panel, biochemical blood tests, and toxico-chemical analyses, registration of circulatory and respiratory system parameters, X-rays and computed tomography of the chest organs, consultations with specialists, and monitoring of identified pathological changes in dynamic during therapy. The presented clinical case not only demonstra tes the importance of careful anamnesis collection and its role in the differential diagnosis of diseases in order to make a true diagnosis in order to correctly sort and timely select evacuation tactics for optimal treatment and reduce the risk of both non-fatal and fatal complications. It also forces us to pay attention to the fact that poisoning with hazardous chemicals (AHS) is found everywhere, both at large enterprises and facilities, and in everyday life, and can be not only widespread, but also include isolated cases, often associated with violations of safety regulations when working with dangerous substances which are used in a wide variety of industries and situations, and that's requires doctors to be very vigilant when receiving information about a patient and his disease.

Keywords: chlorine, chlorine poisoning, pulmonary edema, acute inhalation poisoning, diagnosis, clinical case

DOI: 10.56871/RBR.2025.79.48.011

КЛИНИЧЕСКИЙ СЛУЧАЙ БЫТОВОГО ОТРАВЛЕНИЯ ХЛОРОМ

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Для цитирования: Ананенко В.А., Попов А.В., Фурсова Е.Н., Трофимов М.А., Виноградов А.А. Клинический случай бытового отравления хлором. Российские биомедицинские исследования. 2025;10(2):104–113. DOI: https://doi.org/10.56871/RBR.2025.79.48.011

Поступила: 28.03.2025 **Одобрена: 15.05.2025** **Принята к печати: 24.06.2025**

Резюме. Несмотря на то что у большинства людей хлор, с исторической точки зрения, в первую очередь ассоциируется с химическим оружием, хлор и его соединения массово используются в самых разных сферах человеческой жизнедеятельности, от производства резины и обработки металлов до чистящих средств. Последние находятся в доступности для каждого человека, что повышает риск появления случаев отравлений и обусловливает актуальность изучения каждого такого случая, а также образующихся в результате отравления патологических процессов в человеческом организме. Для освещения данной проблемы представлен следующий клинический случай: мужчина Н., 22 лет, поступил в отделение реанимации и интенсивной терапии (ОРИТ) клиники военно-полевой терапии (ВПТ) Военно-медицинской академии им. С.М. Кирова в состоянии средней степени тяжести с нарушением дыхательных функций вследствие бытового отравления хлором. Пациенту было проведено комплексное клиническое обследование, включавшее общеклинический, биохимический и токсико-химический анализы, регистрацию показателей системы кровообращения и дыхания, выполнение рентгенографии и компьютерной томографии органов грудной клетки, консультации специалистов и контроль выявленных патологических изменений в динамике на фоне проводимой терапии. Данный клинический случай не только демонстрирует важность тщательного сбора анамнеза и его роли в дифференциальной диагностике заболеваний для постановки истинного диагноза с целью корректной сортировки и своевременного выбора тактики эвакуации для назначения оптимального лечения и уменьшения риска прогноза как нелетальных, так и летальных осложнений. Он также заставляет обратить внимание на то, что отравления аварийно-опасными химическими веществами (АОХВ) встречаются повсеместно, как на крупных предприятиях и объектах, так и в быту, и могут нести не только массовый характер, но и включать единичные случаи поражения, зачастую связанные с нарушением соблюдения правил техники безопасности при работе с опасными химикатами в самых разных отраслях и ситуациях, что требует от врачей высокой настороженности при получении информации о пациенте и его заболевании.

Ключевые слова: хлор, отравление хлором, отек легких, острое ингаляционное отравление, диагностика, клинический случай

INTRODUCTION

Chlorine-containing substances have long been used by humans in the production of plastics, paper, rubber, metallurgy and other industrial sectors. Due to their high hazard potential, the use of chlorine compounds necessitates compliance with safety protocols and poisoning prevention measures [1]. By its chemical nature, chlorine is a yellowish-green gas with a characteristic suffocating odor, approximately 2.5 times heavier than air. Chlorine is highly reactive, readily soluble in water, and forms hydrochloric acid and hypochlorous acid. It is stored and transported as a liquefied gas under high pressure. At a concentration of 0.01 g/m³ (10 mg/m³), it irritates the respiratory tract, while exposure to levels exceeding 0.1 g/m³ (100 mg/m³) can cause severe damage, including toxic pulmonary edema [2].

