https://doi.org/10.47183/mes.2025-275

DEVELOPMENTAL FEATURES OF IMMERSION PULMONARY EDEMA IN DIVERS

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Introduction. Immersion pulmonary edema (IPE) is a pathological condition that occurs in an aquatic environment during various activities, such as underwater engineering, scuba diving, triathlon competitions, etc. Despite a significant number of English-language publications, the problem of IPE remains insufficiently studied in Russia.
Objective. Research into the diagnosis, clinical manifestations, treatment, and prevention of IPE to optimize medical care for this pathological condition.
Discussion. The main factors leading to IPE include exposure to cold water, intense physical exertion during swimming, increased blood pressure while in water, excessive fluid intake before swimming, age over 50. Breathing 100% oxygen underwater can cause hyperoxia, oxidative stress, disruption of the alveolar–capillary membrane integrity, and surfactant deficiency, leading to fluid transudation into the pulmonary interstitial tissue and edema. Hyperoxia induces pulmonary vasoconstriction, increases hydrostatic pressure, and enhances fluid filtration into the interstitium, exacerbating IPE and contributing to the development of alveolar pulmonary edema. Clinically, IPE presents with labored breathing, acute dyspnea, coughing with hemoptysis, frothy bloody discharge, and other symptoms. A distinctive feature of this condition is the resolution of key symptoms within 48 h. On physical examination, percussion over the affected lung area reveals dullness, while auscultation detects wet rales in the lungs and murmurs characteristic of acute mitral regurgitation with left ventricular failure. Computed tomography findings include ground-glass opacities, peribronchial infiltration, and pleural effusion, predominantly on the affected side. A major limitation of this method is the inability to perform imaging immediately during an emergency ascent. Ultrasound diagnostic markers of IPE include hyperechoic reverberation artifacts (B-lines), produced by the interaction of ultrasound waves with air-fluid content in the alveoli, typical of pulmonary edema. Clinical and laboratory markers of IPE include elevated levels of copeptin, brain natriuretic peptide (BNP), ischemia-modified albumin, and high-sensitivity troponin T.
Conclusions. IPE remains an understudied yet highly dangerous pathological condition in diving and aquatic swimming. Therefore, it is crucial to educate divers, combat swimmers, professional scuba divers, and athletes (triathletes, swimmers) about preventive measures and symptom recognition when they occur during surface or underwater activities. Implementing a comprehensive approach to IPE prevention will reduce the incidence of this condition and enhance the safety of diving operations.

Keywords: immersion pulmonary edema; IPE; diving; scuba diving; risk factors; pathological condition; diving operations; triathlon; drowning; pulmonary barotrauma; thoracic compression; gas embolism; pulmonary edema symptoms

For citation: Klenkov I.R., Ankudinova Ya.I. Developmental features of immersion pulmonary edema in divers. *Extreme Medicine*. 2025;27(3):375–383. https://doi.org/10.47183/mes.2025-275

Funding: the study was carried out without sponsorship.

Potential conflict of interest: the authors declare no conflict of interest.

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Received: 20 Feb. 2025 **Revised:** 3 June 2025 **Accepted:** 6 Aug. 2025 **Online first:** 20 Aug. 2025

