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Cold urticaria in cold-water and open-water swimmers: anaphylaxis risk, diagnosis and practical recommendations: a narrative review

Dawid Oskar Mazurek

ORCID: https://orcid.org/0009-0004-7501-5941

E-mail: mazurekdawid6@gmail.com

Medical University of Lublin, Lublin, Poland

Julia Weronika Duszejko

ORCID: https://orcid.org/0009-0000-0974-5170

E-mail: dus6@op.pl

Medical University of Lublin, Lublin, Poland

Jarosław Józef Kucharski

ORCID: https://orcid.org/0009-0007-7470-6451

E-mail: jarek.kucharski661@gmail.com

Medical University of Lublin, Lublin, Poland

Zuzanna Zofia Cecot

ORCID: https://orcid.org/0009-0009-6280-4278

E-mail: zuzanna.cecot.048@gmail.com

Medical University of Lublin, Lublin, Poland

Maciej Mariusz Lesiczka
ORCID: https://orcid.org/0009-0003-3234-9549
E-mail: lesiczkamaciek@gmail.com
Medical University of Lublin, Lublin, Poland

Filip Lenartowicz
ORCID: https://orcid.org/0009-0004-1980-9640
E-mail: lenartowiczfilip00@gmail.com
Medical University of Lublin, Lublin, Poland

Oliwia Gałczyńska
ORCID: https://orcid.org/0009-0004-0738-8199
E-mail: oliwiagalczynska07@gmail.com
Medical University of Lublin, Lublin, Poland

Katarzyna Kuklińska
ORCID: https://orcid.org/0009-0007-1242-3958
E-mail: kasia.kuklinska03@gmail.com
Medical University of Lublin, Lublin, Poland

Anna Maciaszczyk
ORCID: https://orcid.org/0009-0003-3872-0310
E-mail: anna.maciaszczyk05@gmail.com
Medical University of Lublin, Lublin, Poland

Alicja Martyna Lewczuk
ORCID: https://orcid.org/0009-0004-8827-3739
E-mail: alicja.lewczuk00@gmail.com
Medical University of Lublin, Lublin, Poland

Corresponding Author
Dawid Oskar Mazurek, mazurekdawid6@gmail.com

Abstract

Background. Cold urticaria (ColdU) is a chronic inducible urticaria in which cold activates skin mast cells. Cold-water and open-water swimming are increasingly popular. In people with ColdU, whole-body immersion can provoke life-threatening reactions.

Aim. To review data on the risk of anaphylaxis in swimmers with ColdU, its diagnosis and management, and to propose practical recommendations for cold-water and open-water swimming

Material and methods. Narrative review of guidelines, systematic reviews, randomised trials, cohort studies and case reports identified in PubMed and reference lists.

Results. In most cohorts, anaphylaxis occurred in 17–37% of patients with ColdU. Complete cold-water immersion was its most frequent trigger. In water, early anaphylaxis is easily mistaken for cold shock, and rescue from the water carries additional risk. Standard cold stimulation tests are negative in up to one third of patients. Open-water races may be held in water as cold as 16 °C, which is below the critical temperature threshold of many patients. Intramuscular adrenaline is the treatment of choice, yet autoinjectors are underprescribed. Higher-dose antihistamines and omalizumab improve disease control but have not been shown to prevent anaphylaxis during immersion.

Conclusions. Swimmers with ColdU need individual risk assessment, education, supervised swimming with adrenaline at hand and horizontal extraction from the water. Prospective data from athletes are lacking.

Keywords: cold urticaria; anaphylaxis; open-water swimming; winter swimming; cold water immersion; epinephrine; athletes; water safety

1. Introduction

Open-water swimming, winter swimming and cold-water bathing have all gained popularity. In the United Kingdom, according to Sport England survey data cited by Massey et al., the number of people who swim in open water at least twice a month has risen by 138% since 2016 [1]. Ice swimming in water colder than 5 °C now has its own competitions [2]. There is no single definition of cold water. Water below about 15 °C is usually regarded as cold, because it evokes a marked cold shock response [2,3]. Many health benefits are claimed for cold-water swimming, but most of these claims rest on small or low-quality studies [3,4], whereas the risks, including drowning, cardiac arrest and hypothermia, are better documented [2,3]. Official UK data recorded 193 accidental water-related deaths in 2024 [5].

For some swimmers, an underlying disease adds a further hazard. Cold urticaria (ColdU) is a form of chronic inducible urticaria in which cold activates mast cells and causes wheals, angioedema or both [6,7]. The reaction is not always limited to the skin: anaphylaxis has been reported in about one fifth (pooled estimate) to more than one third of patients [8,9], and complete immersion in cold water is its most frequently identified trigger [8]. On land, a brief fall in blood pressure or a short loss of consciousness is rarely fatal. In water, it can lead to drowning. Patients also report that the disease limits their everyday activities and hobbies [10].

The relevant evidence comes from two fields that seldom cite each other. Papers on allergy rarely consider what happens to the body in cold water, while papers on cold-water swimming and drowning hardly ever mention ColdU as a possible cause of an incident. Because standard provocation tests are also negative in a sizeable proportion of patients [11], athletes, coaches, lifeguards and doctors may miss the disease or underestimate its danger.

Research objective. The aim of this review is to summarise data on the epidemiology, pathogenesis, diagnosis and treatment of ColdU, with a focus on anaphylaxis during cold-water swimming and other open-water activities.