Moreover, chlorine and its compounds have been utilized as chemical warfare agents. Chlorine was the first chemical weapon of mass destruction, deployed in 1915. German troops conducted a chemical attack against French forces on the front between Bixschoote and Langemarck. The mortality rate reached approximately 20% of personnel, with a case fatality rate of about 8% among hospitalized victims [3]. Subsequently, other more toxic substances with similar mechanisms of action were employed, including phosgene, diphosgene, and others. Although many believe chlorine has now lost its significance as a chemical weapon in modern contexts, international sources contain current evidence of chlorine being considered as a weapon [4], with particular emphasis on terrorist threats at industrial facilities where it remains widely used [5]. During a briefing by Lieutenant General Igor Kirillov, Commander of the Radiation, Chemical, and Biological Defense Troops of the Russian Armed Forces, allegations were presented regarding violations of international chemical and biological weapons prohibitions by Ukraine and the United States. Specific concerns addressed the August 11, 2024 deployment of 155-mm DM-105 cluster munitions by Ukrainian armed groups in Sudzha, Kursk Region, resulting in over 20 civilian and military casualties. Analysis of collected samples conducted at the Organization for the Prohibition of Chemical Weapons (OPCW)-accredited laboratory of the 27th Scientific Center of the Ministry of Defence of the Russian Federation. Results confirmed that personnel injuries were caused by respiratory exposure to an aerosol containing chlorine and asphyxiant chemical agents, where the metal-chloride mixture served solely as camouflage [2].

Thus, the relevance of studying the clinical aspects of chlorine and its compound exposure stems not only from its persistent potential as a weapon, but also from the continuous risk of human exposure during industrial accidents, disasters, and even domestic incidents [6–8].

In Russia, over 400,000 tons of chlorine are produced annually. When safety measures are inadequate, workplace air concentrations can reach irritant levels (10 mg/m³), with large leaks achieving lethal doses (100–200 mg/m³). A concentration of 2,500 mg/m³ causes fatality within five minutes. Although chlorine is classified as a non-persistent hazardous chemical agent, its leakage forms a cloud that can persist for 10–30 minutes. This duration is sufficient to induce acute chemical injury symptoms in victims [2].

The mechanism of pneumotoxic agents involves damage to the air-blood barrier cells, causing blood plasma to infiltrate alveoli and impair oxygen exchange. The pathogenesis of toxic pulmonary edema induced by these substances centers on endothelial cell injury, which increases barrier permeability and disrupts the metabolism of biologically active substances in the lungs [9].

An experimental study was conducted at the Department of Military Toxicology and Medical Protection of the Military Medical Academy named after S.M. Kirov (VMedA — the abbreviation from its Russian name, a leading military medical institution). Microscopic examination revealed that within 0.1 hours post-chlorine exposure, rat lungs exhibited increased volume with smoothed interlobar borders and visible gray-green coagulative necrosis areas on the surface. The lung edges appeared distended and rounded. These macroscopic changes persisted in animal lungs for 6 hours after chlorine exposure. Further histopathological analysis of the chlorine-exposed group demonstrated alveoli filled with homogeneous exudate containing neutrophils, erythrocytes, and fibrin strands, while interalveolar septa were thinned due to compression by edematous fluid [10–12].

It should also be noted that, unlike true pneumotoxic agents, chlorine's ability to cause chemical burns in the upper respiratory tract plays a significant role in the pathogenesis of the clinical presentation in an exposed individual [13].

The progression of chlorine-induced injury follows several distinct stages: reflex (initial), latent period, development of toxic pulmonary edema, edema resolution, and long-term sequelae [13]. Clinical manifestations may vary not only according to poisoning severity but also depending on time elapsed since chlorine exposure and the phase of intoxication, making thorough history-taking crucial for diagnosis. This principle is illustrated by the presented clinical case.

CASE REPORT

Patient N., a 21-year-old male (born in 2003), was admitted to the Pulmonology Department of Hospital X between September 4 and 6 for management of severe

bilateral community-acquired multilobar pneumonia complicated by grade 1 respiratory failure (RF; SMART-COP score: 2).

The medical history, as reported by the patient, began on September 2 with dyspnea and dry cough during morning exercise. By evening, his temperature had risen to approximately 37.0–37.5°C, yet the patient chose not to seek medical attention. The following day (September 3), weakness and dyspnea progressively worsened, intensifying by evening. Consequently, on the morning of September 4, he presented to the medical clinic with complaints of inspiratory difficulty, dizziness, fever (38.8 °C), tachycardia, and nausea, prompting his referral to Hospital X.