УДК 615.835:613.6

ОСОБЕННОСТИ РАЗВИТИЯ ИММЕРСИОННОГО ОТЕКА ЛЕГКИХ У ВОДОЛАЗОВ

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Введение. Иммерсионный отек легких (ИОЛ) — патологическое состояние, которое возникает в водной среде при различных видах деятельности: подводно-технических работах, любительском дайвинге, спортивных соревнованиях по триатлону и т.д. Несмотря на значительное количество англоязычных публикаций, в России проблема ИОЛ остается недостаточно изученной.
Цель. Исследование особенностей диагностики, клинических проявлений, лечения и профилактики иммерсионного отека легких для оптимизации медицинской помощи при данном патологическом состоянии.
Обсуждение. К основным факторам, приводящим к ИОЛ, относятся: нахождение в холодной воде, тяжелая физическая нагрузка при плавании, повышенное артериальное давление в период нахождения в воде, избыточное потребление жидкости перед плаванием, возраст свыше 50 лет; дыхание под водой 100% кислородом, вызывающим гипероксию, оксидативный стресс, нарушение целостности альвеоло-капиллярной мембраны и дефицит сурфактанта, что приводит к транссудации жидкости в интерстициальную ткань легких и отеку. Воздействие гипероксии приводит к вазоконстрикции легочных сосудов, повышению гидростатического давления и усилению фильтрации жидкости в интерстиции, что усугубляет развитие ИОЛ и обусловливает развитие альвеолярного отека легких. Клинически ИОЛ проявляется затрудненным дыханием, острой одышкой, кашлем с кровохарканьем, кровянистыми выделениями пенистой консистенции и другими симптомами. Отличительной чертой паталогического состояния является исчезновение основных симптомов в течение 48 часов. Вместе с тем при проведении перкуссии над пораженным участком легкого отмечается притупление звука, при аускультации в легких выслушиваются влажные хрипы; при аускультации сердца — шумы, характерные для острой митральной недостаточности с левожелудочковой недостаточностью. При компьютерной томографии выяв-

ляются снижение прозрачности легочных полей по типу «матового стекла», перибронхиальная инфильтрация и плевральный выпот, преимущественно на пораженной стороне. Основным ограничением метода является невозможность проведения исследования непосредственно в условиях аварийного спуска. Ультразвуковыми признаками диагностики ИОЛ можно считать наличие гиперэхогенных реверберационных артефактов (B-линий), образующихся при взаимодействии ультразвуковых волн с воздух-жидкостным содержимым альвеол, характерным для отека легких. Клинико-лабораторным маркером ИОЛ является повышение копептина, мозгового натрийуретического пептида, модифицированного ишемией альбумина, сверхчувствительного тропонина Т.
Выводы. ИОЛ остается недостаточно изученным, но крайне опасным патологическим состоянием в дайвинге и плавании на воде, поэтому водолазам, боевым пловцам, дайверам и спортсменам (триатлонистам, пловцам) целесообразно доводить информацию о мерах предосторожности при появлении его симптомов во время плавания на воде и под водой. Применение комплексного подхода в профилактике ИОЛ снизит частоту случаев появления патологического состояния и повысит безопасность водолазных спусков.

Ключевые слова: иммерсионный отек легких; ИОЛ; дайвинг; подводное плавание; факторы риска; патологическое состояние; водолазные спуски; триатлон; утопление; баротравма легких; обжатие грудной клетки; газовая эмболия; симптомы отека легких

Для цитирования: Кленков И.Р., Анкудинова Я.И. Особенности развития иммерсионного отека легких у водолазов. *Медицина экстремальных ситуаций*. 2025;27(3):375–383. https://doi.org/10.47183/mes.2025-275

Финансирование: исследование выполнено без спонсорской поддержки.

Потенциальный конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

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Статья поступила: 20.02.2025 **После доработки:** 03.06.2025 **Принята к публикации:** 06.08.2025 **Online first:** 20.08.2025

INTRODUCTION

The occupational activities of divers predispose them to specific pathological conditions including decompression sickness, ear and pulmonary barotrauma, thoracic compression, arterial gas embolism, and nitrogen narcosis (a pathological state induced by the toxic effects of nitrogen when breathing air at depths exceeding 30 m). International medical practice also recognizes immersion pulmonary edema (IPE) as a potentially fatal emergency condition during submersion [1, 2]. IPE is an acute pathological condition occurring during surface or underwater swimming, frequently affecting individuals without previous history of cardiovascular pathology (e.g., ischemic heart disease, acute/chronic heart failure, or cardiomyopathies) [3].