Research problems. (1) How often, and by what mechanisms, does cold-water immersion lead to systemic reactions in ColdU? (2) Why may the diagnosis be missed in swimmers? (3) How should acute management, rescue and long-term counselling be adapted for patients who want to keep swimming, including competitive athletes?

Research hypotheses. Not applicable. As a narrative review, this work does not test predefined hypotheses.

2. Research materials and methods

2.1. Literature search and study selection

This is a narrative (non-systematic) review. We searched PubMed and screened the reference lists of key papers. The last search was performed on 12 October 2026, and priority was given to studies from the last ten years. We used the following terms and their combinations: "cold urticaria", "cold-induced urticaria", "cold-induced anaphylaxis", "chronic inducible urticaria", "cold stimulation test", "critical temperature threshold", "cold water immersion", "cold shock", "cold water swimming", "winter swimming", "drowning", "anaphylaxis" and "adrenaline autoinjector". For anti-doping aspects we consulted the current World Anti-Doping Agency (WADA) Prohibited List [12].

We included international guidelines and consensus statements, systematic reviews and meta-analyses, randomised controlled trials, cohort studies, and selected case reports and research letters that provided data not available elsewhere. Only publications in English were considered. We excluded papers that did not deal directly with cold urticaria, anaphylaxis or the physiology of cold-water immersion, as well as conference abstracts and other sources that had not been peer reviewed. The only exceptions to the latter rule were the official statistics, regulatory documents, competition rules and product information used in sections 1, 3.6 and 3.7. Three of the authors selected the studies and resolved disagreements by discussion. In total, 56 sources were included (51 peer-reviewed publications and 5 official documents), and the bibliographic details of each were checked in PubMed or, for documents not indexed there, on the website of the issuing organisation. Older papers were kept only if they remain the main source on a given question and have not been superseded; on this basis, 6 of the 56 cited sources were published before 2016.

Because the included studies differ in design, population and outcome definitions, particularly in how they define anaphylaxis, we synthesised the data qualitatively and did not perform a formal risk-of-bias assessment, meta-analysis or statistical analysis. Numbers are given as reported in the original papers, and the main cohorts are summarised in Table 1.

3. Research results

3.1. Epidemiology and classification

Urticaria is a mast cell-driven disease that presents with wheals, angioedema or both. The current international guideline divides chronic urticaria into a spontaneous form, in which symptoms arise without a specific external trigger, and chronic inducible urticaria (CIndU), in which a defined physical or other stimulus provokes them; both forms impair quality of life as well as performance at work and school [7]. ColdU belongs to the CIndU group, with wheals and/or angioedema appearing after exposure to cold [6,7].

In a meta-analysis, ColdU accounted for 7.6% of chronic urticaria and 26.1% of CIndU cases, which places it among the more common inducible urticarias [9]. It affects children as well as adults. Some patients only ever notice a few wheals on cold-exposed skin, while others react with generalised symptoms or anaphylaxis [6,8]. The largest study so far, the international cross-sectional COLD-CE study (comprehensive evaluation of cold urticaria and other cold-induced reactions), enrolled 551 patients in 32 urticaria centres of reference and excellence; 412

(75%) of them had typical ColdU, and 91% of these were adults [8]. Much of the remaining data comes from paediatric cohorts [13,14,15].

ColdU can be acquired or hereditary, and the acquired form predominates. Hereditary cold-induced syndromes are rare monogenic disorders with a different course and need a separate diagnostic approach [16]. Acquired ColdU is further split into a typical form, in which the standard cold stimulation test (CST) is positive, and an atypical form, in which the test is negative even though the history clearly points to cold-induced symptoms [6]. The two groups differ in age at onset, comorbidities and the frequency of severe symptoms [11], so the distinction has practical weight, and atypical ColdU itself covers several distinct clinical subtypes [17]. Most acquired cases are primary (idiopathic). Secondary forms linked to infections, cryoproteins or haematological malignancies are uncommon [6,18,19].

ColdU tends to persist, in an athlete usually through several training seasons. In a 20-year follow-up study, data were obtained from 41 of 99 patients; the estimated resolution rate was 17.9% at 5 years and 24.5% at 10 years, and 55% reported stable or worsening disease, although patients with persistent disease may have been more likely to respond [18]. In a Spanish tertiary-centre cohort of 74 patients, 25.7% achieved complete resolution, less often after paediatric onset (5.0% vs 33.3% with adult onset) and after severe systemic reactions (7.1% vs 36.0% with localised symptoms) [20].

3.2. Pathogenesis

Skin mast cells are the main effector cells. On cold exposure they degranulate and release histamine and other mediators, which increase vascular permeability and dilate vessels, so that wheals, itch and angioedema follow [6,21]. The wheals usually appear only once the skin warms up again and most fade within an hour [6], so mediators may keep entering the circulation after the cold exposure has ended.

According to the most widely accepted hypothesis, cold induces the formation of skin autoallergens, which are then recognised by IgE bound to mast cells [6]. The response to omalizumab, an anti-IgE antibody, supports a role for IgE [21,22], although the autoallergens themselves have not yet been identified. Besides the IgE pathway acting through the high-affinity receptor FcεRI, IgE-independent mechanisms have been proposed, including mast cell activation via the Mas-related G protein-coupled receptor X2 (MRGPRX2) and a contribution from neuropeptides [21]. These pathways could explain why some patients respond poorly to anti-IgE treatment, and several drugs now in development target them [21].