On admission, the patient reported dry cough, moderate general weakness, and throat irritation. Physical examination revealed stable condition of moderate severity. Room air oxygen saturation (SpO_2) was 91–92%. Lung auscultation demonstrated vesicular breath sounds, diminished in the lower lung fields, with left basal crackles. Percussion yielded clear pulmonary resonance. Respiratory rate was 17–19 breaths per minute. With humidified O_2 insufflation via nasal cannula at 3 L/min, SpO_2 improved to 97–98%. No active sputum production was observed. The patient remained hemodynamically stable with blood pressure 118/70 mm Hg and pulse 65–70 bpm demonstrating normal volume. Cardiac examination showed normal heart borders and clear rhythmic tones. Sinus rhythm was noted on electrocardiogram (ECG). The tongue appeared moist with a white coating.

Laboratory tests revealed abnormal findings in complete blood count (CBC), blood chemistry (BC), and acid-base balance (ABB), as presented in Table 1.

Rapid tests for viral infections, influenza, and novel coronavirus infection (COVID-19) returned negative results.

The following examination data were obtained: SpO_2 was 93% on September 4 and 91% on September 5. Chest computed tomography (CT) performed on September 4 revealed ground-glass opacities with consolidation areas in the upper and predominantly lower lobes of both lungs, along with interlobular septal thickening. The radiological findings were consistent with bilateral alveolitis.

The clinical presentation included fever, cough, and weakness. Auscultation revealed vesicular breath sounds, diminished in the lower lung fields, with left basal crackles. The diagnosis of severe bilateral community-acquired multilobar pneumonia was established at Hospital X based on these findings combined with laboratory (Table 1) and imaging results (chest CT, SpO_2). The patient developed grade 1 respiratory failure (RF; SMART-COP score: 2). The following treatment regimen was initiated: antibacterial therapy (amoxicillin/clavulanate 1.2 g every 8 hours plus levofloxacin 500 mg twice daily), mucolytics (Fluimucil 300 mg twice daily with nebulized ambroxol 2 mL three times daily), bronchodilators (nebulized Berodual 2.0 mL once daily), anticoagulation (enoxaparin 0.4 mL once daily subcutaneously), and antiviral therapy (umifenovir 200 mg four times daily). All management was supported by close monitoring of vital signs and laboratory parameters.

Table 1

Laboratory data from hospital X

Таблица 1

Лабораторные данные, полученные из госпиталя X

Показатели / Parameters	04.09	05.09	Референтные значения / Reference values
Гемоглобин / Hemoglobin	169	169	130–160 г/л / g/l
Эритроциты / Erythrocytes	5,79	5,53	4,5–5,5×10^9/л / 4,5–5,5×10^9/l
Гематокрит / Hematocrit	56	52,7	36–42%
Лейкоциты / Leukocytes	20,2	26,11	4,5–8,5×10^9/л / 4,5–8,5×10^9/l
Скорость оседания эритроцитов / Erythrocyte sedimentation rate (ESR)	15	12	0–15 мм/ч / mm/h
Фибриноген / Fibrinogen	5,5	6	2,3–5 г/л / g/l
С-реактивный белок / C-reactive protein	97,9	92,9	Менее 10 г/л / Less 10 g/l
pH	7,471	7,456	7,350–7,450
pCO_2	39,9	47,5	35–45 мм рт.ст. / mmHg
pO_2	94,4	86,3	83–108 мм рт.ст. / mmHg

Given the anticipated prolonged treatment duration, need for additional diagnostic workup, and the patient's condition, he was transferred via air medical transport to the Military Field Therapy Clinic of the VMedA, accompanied by a critical care transport team.

On September 6, the patient was admitted to the Military Field Therapy Clinic of the VMedA presenting with discomfort and pain in the upper third of the chest (retrosternal area) exacerbated by deep inspiration, intermittent nonproductive cough with scant mucous sputum, exertional dyspnea, diffuse throbbing headache during febrile episodes (with maximum recorded temperature of 38.8 °C), and throat irritation.

Physical examination revealed blood pressure of 120/75 mm Hg bilaterally. The pulse was regular at 80 beats per minute, with satisfactory volume and no tension. The apical impulse was non-palpable. Cardiac auscultation demonstrated clear, rhythmic heart sounds without pathological murmurs. The chest was symmetrically shaped and non-tender on palpation, with accessory muscle use during respiration. The respiratory rate was 20–22 breaths per minute at rest, with SpO_2 89–91% on room air, improving to 98–99% with oxygen insufflation at 5 L/min. Comparative percussion revealed dullness over the left lung base. Auscultation identified harsh breath sounds transmitted throughout all lung fields, diminished in both lower zones, with fine crackles audible. The patient's condition was classified as moderate severity due to endogenous intoxication syndrome and acute respiratory failure.