The actual prevalence of IPE is unknown; however, there are reports of an estimated range of 1.8% (in experienced) to 60.0% (in novice) among combat swimmers, and 1.4% among triathletes. IPE can cause severe clinical manifestations, including acute respiratory distress, hemoptysis due to erythrocyte diapedesis, paroxysmal supraventricular tachycardia, and syncope caused by myocardial hypoxia and cerebral hypoperfusion [3, 4]. At the same time, minor manifestations in divers are rarely described in the literature [3, 5]. Most of the publications on the IPE problem are presented in the form of clinical cases and focused retrospective studies. In the Russian-language literature, separate references to the manifestations of IPE can be found, mainly describing atypical forms of already known diseases (drowning, chest compression, pulmonary barotrauma, arterial gas embolism, etc.).

Fatal cases of IPE are rarely documented due to the diagnostic challenge of distinguishing them from other diving-related fatalities (drowning, ischemic heart disease, pulmonary barotrauma, etc.) [6, 7]. To date, no autopsy-confirmed cases of IPE have been reported in Russia, whereas there exist internationally established diagnostic criteria to differentiate this condition from other pathologies [8–10].

In this article, we aim to analyze the diagnostic markers, clinical presentation, management protocols, and preventive strategies for IPE to optimize the respective therapeutic interventions.

MATERIALS AND METHODS

We conducted a systematic literature review incorporating prospective and retrospective studies along with clinical case analyses. Our search strategy included international (PubMed, MEDLINE, Embase, Cochrane Library, Scopus, Web of Science) and Russian (eLibrary, CyberLeninka, RSCI) databases using the following keywords individually and in combination: immersion, exercise-induced, cold-induced, pulmonary edema, hemodynamics, cardiovascular response, water immersion, drowning, thoracic compression, pulmonary barotrauma, pulmonary edema, pulmonary hypertension, and cold shock response.

RESULTS AND DISCUSSION

Clinical epidemiology

The first documented cases of immersion pulmonary edema (IPE) were reported by Wilmshurst et al. in 1981, occurring in 71 individuals (0.18%) participating in open-water swimming events [8]. All affected individuals were ostensibly healthy adolescents aged 18–19 years. At that time, IPE diagnosis was confirmed based on the acute onset of severe dyspnea and coughing during

or immediately after swimming, coupled with auscultatory findings of pulmonary edema (inspiratory crackles throughout all lung fields) [11].

Pons et al. (1995) conducted a survey of 460 divers, identifying only 5 cases (1.1%) with IPE symptoms, including exercise-induced respiratory distress, involuntary coughing, hemoptysis (with or without frothy sputum production). With the purpose of investigating IPE pathophysiology, the researchers evaluated forearm vascular resistance, vasoactive hormone levels, biventricular function (via Doppler echocardiography) under both normothermic and cold stress conditions. Characteristic IPE hemodynamic changes were observed in just 1 out of 10 study participants [12].

A 2002 study by Mahon et al. revealed frequent IPE occurrences among U.S. Navy SEAL candidates undergoing intensive training, with an annual incidence of about 20 cases, predominance among recruits, documented recurrence in some operators, suggesting individual predisposition [13].

A review 1400 U.S. triathletes conducted by Miller et al. identified 20 cases (1.4%) with definitive IPE symptoms (exercise-associated hemoptysis with frothy secretions). The study established systemic hypertension and left ventricular hypertrophy to be the key risk factors. These conditions promote diastolic dysfunction, increasing myocardial preload and afterload [14]. Current epidemiological data indicate IPE incidence rates of 1.1% in professional divers and 1.8% among experienced combat swimmers, technical divers, triathletes [4, 11, 15–17].

Risk factors and pathogenesis

The main factors leading to immersion pulmonary edema (IPE) include exposure to cold water [13, 18, 19–21], intense physical exertion while swimming [22], elevated blood pressure during immersion [11], excessive fluid intake before swimming [13], and age over 50 [22]. Another common cause of IPE is breathing 100% oxygen underwater [19], which induces hyperoxia, oxidative stress, disruption of the alveolar-capillary membrane integrity, and surfactant deficiency, leading to fluid transudation into the pulmonary interstitium and edema. Hyperoxia causes pulmonary vasoconstriction, increased hydrostatic pressure, and enhanced fluid filtration into the interstitium, exacerbating IPE and causing alveolar pulmonary edema [23].