How much skin is cooled shapes the clinical picture. A cold object usually causes wheals only where it touches the skin, whereas cooling of a large part of the body surface, as in swimming, may release enough mediators to cause systemic symptoms and anaphylaxis [23]. A case report of monoclonal mast cell activation syndrome with a KIT D816V mutation that presented as severe ColdU suggests that a clonal mast cell disorder should be considered in patients with severe disease [24].

Cryoproteins may matter in a subgroup of patients, but they do not explain ColdU as a whole. In pooled published data, cryoglobulins, cryofibrinogens and cold agglutinins were elevated in 3.0%, 1.1% and 0.7% of patients, respectively, and none of 49 prospectively tested patients had cryoglobulins or cryofibrinogens [19]. A prospective single-centre cohort of 35 patients, however, gave a different picture. Cold agglutinins were found in 46% and

cryoglobulins in 27% of those tested, and patients with cold agglutinins more often reacted to cold air and to immersion in water below 25 °C (75% vs 37%) [25].

Current data argue against an autoinflammatory basis of acquired ColdU, although how mast cells are activated in the disease is still only partly understood. Patients with acquired ColdU did not carry pathogenic variants in genes linked to autoinflammatory syndromes [26], and in a randomised placebo-controlled trial in 20 patients, interleukin-1 (IL-1) blockade with rilonacept did not change the critical temperature threshold [27]. In a phase 3 trial, dupilumab did not improve the signs and symptoms of ColdU or the ice cube test result compared with placebo [28].

3.3. Cold-water immersion as a specific trigger

For a patient with ColdU, cold water is a much harsher stimulus than cold air or a cold object. Air or an object usually cools a limited patch of skin, whereas immersion cools nearly the whole body surface at once, and because the thermal conductivity of water is 24 times that of air, skin temperature falls within seconds of entry [3]. Mast cells can then degranulate over a large area at the same time, and the amount of histamine and other mediators reaching the circulation may be enough to cause systemic symptoms [6,23].

In COLD-CE, complete cold-water immersion was the most frequent trigger of cold-induced anaphylaxis (ColdA): it had caused ColdA in 27% (112 of 412) of patients with typical ColdU, twice as often as cold air (13%), although in tropical countries cold air played a larger role [8]. In an Australian series of 99 patients, every episode of anaphylaxis followed immersion in cold water [18], and in a Spanish cohort cold water was reported by 13 of 14 patients with severe systemic reactions [20]. Children show a similar pattern. Swimming caused 77.6% of grade 3 reactions in a cohort of 415 paediatric patients [13], and in another paediatric cohort swimming on windy days was reported more often by children with anaphylaxis than by other children with ColdU [15].

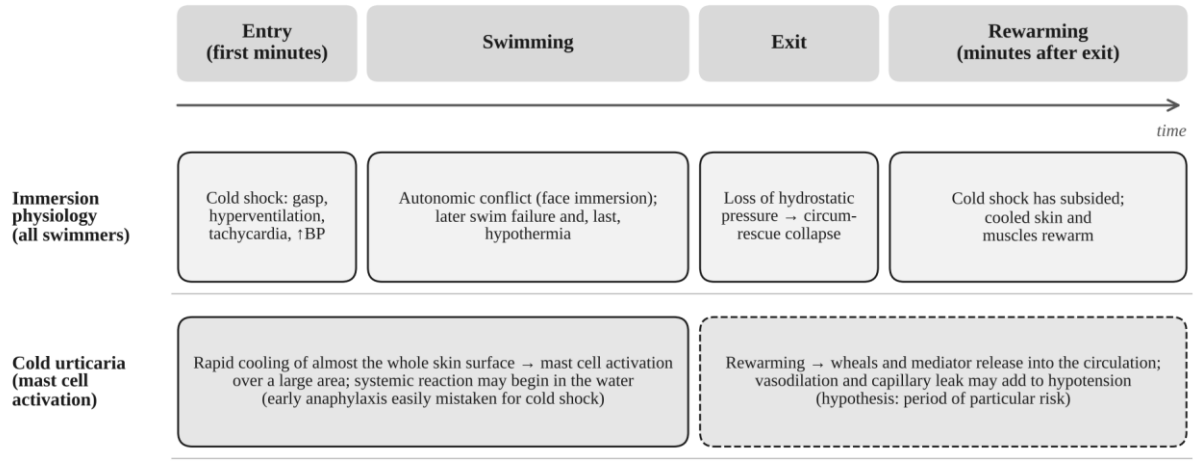
Reactions are not limited to cold open water. In 45 patients with acquired ColdU, the critical temperature threshold (CTT) ranged from 4 to 27 °C, with almost half of the patients at 20 °C or higher and 71% at 16 °C or higher. Patients with severe disease had higher thresholds than those with mild disease (22.0 vs 12.3 °C) [29]. In the Spanish cohort, CTT values ranged from 4 to 26 °C [20]. A scientific diver even continued to have symptoms in pools heated to about 32 °C (90 °F), despite taking cetirizine at up to four times the standard dose [30].

Immersion also sets off physiological responses that have nothing to do with ColdU. Sudden entry into cold water triggers the cold shock response, with an involuntary gasp, hyperventilation that is hard to control, tachycardia, peripheral vasoconstriction and a rise in blood pressure. It peaks in the first minutes and can lead to aspiration and drowning, particularly in people who are not used to cold water [2,3,31]. When the face is submerged and the breath is held, the diving reflex adds parasympathetic drive to the sympathetic one, and this "autonomic conflict" can provoke cardiac arrhythmias [32]. Longer immersion cools muscles and peripheral nerves and makes swimming less effective (swim failure), while hypothermia develops last [2,33].