During detailed history-taking, it was established that on September 1, the patient had inhaled small amounts of chlorine vapors for over 30 minutes while disinfecting an unventilated bathroom with a self-prepared solution containing active chlorine compounds. This exposure caused eye irritation and lacrimation, and throat irritation, which resolved after copious water irrigation.

The diagnosis was established based on the patient's complaints, medical history, physical examination findings, and instrumental test results. The complaints included discomfort and pain in the upper third of the chest (retrosternal location) exacerbated by deep breathing, intermittent nonproductive cough with scant mucous sputum, and exertional dyspnea. The medical history revealed 30-minute inhalation of chlorine vapors in an unventilated space on September 1. Physical examination demonstrated a respiratory rate of 20–22 breaths per minute, SpO_2 of 89–91% on room air, harsh breath sounds with diminished intensity in both lower lung fields, and fine crackles. Instrumental evaluation (chest CT from September 4) showed ground-glass opacities with consolidation areas (predominantly in the lower lobes) and interlobular septal thickening. The final diagnosis was: acute moderate accidental household chlorine vapor poisoning (September 1) complicated by bilateral toxic pneumonitis and grade I respiratory failure (both September 4).

Due to significant respiratory dysfunction and clinical instability, the patient was transferred to the ICU for further diagnostic evaluation and treatment to achieve clinical stabilization.

The patient was treated in the ICU from September 6 to 11. A combined multi-component treatment regimen was initiated. The core pathogenetic therapy included oxygen support via non-invasive ventilation with PEEP , delivered through an antifoaming agent. Hormonal membrane-stabilizing therapy with dexamethasone was started at 24 mg followed by a daily reduction of 4 mg. Diuretic therapy consisted of furosemide (80 mg on September 6–7, reduced to 40 mg on September 8–9). Bronchodilator treatment included budesonide (500 mg) and aminophylline (2.4%, 10 mL on September 6–7), along with a nebulized combination of ipratropium bromide, fenoterol, and ambroxol (2.0 mL twice daily). Symptomatic therapy included acetylcysteine (600 mg daily). Prophylactic measures encompassed several areas. These included anti-inflammatory therapy with chloropyramine (2.0 mL intramuscularly for two days), anticoagulation with daily enoxaparin sodium (0.4 mL), and myocardial metabolic support with glucose-insulin-potassium (GIK) solution (400 mL). Secondary infection prevention was achieved with a combination of levofloxacin (500 mg twice daily) and amoxicillin-clavulanate (1 g twice daily).

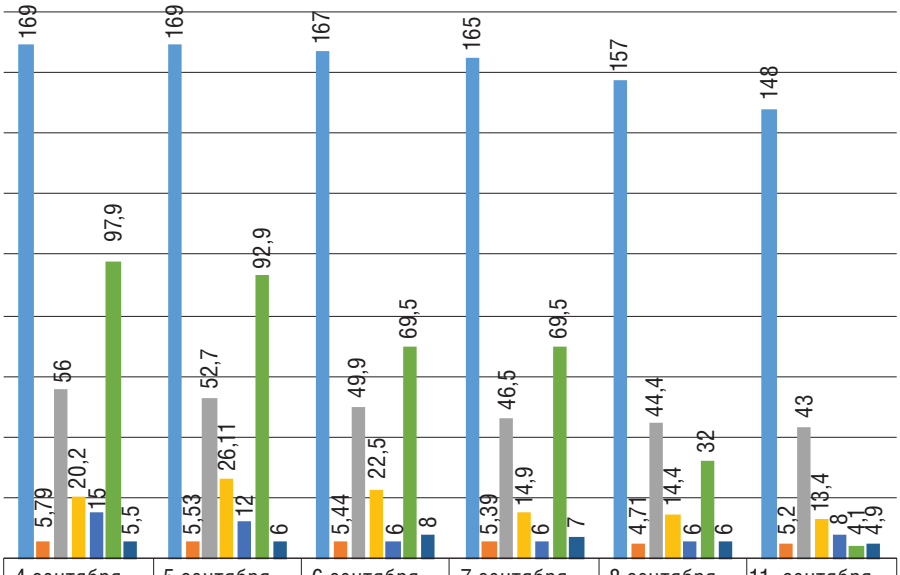
To assess the clinical status and treatment efficacy, repeat chest CT, body plethysmography, spirometry, as well as CBC, BC, ABB, and dynamic oxygen saturation monitoring were performed.