Consumption of 1–3 liters of water before swimming was found to increase the risk of IPE [18, 23]; however, cases where IPE occurred without prior fluid loading were also reported. An increase in circulating blood volume contributes to IPE due to resulting hypertension in the pulmonary circulation [20, 21]. The study [22] demonstrated a correlation between aspirin intake or fish oil consumption and IPE.

Three groups of factors contribute to the development of IPE:

1. *Physiological factors* (arterial hypertension, left ventricular hypertrophy, cardiac arrhythmia). Physically fit individuals and athletes may be more susceptible to IPE [10, 24], largely due to the high prevalence of left and right ventricular hypertrophy [21] and reduced cardiac chamber compliance caused by physical training [6]. Individuals with functional cardiovascular changes, such as left ventricular hypertrophy, are at a higher risk of IPE [8, 16]. Reduced lung volume due to compression under increased pressure can trigger IPE. A low number of interlobular septa and lymphatic vessels in the lungs also contributes to IPE predisposition by impairing the clearance of alveolar fluid via the lymphatic system [9, 21, 23].

2. *External factors* (cold water, intense physical exertion, depth and duration of immersion). Low water temperatures and ill-fitting wetsuits stimulate peripheral vasoconstriction, increasing preload on the left and right ventricles, thus elevating pressure in the left heart chambers and pulmonary artery [10, 20]. Exposure to cold temperatures leads to blood flow centralization, further increasing pulmonary vascular pressure, while a tight wetsuit worsens blood flow in these vessels [25].

3. *Individual characteristics* (female sex, age, obesity) [5, 6, 22]. Females are more prone to IPE, possibly due to anatomical and physiological differences in the cardiovascular system and hormonal influences. Older individuals face a higher risk of IPE due to age-related changes in the heart, blood vessels, and lungs. Excess weight places additional strain on the cardiovascular and respiratory systems, particularly during physical activity in water.

The pathogenesis of IPE is primarily driven by physiological and pathophysiological processes leading to pulmonary edema.

Hydrostatic pressure and blood flow centralization [26]. Immersion in water increases external pressure (especially in an upright position), compressing peripheral vessels and enhancing venous return to the heart. This raises pressure in the right atrium and pulmonary vessels [20].

Increased pulmonary capillary pressure (capillary stress). Elevated circulating blood volume increases hydrostatic pressure in pulmonary capillaries. When it exceeds 25–30 mmHg, transudation of fluid into the pulmonary interstitium intensifies [27].

Cold stress and vasoconstriction. Cold water triggers reflexive peripheral vasoconstriction to preserve core temperature, redistributing blood to central organs and further increasing pulmonary vascular load [28].

Impaired cardiac function. Some divers, particularly those with long-term experience, exhibit reduced myocardial adaptability to hemodynamic stress during dives. This leads to elevated pulmonary capillary pressure and acute cardiogenic pulmonary edema [23, 29].

Endothelial damage and inflammation. Hypoxia, mechanical vascular stretching, and oxidative stress injure

capillary endothelium. Inflammatory mediators (histamine, bradykinin) are released, increasing vascular permeability [30].

Lymphatic drainage insufficiency [31]. The lymphatic system fails to clear excess fluid from the lungs, exacerbating edema progression.

Independent studies by Kumar et al. indicate that a prone (face-down) position during immersion intensifies blood flow centralization, contributing to unilateral IPE linked to lateral positioning [3, 22]. The design features of breathing apparatuses can alter inspiratory/expiratory airflow, further promoting IPE.

In rebreather diving, the regulation via an automated gas supply valve creates high inspiratory resistance and restricted ventilation. This underscores the critical role of negative-pressure breathing in interstitial pulmonary edema development. Such breathing reduces intrathoracic, airway, and interstitial pressures, elevating capillary pressure with each breath. Repeated dives and ascents cause interstitial fluid accumulation, triggering pathological decompensation and IPE symptoms [4, 24].