Regular winter swimmers can blunt the cold shock response through repeated immersions [2,3]. No study, however, has shown that this adaptation lowers mast cell reactivity in ColdU, and habituation acquired informally through winter swimming cannot be regarded as treatment. Deliberate cold desensitisation, with gradually increasing exposure to cold water followed by daily maintenance exposure, may reduce ColdU symptoms, but it is rarely used because it requires supervised, repeated exposure and itself carries a risk of systemic reactions [21].

In a swimmer with ColdU these mechanisms overlap. Breathlessness, a racing heart, dizziness and weakness are easily attributed to ordinary cold shock, and early anaphylaxis may go unrecognised. A short loss of consciousness or sudden weakness in the water usually leads to submersion [31], after which the original cause of the incident may never be identified. There are no reliable data on the fatality rate of ColdU [6].

The timing of symptoms is also important. In ColdU, wheals typically develop during rewarming [6], that is, after the swimmer has left the water and blood returns to the cooled skin. Leaving the water also removes the hydrostatic pressure on the body and reduces venous return, which is the mechanism behind circum-rescue collapse in immersion victims [33], and histamine-induced vasodilation and capillary leak may lower blood pressure further. This sequence has not been studied directly in patients with ColdU. If it holds, the first minutes after exit would be a period of particular risk, and the swimmer should not be left unobserved. Systemic reactions can, however, also begin while the swimmer is still in the water. The sequence of hazards from entry to rewarming is summarised in Figure 1.



Source: own elaboration based on [3,6,23,32,33].

Figure 1. Overlap of immersion physiology and cold urticaria during a cold-water swim. The dashed box marks a hypothesis that has not been studied directly.

3.4. Risk of anaphylaxis

Systemic reactions are common in ColdU and can be life-threatening. COLD-CE defined ColdA as acute cold-induced involvement of the skin or visible mucosa with at least one cardiovascular (hypotension, loss of consciousness or presyncopal symptoms), respiratory or gastrointestinal symptom. By this definition, 151 (37%) of the 412 patients with typical, CST-positive ColdU had experienced ColdA [8]. A meta-analysis of 14 studies put the pooled frequency of anaphylaxis at 21.49% (95% confidence interval 15.79–28.54), with substantial heterogeneity ($I^2 = 69\%$) [9].

Single-centre and paediatric cohorts report somewhat lower figures, of about 17–19% (Table 1) [13,14,20]. An even lower figure, 1 of 27 patients (3.7%), came from a small cohort in a tropical country [34], which fits the lower share of immersion-related reactions in tropical climates [8]. The spread may reflect different definitions of anaphylaxis, retrospective data collection and the higher share of severe cases in specialist centres.

Table 1. Frequency of systemic reactions and the role of swimming in the main cohorts of patients with cold urticaria

Study	Design and population	n	Outcome definition	Frequency	Role of swimming / immersion
Bizjak et al. 2022 (COLD-CE) [8]	International multicentre cross-sectional study (32 centres); typical ColdU; 91% adults	412	ColdA: skin or mucosal symptoms plus ≥1 cardiovascular, respiratory or gastrointestinal symptom	37%	Complete cold-water immersion caused ColdA in 27% of patients (cold air 13%)
Prosty et al. 2022 [9]	Systematic review (22 studies) and meta-analysis (14 studies); studies published 2011–2021	–	Anaphylaxis as defined in primary studies	21.49% (95% CI 15.79–28.54; I^2 = 69%)	–
Deza et al. 2016 [20]	Single tertiary centre (Spain), retrospective; adults and children (27% paediatric onset)	74	Severe systemic reactions (type III)	18.9%	Cold water was a trigger in 73% of patients and in 13 of 14 with type III reactions
Yee et al. 2019 [13]	Single paediatric tertiary centre (USA), retrospective, 1996–2017	415	Grade 3 reactions (anaphylaxis)	18.6%	Swimming caused 77.6% of grade 3 reactions
Jain and Mullins 2016 [18]	Single allergy practice, retrospective, with follow-up questionnaire; adults and children	99	Anaphylaxis (respiratory and/or cardiovascular compromise)	28.3%	All episodes of anaphylaxis followed immersion in cold water
Prosty et al. 2022 [14]	Single paediatric centre (Canada), prospective, 2013–2021	52	ColdA: ≥2 organ systems or hypotension	17.3%	Swimming triggered ColdA in 4 of 9 children, cold air in 4 and both in 1
Karabag Citlak et al. 2023 [15]	Multicentre (11 paediatric centres in	83	ColdA	–	Swimming reported by 70.0% of children

Study	Design and population	n	Outcome definition	Frequency	Role of swimming / immersion
	Turkey, 2010–2022), retrospective				with ColdA vs 28.9% of others; swimming in wind 50.0% vs 4.1%

Source: own elaboration based on the cited studies.

Notes: ColdA, cold-induced anaphylaxis; ColdU, cold urticaria; CI, confidence interval; –, not reported or not applicable. For the meta-analysis, the number of pooled studies is given in the Design and population column instead of n.

Swimming is thus the main trigger of systemic reactions in adults and children (see section 3.3) [8,13,15]. Because none of these studies recorded how often patients swim, however, the figures describe the share of reactions attributable to swimming and not the risk per swim.