During the treatment course, daily improvements were observed in both CBC and biochemical parameters (Fig. 1), along with enhanced oxygenation indices and ABB (Fig. 2).

Elevated hemoglobin with hematocrit and erythrocytosis indicates hemoconcentration due to fluid redistribution, leading to increased blood viscosity and impaired circulatory function, thereby promoting circulatory hypoxia. Without therapeutic intervention, this condition would progress to profound hypoxemia with cyanosis and exponential clinical deterioration, as evidenced by practical studies conducted at the VMedA.

The persistent leukocytosis without left shift and elevated ESR are attributable to the systemic effects of dexamethasone administration.

Figure 3 demonstrates positive trends in oxygen saturation and ABB, manifested through progressive increases in both oxygen saturation levels and partial pressure of oxygen



	4 сентября (4 sep)	5 сентября (5 sep)	6 сентября (6 sep)	7 сентября (7 sep)	8 сентября (8 sep)	11 сентября (11 sep)
Гемоглобин, г/л / HGB, gr/l	169	169	167	165	157	148
Эритроциты, x10^9 / RBC, x10^9	5,79	5,53	5,44	5,39	4,71	5,2
Гематокрит, % / HCT, %	56	52,7	49,9	46,5	44,4	43
Лейкоциты, x10^9/л / WBC, x10^9/l	20,2	26,11	22,5	14,9	14,4	13,4
СОЭ, мм/ч / ESR, mm/h	15	12	6	6	6	8
СРБ, г/л / CRP, gr/l	97,9	92,9	69,5	69,5	32	4,1
Фибриноген, г/л / Fibrinogen, gr/l	5,5	6	8	7	6	4,9

Fig. 1. Dynamic of blood panel and biochemical blood tests

Рис. 1. Динамика общего и биохимического анализа крови

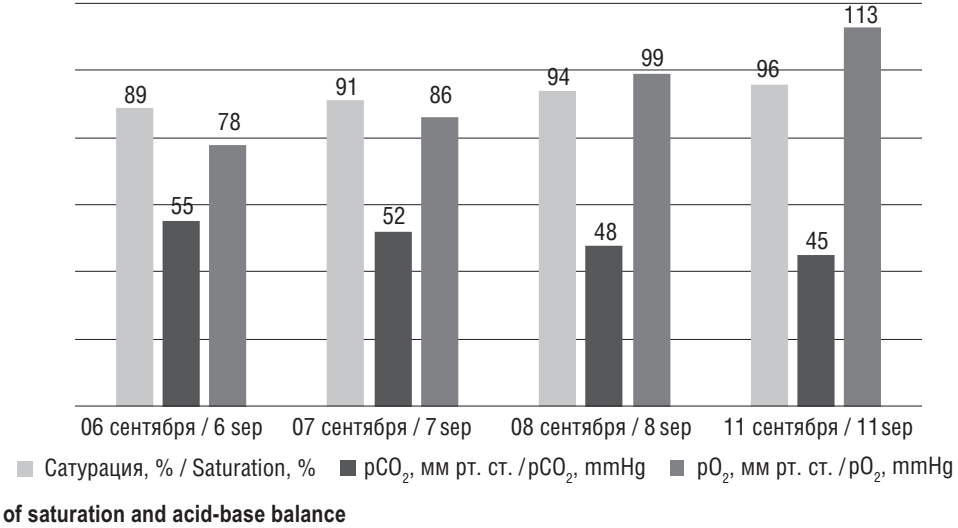
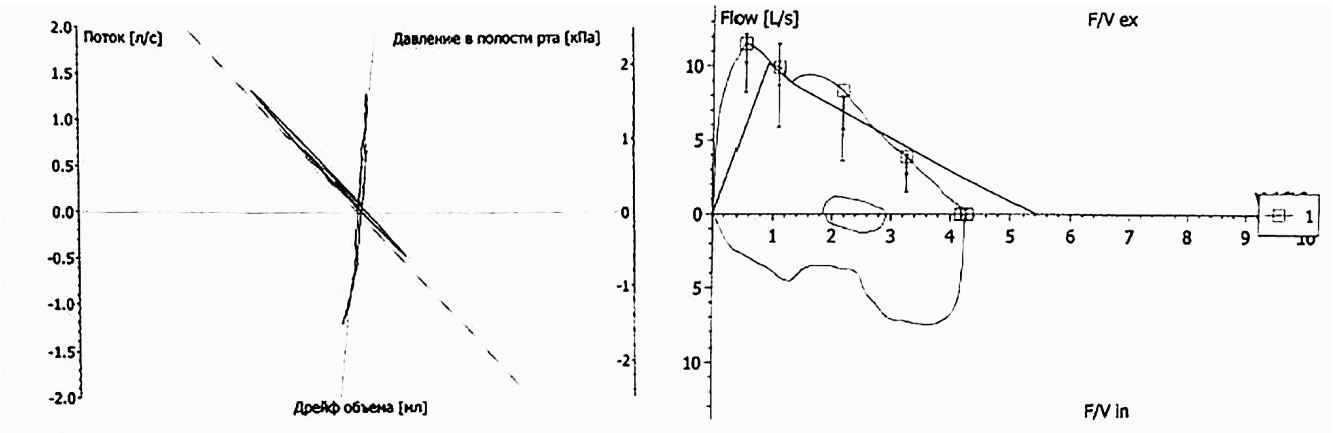