Moreover, over 20% of hospitalized divers with IPE report prior episodes with high recurrence rates, supporting the concept of individual susceptibility. Growing evidence suggests genetic predisposition linked to polymorphisms in genes encoding surfactant proteins and endothelial growth factors [32].

Clinical Presentation and Diagnosis of Immersion Pulmonary Edema

Clinical symptoms of IPE in the presence of triggering factors (cold water, strenuous exertion, breathing 100% oxygen, etc.) may appear within 10–30 min of being in the aquatic environment [6]. In 90% of divers with IPE, dyspnea, cough, and sputum production are observed [11, 12, 19, 46], while approximately 50% experience hemoptysis [11, 18].

According to Adir et al., other symptoms (weakness, orthopnea, chest discomfort [11], dizziness [11, 12, 9], and loss of consciousness [24]) occur less frequently. Percussion over the affected area of the lung reveals dullness, which may indicate infiltration, pleural effusion, or other pathological processes [11]. On auscultation, wet rales are heard in the lungs, while cardiac auscultation may reveal murmurs characteristic of acute mitral insufficiency with left ventricular failure (gallop rhythm (S3) and pansystolic murmur of mitral regurgitation) [12]. Pulse oximetry typically shows tissue oxygen saturation below 85% [11, 8].

The differential diagnosis of IPE is generally performed with cardiovascular diseases, drowning, thoracic compression, respiratory failure, pulmonary barotrauma, and bronchial asthma [24, 33–35].

Chest X-ray examinations conducted within the first 12–18 h after IPE onset may reveal no pathological changes [11, 22]. However, typical radiographic signs may later appear, including pulmonary artery dilation, redistribution of blood flow to the upper lung lobes (cephalization), and Kerley B lines, reflecting interstitial or alveolar edema with thickening of interlobular septa [12, 13, 17, 37]. Computed tomography reveals ground-glass opacities [36], peribronchial infiltration, and pleural effusion [37, 38], predominantly on the affected side, which is associated with increased blood flow, pressure gradient, and the development of mitral regurgitation [13, 22, 39].

During lung ultrasound, parenchymal visualization is possible in the presence of pathological changes accompanied by reduced alveolar aeration, allowing the ultrasound beam to partially penetrate the interlobular septa. Diagnostic ultrasound signs of IPE include hyperechoic reverberation artifacts (B-lines), which form due to the interaction of ultrasound waves with the air-fluid content of alveoli, characteristic of pulmonary edema. Concurrently, A-lines are observed as horizontal hyperechoic structures resulting from reflection artifacts off the visceral pleura, spaced at equal intervals from one another and from the pleural line. The barcode sign is a diagnostically significant finding, indicating the cessation of lung sliding and the presence of pneumothorax as a consequence of barotrauma. Lung ultrasound allows differentiation between immersion pulmonary edema and barotrauma-induced injury, while quantitative assessment of B-lines enables determination of the degree of interstitial or alveolar edema. This method is of high diagnostic value due to its simplicity, non-invasiveness, and rapid execution [35, 38, 40].

Computed tomography is the most informative and sensitive diagnostic method for IPE [36, 41]. Characteristic signs of IPE include pleural effusion, ground-glass opacities with lobar distribution, and thickening of interlobular septa [41]. The main limitation of this method is the inability to perform the examination directly during an emergency dive.

Zavorsky et al. and Gempp et al. have identified electrocardiographic changes in IPE, manifested as nonspecific myocardial repolarization disturbances (ST-segment elevation/depression), reflecting hypoxic myocardial injury [38, 42].

Clinical and laboratory markers of IPE include elevated levels of:

- copeptin (an indirect indicator of antidiuretic hormone activity);
- brain natriuretic peptide (BNP);
- ischemia-modified albumin;
- high-sensitivity troponin T.