Some clinical features point to patients at higher risk. In COLD-CE, generalised wheals, angioedema, acral swelling, oropharyngeal or laryngeal symptoms and itchy earlobes marked severe disease, and all of these except generalised wheals and acral swelling also predicted cardiovascular symptoms, as did a previous systemic reaction to a Hymenoptera sting. Hypotension or loss of consciousness after complete immersion was linked to paediatric onset, although all these risk factors were based on patient history [8]. In the extended COLD-CE cohort (535 patients), oropharyngeal mucosal angioedema after cold food or drinks occurred in 32.1% of patients and was linked to cardiovascular, respiratory and gastrointestinal symptoms [35]. In children, a raised eosinophil count was associated with ColdA (adjusted odds ratio 1.38) [14], and 11.7% of children with a negative CST had also had anaphylaxis [13].

Among patients with typical ColdU in COLD-CE, those who also had chronic spontaneous urticaria (40 patients, 10%) had ColdA much less often than those who did not (4% vs 39%), for reasons that remain unclear [8]. The risk does not fade quickly. In one paediatric cohort the resolution rate was only 4.8 per 100 patient-years [14], and in the largest one 7% of 415 children had more than one episode of anaphylaxis [13].

3.5. Diagnosis and diagnostic pitfalls

Diagnosis rests on the history and is confirmed by a CST [6,36]. In the standard test, a melting ice cube in a thin plastic bag is placed on the forearm for 5 minutes and the site is assessed 10 minutes later, once the skin has rewarmed. Cold-water arm immersion is an alternative when the ice cube test is negative, but it carries a risk of systemic reactions [36]. The TempTest® device measures the CTT along a gradient from 4 to 44 °C with an accuracy of 1 °C [37]. The two tests are not interchangeable, since 16.7% of 318 COLD-CE patients tested with both reacted only to the ice cube [35]. In a small study of 45 patients, the CTT correlated with patient-rated disease severity ($r = 0.53$) and its changes followed changes in disease activity ($r = 0.64$), so it can also be used to monitor treatment [29]. The Cold Urticaria Activity Score (ColdUAS), a four-item patient-reported questionnaire with a 24-hour recall period, is designed for prospective monitoring and was still being validated at the time of its publication [38].

The most important pitfall is a negative standard test in a patient whose history is typical. In an analysis of COLD-CE limited to the 11 centres that enrolled both test-positive and test-negative patients, 121 of 364 patients (33.2%) had a negative standard CST [11]. The overall COLD-CE proportion of 75% test-positive patients [8] therefore underestimates how common negative tests are, because some centres enrolled only test-positive patients [11]. Earlier cohorts reported negative or non-classical test results in 8–41% of patients, with different definitions and protocols [11]. In a tertiary centre cohort, 28.4% had negative tests and were classified as atypical ColdU [20], and in paediatric cohorts the test was negative in 30.1% [13] and 28.9% [15] of the children tested.

CST-negative patients were younger and had earlier disease onset; they more often had a family history of ColdU and concomitant chronic spontaneous urticaria (24.0% vs 9.1%) and more often developed symptoms only when they felt cold (28.1% vs 9.1%) [11]. A recent systematic review described 10 subtypes of atypical ColdU, among them delayed, extended-exposure, generalised-exposure, localised, water-dependent and food-dependent ColdU and cold-induced cholinergic urticaria, and proposed modified cold stimulation tests for them, eight of which were successfully pilot-tested [17]. In some patients with a negative TempTest, the diagnosis was confirmed only by immersing the hand and forearm in iced salt water [34], a test much closer to the exposure of a swimmer.

Test-negative patients are not a low-risk group. Difficulty breathing (28.4% vs 10.7%) and cardiovascular symptoms (31.7% vs 15.7%) were more frequent in CST-positive patients. Full cold-water immersion, however, was still a trigger in 68.6% of CST-negative patients, and only 9.1% of them had been prescribed an adrenaline autoinjector, compared with 30.5% of CST-positive patients [11]. A convincing account of reactions after swimming outweighs a negative test result [11,23]. The test itself is not free of risk: a woman with previous cold-induced anaphylaxis had two episodes of anaphylaxis 5–10 minutes after diagnostic provocation [39]. Such reactions appear to be rare, but patients with previous systemic reactions should be tested only where emergency treatment is available and observed afterwards.

Differential diagnosis and secondary causes are another source of error. Cryoproteins are found in only some patients and their clinical meaning is uncertain (see section 3.2) [19,25], and screening for infections is recommended only when the history suggests it [6]. Severe ColdU may point to a clonal mast cell disorder, which justifies measuring basal serum tryptase and, if it is raised, testing for the KIT D816V mutation [24]. Hereditary and autoinflammatory syndromes (e.g. those linked to NLRP3, NLRP12, NLRC4 or PLCG2) should be considered in patients with early onset, fever or a positive family history [16,26], although no pathogenic variants were found in 50 patients with acquired ColdU [26]. Secondary causes are rare; in the 20-year follow-up study they were identified in only 2 of 99 patients, both with a B-cell malignancy [18].

In athletes, overlapping symptoms make diagnosis even more difficult. Breathlessness and tachycardia in cold water may be taken for cold shock, and in divers the skin lesions of ColdU may resemble decompression sickness [30]. Exercise-induced anaphylaxis is another possibility; in its food-dependent form, symptoms typically develop when exercise takes place within about 4 hours of eating a food to which the patient is sensitised [40]. Cold-induced cholinergic urticaria, an atypical ColdU subtype provoked by exercise in a cold environment, is an obvious candidate in swimmers who train in cold water [17].