Fig. 2. Dynamic of saturation and acid-base balance

Рис. 2. Динамика сатурации и кислотно-щелочного состояния

(PaO_2), alongside gradual decreases in partial pressure of carbon dioxide ($PaCO_2$).

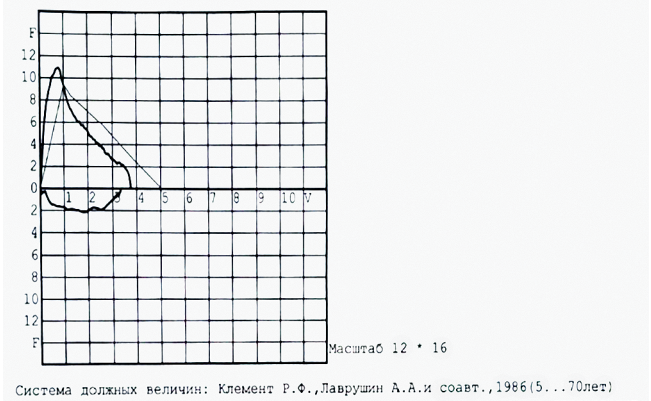
Given the need for comprehensive multifactorial assessment of all lung volume components, including airway resistance, body plethysmography was selected as the primary functional diagnostic method. This approach enables detection of functional impairments potentially overlooked by routine spirometry, which is critical during initial patient



		Д	П1	% (П1/Д)
SR eff	[kPa*s]	1.18	0.31	25.98
R eff	[kPa*s/L]	0.30	0.10	32.30
ITGV	[L]	3.37	2.62	77.71
RV	[L]	1.69	1.20	70.66
TLC	[L]	7.38	5.53	74.90
VC IN	[L]	5.69	4.33	76.16
RV % TLC	[%]	23.71	21.61	91.16

Fig. 3. Body plethysmography from 06.09

Рис. 3. Бодиплетизмография от 06.09



Система должных величин: Клемент Р.Ф.,Лаврушин А.А.и соавт.,1986(5...70лет)

ПАРАМЕТР		Должн.	Факт.	(%)	Град.	Оценка
ЖЕЛвд	(л)	5,20	3,60	69,22	4,9	Легкое сниж.
ФЖЕЛ	(л)	5,06	3,75	74,20	4,0	Очень легкое сниж.
ОФВ1	(л)	4,38	3,73	85,20	2,3	Условная норма
ТИФФНО	(%)	85,2	99,4	116,66	0,6	Выше нормы
ПОС	(л/с)	9,43	10,94	115,98	1,0	Выше нормы
МОС25	(л/с)	8,51	9,32	109,46	1,2	Норма
МОС50	(л/с)	5,96	5,47	91,83	1,7	Норма
МОС75	(л/с)	2,88	3,13	108,86	1,3	Норма
СОС	(л/с)	5,13	5,10	99,38	1,5	Норма
ОФВпос	(л)	----	0,77	----	----	
Тпос	(с)	----	0,13	----	----	
Твыд	(с)	----	1,09	----	----	
ЖЕЛмах	(л)	----	3,75	----	----	
Sвыд		----	20,5	----	----	
индЖЕЛ	(у.е.)	----	4,02	----	----	
индПДП	(у.е.)	----	----	----	----	
МВЛпр.	(л/мин)	----	149	----	----	