It was shown in [32, 43, 44] that the combination of elevated troponin T and BNP has the highest diagnostic specificity for differentiating IPE, confirming the cardiogenic component of its pathogenesis. According to the same researchers, echocardiography in IPE reveals signs of systolic dysfunction, such as global or regional

hypokinesia, reduced ejection fraction, left ventricular hypertrophy [31, 38, 42, 44].

Patients with IPE exhibit restrictive ventilation patterns, including decreased forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) with preserved Tiffeneau index (FEV1/FVC) [11, 16, 38].

Additionally, reductions were observed in absolute FVC values, decreased maximum expiratory flow rates at 25% and 75% of forced vital capacity (MEF25%, MEF75%), and impaired lung diffusion capacity [11]. While lung diffusion capacity normalizes within 24 h, other pulmonary function parameters typically recover within approximately one week.

According to Casey et al., bronchoalveolar lavage analysis revealed the presence of erythrocytes and high-molecular-weight proteins (albumins and globulins) in the lavage fluid. Notably, systemic inflammation markers (C-reactive protein, procalcitonin, neopterin, presepsin, tumor necrosis factor-α), complete blood count parameters (leukocytes, neutrophils, ESR), and protein profile indicators (total protein, IgM) remained within normal reference ranges [45].

Treatment and prevention

The IPE treatment is based on clinical guidelines for managing pulmonary edema patients, due to the lack of randomized controlled trials in divers [35, 46–48]. Primary interventions include immediate extraction from water, transfer to a warm environment, and removal of the wetsuit/dry suit. Secondary measures involve oxygen therapy and pharmacological support (diuretics, β_2-agonists, and antibiotics and corticosteroids when indicated).

With prompt treatment, 82% of patients experience complete resolution of symptoms within 48 h [30, 47–50]. However, recurrence rates in certain groups (particularly athletes) range from 13–75% [20, 49, 50]. According to Shupak et al., 75% of cases demonstrate progressive clinical manifestations during recurrent episodes, indicating individual predisposition [20, 49, 50].

Preventive measures include administration of dihydropyridine calcium channel blockers and selective PDE5 inhibitors (sildenafil) prior to diving to reduce systemic blood pressure and pulmonary hypertension [35, 46]. Mechanism of action is vasodilation (reducing smooth muscle tone and increasing venous capacitance), which may minimize IPE risk.

Clinical cases of IPE development and their analysis

We analyzed 80 incident reports and identified 16 cases that best matched the clinical presentation of IPE. In this article, we report four representative cases of IPE in divers. Among them, the first case report details the author's personal experience in diagnosing and managing IPE in a professional diver [7].

Case 1. The case involved a 35-year-old male diving instructor (designated as Z.) with one year of diving experience and 25 logged diving hours. During a dive in the Black Sea using an IDA-71P apparatus at 6 m depth, the diver experienced respiratory distress and involuntary coughing that forced him to abort the dive. Upon surfacing and boarding the support vessel, he continued coughing, producing bloody, frothy, bright red sputum. Initial physical examination revealed no pathological findings on auscultation or percussion, although chest radiography showed increased pulmonary vascular markings in the lower lung fields.

To confirm the diagnosis, a thoracic computed tomography (CT) scan was performed, revealing evidence of fluid accumulation in the lung tissue. Based on these findings, the preliminary diagnosis was pulmonary barotrauma, and therapeutic recompression was initiated using Protocol II. The diver reported symptomatic improvement during compression at 0.8 MPa. Follow-up CT imaging after completion of recompression therapy showed complete resolution of the previously observed pathological changes. Following medical rehabilitation, the diver successfully returned to professional diving activities [7].

The chest CT scan (axial projection, the lung window at the level of the right ventricular outflow tract) of diver Z. demonstrated marked irregular pulmonary aeration patterns resulting from hemorrhagic infiltration with thickening of interacinar and interlobular septa — presenting as a ground-glass opacity pattern (Fig. 1A). Additionally, thickening of interacinar and interlobular septa was observed with alveolar spaces filled by hyperdense material (blood), predominantly located in subpleural regions of the posterior and lateral segments S_{III}–S_{IX} of the right lung and S_{III}–S_{V} of the left lung (Fig. 1B). The follow-up CT scan performed after therapeutic recompression showed complete resolution of all previously identified pathological findings (Figs. 1C and 1D).