Breathlessness, cough and frothy or blood-stained sputum during open-water swimming or the swim leg of a triathlon suggest swimming-induced pulmonary oedema, which typically develops during heavy exertion, often in

cold water, and has been proposed as a cause of deaths in triathletes [41]. An unexplained loss of consciousness or collapse in cold water calls first for cardiological assessment, because arrhythmias related to autonomic conflict or channelopathies such as congenital long QT syndrome may precipitate submersion [32,33]. ColdU belongs in the differential diagnosis of such events, but not as the only explanation. A generalised rash, angioedema or systemic symptoms after cold-water swimming should prompt referral to an allergist.

3.6. Acute management and treatment

Intramuscular adrenaline is the first-line treatment of anaphylaxis, including ColdA [23,42,43]. In healthcare settings it is injected into the mid-thigh at a dose of 0.01 mg/kg, up to 0.5 mg [42,43]. For adolescents and adults at risk, autoinjectors of at least 0.3 mg should be prescribed, and the dose should be repeated if symptoms do not improve, or if they worsen, 5–10 minutes after the first injection [42]. International guidance notes that adrenaline is still underused and recommends that every patient who has had anaphylaxis be referred to a specialist to identify the cause and learn self-management [43]. Patients at risk should be prescribed an adrenaline autoinjector and receive structured training in its use [42].

In ColdU, autoinjectors are underprescribed. Among COLD-CE patients with typical ColdU, no concomitant chronic spontaneous urticaria and previous ColdA, only 37% had an autoinjector and only 8% had ever been treated with adrenaline; among those whose ColdA had been caused by complete cold-water immersion, the corresponding figure was 41% [44]. The COLD-CE investigators proposed prescribing an autoinjector to patients with typical ColdU who have had ColdA and to those who report cold-induced angioedema, oropharyngeal or laryngeal symptoms or itchy earlobes [8]. In their view, long disease duration, paediatric onset, generalised wheals, acral swelling, a short critical stimulation time threshold and large wheals with pseudopodia also deserve attention [8]. Neither antihistamines nor glucocorticoids replace adrenaline: antihistamines have been shown to relieve only cutaneous symptoms, and evidence for the benefit of glucocorticoids is limited [42]. Serum tryptase helps to confirm anaphylaxis and may reveal an underlying mast cell disorder [24,42]. Ideally, it is measured between 30 minutes and 2 hours after the onset of the reaction and compared with a baseline value obtained at least 24 hours after recovery.

Rescue from the water changes several steps of management. The swimmer has to be brought out first, but the extraction itself carries risk, because lifting a casualty out vertically removes the hydrostatic pressure on the body, reduces venous return and can cause circum-rescue collapse; horizontal extraction is therefore recommended whenever possible [33]. A UK series of fatal anaphylaxis points the same way [45]. In all 10 out-of-hospital deaths from anaphylactic shock with a known postural history, the patient had either moved to a more upright posture or been supported sitting up, and in 4 of them death followed within seconds of the change. The proposed mechanism, loss of venous return emptying the vena cava, parallels circum-rescue collapse [45].

A swimmer with suspected ColdA should thus be laid flat with the legs raised, and any abrupt change to a more upright posture should be avoided [42,45]. An unconscious casualty who is breathing is placed in the recovery position, and a casualty in respiratory distress may sit up with the legs elevated [42]. The casualty should be given adrenaline without delay, shielded from wind and further cooling, and taken to hospital. Because protracted and biphasic reactions can occur, EAACI suggests monitoring for 6–8 hours after respiratory compromise and for at least 12–24 hours after hypotension [42]. If cardiac arrest occurs, resuscitation follows the current European

Resuscitation Council guidelines for special circumstances, which cover drowning, hypothermia and anaphylaxis [46]. In drowning, the arrest is primarily hypoxic, so effective ventilation comes first [46].

Long-term management consists of cold avoidance and regular second-generation H1-antihistamines (sgAH) [6,7], which are still the only drugs licensed for ColdU [21]. If the standard dose does not control symptoms, the guideline recommends increasing it up to fourfold, which is an off-label use [7]. Severe ColdU responds less well to antihistamines and may warrant early higher dosing and closer follow-up [20]. A systematic review and meta-analysis found higher doses of sgAH more effective than standard doses in raising cold provocation thresholds, with no significant rise in adverse effects [47]. In randomised, placebo-controlled crossover trials, desloratadine 20 mg was superior to 5 mg in 30 patients without causing more drowsiness [48], which matters for athletes whose safety in water depends on alertness. Rupatadine 20 mg and 40 mg also improved both thresholds compared with placebo in 23 patients, although only 30% and 50%, respectively, were free of wheals on cold provocation [49].

Patients who do not respond to antihistamines may receive omalizumab off-label [6,21]. In a randomised placebo-controlled trial in 31 such patients, omalizumab 150 mg or 300 mg every four weeks lowered the CTT, and 40–44% had a complete response at week 10, compared with none on placebo; the effect was lost within 6 weeks of the last injection [22]. In a meta-analysis, however, only 5.95% of patients had received omalizumab [9]. Dupilumab and remibrutinib, which the current guideline includes for chronic spontaneous urticaria [7], have not been shown to be effective in ColdU. In a phase 3 trial in 82 adults, dupilumab did not differ from placebo on any efficacy endpoint over 24 weeks [28], and rilonacept did not change the CTT [27]. Bruton's tyrosine kinase inhibitors, anti-KIT antibodies and MRGPRX2 antagonists are under investigation [21]; in an open-label study, a single intravenous dose of the anti-KIT antibody barzolvolimab led to a complete response in all ten treated patients with ColdU [50].