Scale: 12 * 16

System of Expected Values: Klement R.F., Lavrushin A.A. et al., 1986 (5...70 years old)

PARAMETER	Unit	Expected	Actual	(%)	Grade. Assessment
ЖЕЛв (FVC)	(l)	5.20	3.60	69.22	4.9 Mild reduction
ФЖЕЛ (FEV1)	(l)	5.06	3.75	74.20	4.0 Very mild reduction
ОФВ1 (FEV1)	(l)	4.38	3.73	85.20	2.3 Conditional normal
ТИФФНО (FEV1/FVC)	(%)	85.2	99.4	116.66	0.6 Above normal
ПОС (PEF)	(l/s)	9.43	10.94	115.98	1.0 Above normal
МОС25 (FEF25)	(l/s)	8.51	9.32	109.46	1.2 Normal
МОС50 (FEF50)	(l/s)	5.96	5.47	91.83	1.7 Normal
МОС75 (FEF75)	(l/s)	2.88	3.13	108.86	1.3 Normal
СОС (MMEF)	(l/s)	5.13	5.10	99.38	1.5 Normal
ОФВпос (FEVpos)	(l)	-	0.77	-	-
Тпос (Tpos)	(s)	-	0.13	-	-
Твыд (Texit)	(s)	-	1.09	-	-
ЖЕЛмакс (FVCmax)	(l)	-	3.75	-	-
Sвид (SBI)		-	20.5	-	-
индЖЕЛ (relative FVC)		-	4.02	-	-
индПДП (relative PDP)		-		-	-
МВЛпр. (MVVpredicted)		-	149	-	-

Fig. 4. Spirometry from 08.09

Рис. 4. Спирометрия от 08.09

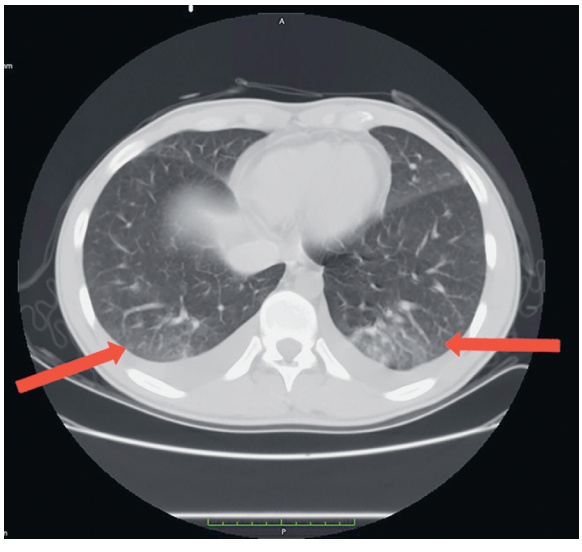


Fig. 5. **Computed tomography of the organs of the chest cavity from 06.09. The arrows indicate changes in the lungs**

Рис. 5. **Компьютерная томография органов грудной клетки от 06.09. Стрелки указывают на изменения в легких**

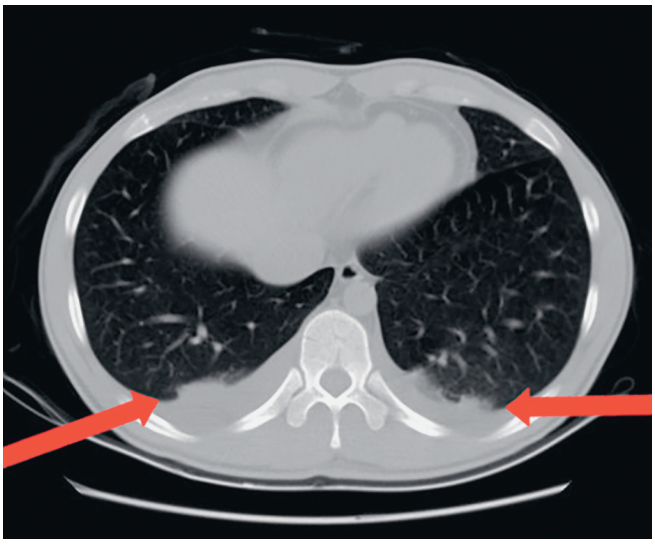


Fig. 6. **Computed tomography of the organs of the chest cavity from 08.09. Arrows indicate residual pathological changes**

Рис. 6. **Компьютерная томография органов грудной клетки от 08.09. Стрелки указывают на остаточные патологические изменения**