It should be noted that diver Z's radiographic findings were not characteristic of typical clinical forms of pulmonary barotrauma. The tomogram revealed signs of pronounced pulmonary edema without evidence of emphysema or pneumothorax, which are hallmark features of pulmonary barotrauma. At that time, the evident pulmonary edema was classified as an atypical form of pulmonary barotrauma. Moreover, the pathogenesis and clinical manifestations of this pathological condition distinguished it from the typical form of pulmonary barotrauma, which results from pulmonary hypertension.

Consequently, in this case, the most probable cause of the emergency situation was the development of IPE, as the clinical symptoms were specifically characteristic of this pathology.

Case 2. Diver A. was performing a dive using an IDA-71P closed-circuit rebreather to a depth of 7 m. After 25 min underwater, diver A. surfaced and reported severe coughing and chest pain. Light brown inclusions were observed in the diver's saliva. The diving physician diagnosed pulmonary barotrauma and performed therapeutic recompression, resulting in a successful outcome [7].

Case 3. Diver C. descended to 10 m using a closed-circuit breathing apparatus. After 40 min, the diver stopped responding to status checks and was brought to the surface unconscious by the safety diver. Upon regaining consciousness, diver C. complained of retrosternal pain (worsening with inspiration), progressive weakness, and coughing. Pink, frothy sputum was observed. The diving physician diagnosed pulmonary barotrauma and conducted therapeutic recompression with full recovery [38]. No differential diagnosis was performed, the root cause of the incident remained undetermined.

Thus, in the above three cases, the divers were diagnosed with pulmonary barotrauma and underwent therapeutic recompression, as there was insufficient evidence to confirm IPE (no chest CT or lung ultrasound data available). This decision was made to prevent arterial gas embolism. A potential solution in such situations would be to perform differential diagnosis using lung ultrasound to detect A- or B-lines, which serve as diagnostic criteria for pulmonary barotrauma and IPE.

Case 4. Recreational divers K. and M. conducted a dive using an AVM-5 apparatus to a depth of 7 m for amber collection in water at +3°C and air temperature of –7°C. No other individuals were present at the dive site. Upon surfacing, diver M. noticed that diver K. was missing. The following day, rescue personnel recovered diver K.'s body. Inspection of the equipment confirmed the AVM-5 was functional, with the cylinder pressure of 20 MPa and air quality meeting regulatory standards. The investigative commission concluded that the fatality resulted from drowning, hypothesizing that the AVM-5 regulator of diver K. froze in the extreme cold, ceasing air delivery. Experiencing breathing difficulties underwater, diver K. expelled the mouthpiece but failed to surface due to negative buoyancy [7].

Research on cold-water immersion demonstrates that hemodynamic changes induced by cold exposure elevate pulmonary artery pressure and increase pulmonary ventilation, disrupting the alveolar–capillary barrier.