Competitive athletes have to take anti-doping rules into account. Adrenaline (except local administration) and systemic glucocorticoids are prohibited in competition but not out of competition, which covers most training swims, whereas H1-antihistamines and omalizumab are not on the Prohibited List [12]. Life-saving treatment must never be delayed for this reason. After emergency use of an autoinjector during a competition, the athlete can apply for a retroactive therapeutic use exemption, which the International Standard allows when emergency treatment was necessary (Article 4.1a) [51].

Drug treatment raises the threshold at which reactions occur, but since no drug has been shown to prevent anaphylaxis during whole-body immersion, a patient whose disease is well controlled should still carry an adrenaline autoinjector when swimming in cold water.

3.7. Practical recommendations

We found no studies of safety strategies in athletes or regular cold-water swimmers with ColdU, so the recommendations are extrapolated from cohort studies of patients with ColdU, from anaphylaxis guidelines and from the physiology of cold-water immersion. They are listed in Table 2 together with the type of evidence behind each; the text below explains only the points that need further comment.

Anyone who has had wheals, itching, swelling, dizziness or collapse during or after a cold-water swim should be referred to an allergist, even if a standard CST is negative [11,13]. Risk assessment and autoinjector prescription should then follow the criteria proposed by the COLD-CE investigators (see section 3.6) [8,35]. The CTT can be

compared with the temperature of the water in which the athlete trains and competes [29], bearing in mind that one patient reacted even in a pool at about 32 °C [30].

Competition rules also need to be considered. In open-water events governed by World Aquatics, the minimum water temperature is 16 °C [52]. Because 71% of patients in the CTT study had a threshold of 16 °C or higher and almost half had one of 20 °C or higher [29], many athletes with ColdU would race in water below their own threshold. Whether such an athlete competes should therefore be decided individually and not on the basis of the official temperature limit alone.

Wetsuits are compulsory in World Aquatics events below 18 °C [52], and many recreational swimmers use them as well. A wetsuit, neoprene cap, gloves and socks reduce the area of skin in contact with cold water, but their effect has not been studied in ColdU. A wetsuit also leaves the face and often the hands and feet exposed and should not be regarded as protection against anaphylaxis. In healthy volunteers, a partial-coverage (trunk and arms) wetsuit gave little protection against the initial cardiac and ventilatory responses to immersion in 5 °C water, whereas a drysuit protected significantly better [53].

Patients who have had ColdA or who show markers of severe disease should be advised against cold-water and ice swimming and against open-water sports that carry a real risk of an unplanned fall into the water. Diving calls for particular caution, because a reaction under water is especially dangerous and the skin lesions can be confused with decompression sickness. In the case cited above, the diver was not cleared to dive even though she was certified to use a drysuit [30].

Two adrenaline autoinjectors are useful only if they are within reach in the water, for instance with an accompanying kayaker or boat or in a waterproof pouch on a tow float, and companions should know how to use them [42,44]. The summary of product characteristics of a widely used autoinjector advises storage at no more than 25 °C and states that it must not be refrigerated or frozen [54]. On cold days the devices should therefore be kept close to the body or in an insulated pouch. Second-generation antihistamines can be taken regularly or as short-term prophylaxis before a planned swim, a choice that the current guideline leaves to individual decision in inducible urticaria [6,7,47].

General advice for cold-water swimmers applies to patients with ColdU in addition to the measures in Table 2. It includes avoiding sudden entry, swimming under supervision and, after an unexpected fall into the water, floating until the cold shock response subsides [3,33,55]. Safety staff at open-water events and winter swimming clubs should be able to tell anaphylaxis from an ordinary cold shock response, have adrenaline available and know how to lift a casualty out of the water safely [33,46,55].

Table 2. Practical recommendations for swimmers with cold urticaria and the type of evidence on which they are based

Recommendation	Main sources	Type of evidence
Refer anyone with symptoms during or after a cold-water swim for allergological work-up; do not stop at a negative standard CST	[11,13]	Cohort studies
Prescribe an autoinjector after previous ColdA, or when there is cold-induced angioedema, oropharyngeal/laryngeal	[8,35]	Criteria proposed on the basis of a cross-

Recommendation	Main sources	Type of evidence
symptoms or itchy earlobes; also consider systemic reactions to Hymenoptera stings		sectional study
Compare the CTT with the temperature of the training and competition water	[29,30,52]	Cohort data, case report, competition rules; extrapolation
Advise against cold-water and ice swimming after ColdA or with markers of severe disease	[8]	Expert opinion based on cohort data
Never swim alone, stay close to the shore and inform companions about the disease and anaphylaxis	[18]	Expert opinion; clinical practice report
Keep two adrenaline autoinjectors within reach, train companions in their use and protect the devices from freezing	[42,44,54]	Guideline; cross-sectional study; product information
Take sgAH at a dose that controls symptoms, regularly or before a planned swim	[7,47]	Guideline; small randomised trials with threshold outcomes only; no data on prevention of anaphylaxis
Consider a wetsuit and neoprene accessories, without regarding them as protection against anaphylaxis	[53]	Physiological study in healthy volunteers; expert opinion
Avoid swimming on windy days and minimise time in wet clothing	[15]	Retrospective cohort; expert opinion
Suspected anaphylaxis in water: horizontal extraction, lay the casualty flat, give intramuscular adrenaline without delay, protect from wind and transfer to hospital; observe the swimmer during the swim and after exit	[33,42,45]	Guideline; physiology review; case series
Competitive athletes: emergency use of adrenaline must not be delayed; apply for a retroactive TUE	[12,51]	Regulation

Source: own elaboration based on the cited studies.