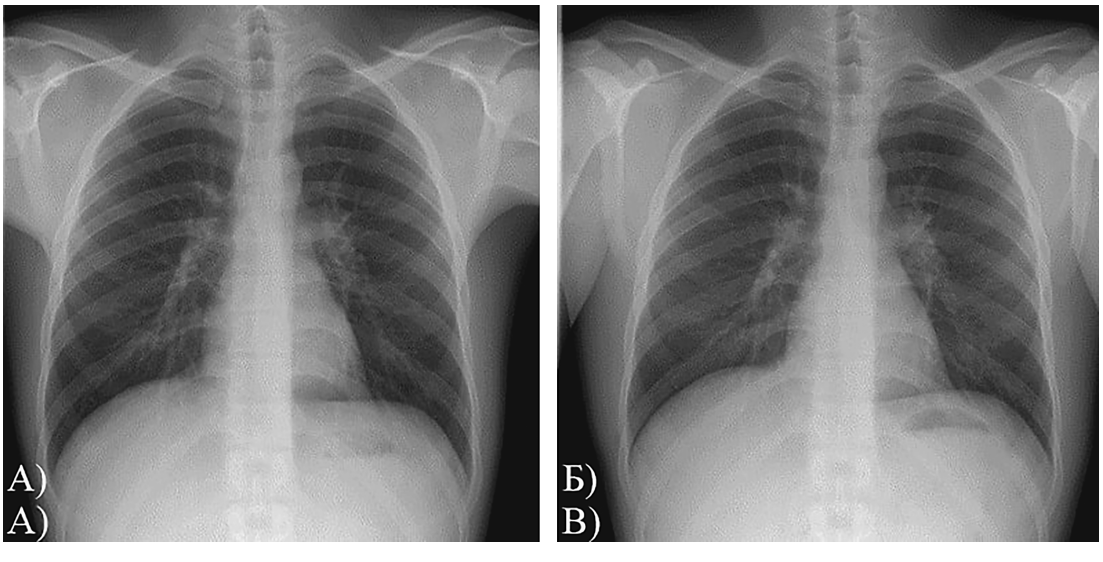


Fig. 7. **Chest X-ray: *a* — from 18.09; *b* — from 29.09**

Рис. 7. **Рентгенография органов грудной клетки: *а* — от 18.09; *б* — от 29.09**

assessment for accurate diagnosis and evaluation of respiratory system involvement. Spirometry remains appropriate for subsequent monitoring phases.

Consequently, spirometry performed on September 8 (Fig. 4) demonstrated a mild reduction in vital capacity (VC), while airway patency reached the lower limit of normal. This indicates a positive treatment response when compared with body plethysmography results obtained earlier on September 6 (Fig. 3).

The September 6 chest CT (Fig. 5) demonstrates bilateral pulmonary infiltrates (indicated by arrows), consistent with probable pneumonitis manifesting as basal consolidations.

Comparative analysis of the September 8 chest CT (Fig. 6) with the September 6 study (Fig. 5) demonstrates significant reduction in the extent of ground-glass opacities (indicated by arrows). These changes indicate rapid reversibility of the pathological process in response to appropriate pathogenetic therapy.

Therapeutic monitoring demonstrated patient stabilization with complete resolution of respiratory failure by September 11, enabling transfer from the ICU to the gen-

eral medical ward for continued management. Subsequent treatment achieved normalized clinical and laboratory parameters along with resolution of pulmonary infiltrates. Serial imaging confirmed this improvement: September 18 controls (Fig. 7, *a*) showed residual post-inflammatory vascular redistribution, while September 29 studies (Fig. 7, *b*) exhibited complete radiographic resolution.

Thus, all inpatient treatment objectives were successfully achieved, and the patient was discharged in satisfactory condition to complete phase III medical rehabilitation.

CONCLUSION

This clinical case report illustrates the full spectrum of diagnostic challenges clinicians face in managing chlorine poisoning: a prolonged latent period, atypical clinical presentation, and the condition's tendency to mimic other disease entities with similar symptomatology and physical examination findings. Timely detection of pneumotoxic lung damage and targeted comprehensive pathogenetic therapy are crucial for preventing toxic pulmonary edema, secondary complications, and the development of chronic nonspecific lung diseases. These findings underscore the need for physicians across all specialties to maintain heightened clinical vigilance against toxic exposures at every stage of patient care.

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.

Funding source. This study was not supported by any external sources of funding.

Consent for publication. Written consent was obtained from the patient for publication of relevant medical information within the manuscript.

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.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили письменное согласие пациента на публикацию медицинских данных.

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