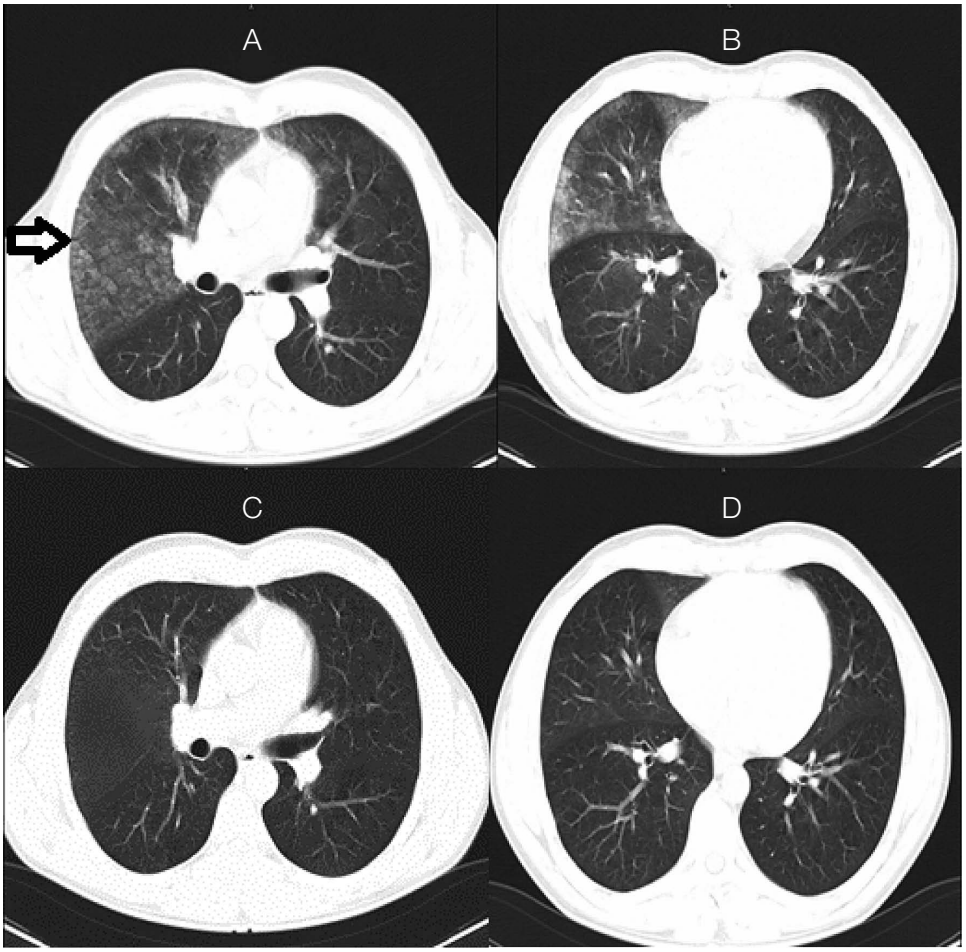

Figure prepared by the authors based on their own data

Fig. 1. Computed tomography of the thorax in diver Z. demonstrating ground-glass opacity (arrow)

These alterations promote fluid transudation into alveolar spaces.

This case bears strong resemblance to IPE-related incidents described in international literature [8, 11, 13, 18], where cold water exposure constitutes the primary risk factor for this pathology. This factor, particularly when combined with strenuous underwater exertion, frequently leads to IPE and subsequent drowning.

CONCLUSION

Despite the considerable number of English-language publications on immersion pulmonary edema (IPE), this subject remains insufficiently studied in Russia. The epidemiology of IPE varies significantly across different populations: the highest incidence (up to 60%) is observed among combat swimmer recruits, while among triathletes, swimmers, and professional divers it does not exceed 1.8%.

The key risk factors for IPD include hypothermia, hypertension, physical exertion in aquatic environments, overhydration, breathing hyperoxic gas mixtures, and age over 50 years. The primary IPE symptoms are inspiratory dyspnea and non-productive cough progressing to bloody sputum production.

The gold standard diagnostic methods for IPE are chest CT scans and lung ultrasound examinations. Due to the lack of evidence-based protocols, treatment remains symptomatic and follows general clinical guidelines for managing pulmonary edema. The main preventive measure involves medical screening to identify individuals predisposed to IPE development.

The conducted analysis of diving accidents in Russia confirmed the occurrence of cases with IPE symptomatology. Promising research directions include in-depth investigation of IPE pathogenesis and development of rapid diagnostic methods that could enable immediate identification of this condition at dive sites.

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Authors' contributions. All authors confirm that their authorship meets the ICMJE criteria. The greatest contribution is as follows: Ilyas R. Klenkov — administrative management, study design, literature and legislation analysis, clinical guidelines analysis, and text writing; Yana I. Ankudinova — study conducting, data processing and analysis, and text writing.

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