Notes: CST, cold stimulation test; CTT, critical temperature threshold; ColdA, cold-induced anaphylaxis; sgAH, second-generation H1-antihistamines; TUE, therapeutic use exemption.

Because ColdU is chronic (see section 3.1), decisions about sport should be revisited from time to time, for instance before each season, with repeated CTT measurement and prospective ColdUAS monitoring [29,38]. In a

qualitative interview study, 11 of 13 adults and adolescents reported that the disease affected sport and exercise [10], and in one follow-up study 41% of patients restricted leisure and sporting activities such as swimming [18]. Counselling should aim at a shared decision that allows safe physical activity, in line with the guideline's call to balance trigger avoidance against quality of life [7].

4. Discussion

In swimmers with ColdU, the hazards of the disease and those of cold-water immersion combine. Mast cell activation over a large area of skin [6,23] coincides with cold shock, autonomic conflict, swim failure and circum-rescue collapse [3,32,33], and the two may mask or aggravate each other.

Existing reviews of ColdU warn that swimming can trigger anaphylaxis [6,23], but they concentrate on diagnosis and drug treatment. The literature on cold-water immersion, in turn, describes the physiological hazards in detail [3,31,33] without mentioning mast cell disease, and general advice for cold-water swimmers does not address ColdU [55]. Practical advice from clinicians, such as avoiding or limiting aquatic activities [20], never swimming alone, staying close to the shore and carrying an autoinjector [18], has been followed poorly, with only one of nine patients who were advised to carry an autoinjector actually doing so [18]. Education probably needs to be repeated and practical. By bringing the two bodies of evidence together and stating the evidence behind each recommendation (Table 2), this review tries to close the gap between the two fields, which matters most where competition rules allow racing in water as cold as 16 °C [52].

Several points remain unresolved. Data on cryoproteins conflict [19,25], possibly because of differences in laboratory methods, sample size and patient selection, and it is unclear why ColdA is less frequent in patients with concomitant chronic spontaneous urticaria [8]. Because standard tests are often negative and the atypical forms are heterogeneous [11,17], some patients at risk may never be diagnosed.

Evidence on treatment is limited to small trials with threshold outcomes [48,49] and to negative trials of dupilumab and rilonacept [27,28], and no study has tested whether any drug prevents anaphylaxis during immersion. Omalizumab is effective [22] but reaches few patients [9], and autoinjectors are underprescribed [44]. Meanwhile, cold-water immersion continues to gain popularity despite weak evidence of health benefits [2,3,4]. Other inducible urticarias raise similar questions in sport; in cholinergic urticaria, which is triggered by exercise and sweating, research in physically active people is likewise still needed [56].

4.1. Limitations

Our review has several limitations. It is narrative rather than systematic, so the choice of papers may be biased, and we did not formally assess study quality. Most data come from retrospective cohorts treated in referral centres, which may overstate disease severity, and much of the evidence concerns children [13,14,15]. The studies define anaphylaxis differently, which makes comparisons difficult. Some sources are research letters, case reports or editorials [1,22,24,29,30,35,37,39,44,45,55], and their conclusions carry less weight. The search was limited to one database (PubMed) and to publications in English, so relevant work indexed elsewhere, for instance in sport science databases, may have been missed. Much of the evidence on ColdU comes from a single international network of urticaria centres, which may limit the diversity of perspectives. Finally, no study has looked directly at

athletes or regular cold-water swimmers with ColdU, so the recommendations in section 3.7 rest on extrapolation and expert opinion.

Prospective work is needed in several areas: registries of reactions during cold-water sports, validation of the CTT as a predictor of risk at a given water temperature, and trials testing whether drug prophylaxis or protective equipment prevents systemic reactions during whole-body immersion. Training programmes for lifeguards, event organisers and winter swimming groups have not been evaluated either.

5. Conclusions

Complete immersion in cold water is the most frequent trigger of anaphylaxis in cold urticaria, and in water its early signs overlap with the physiological responses to immersion, which may delay recognition and lead to drowning. A negative standard cold stimulation test does not exclude this risk. Although intramuscular adrenaline is the first-line treatment, autoinjectors remain underprescribed, and drug treatment, which raises reaction thresholds, has not been shown to prevent anaphylaxis during immersion. Open-water competition rules also permit water temperatures below the critical temperature threshold of many patients. Swimmers with cold urticaria therefore need individual risk assessment, and prospective studies in this group are needed to turn extrapolated advice into evidence-based recommendations.

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Author contributions

Conceptualization: Dawid Mazurek

Methodology: Dawid Mazurek, Zuzanna Cecot, Filip Lenartowicz, Maciej Lesiczka

Software: Not applicable

Validation: Filip Lenartowicz, Anna Maciaszczyk

Investigation: Zuzanna Cecot, Alicja Lewczuk, Katarzyna Kuklińska

Resources: Katarzyna Kuklińska, Anna Maciaszczyk

Data curation: Alicja Lewczuk, Jarosław Kucharski

Writing – original draft: Dawid Mazurek, Zuzanna Cecot, Oliwia Gałczyńska, Alicja Lewczuk

Writing – review and editing: Maciej Lesiczka, Katarzyna Kuklińska, Julia Duszejko, Jarosław Kucharski, Filip Lenartowicz, Anna Maciaszczyk

Visualization: Alicja Lewczuk, Julia Duszejko

Supervision: Dawid Mazurek

Project administration: Dawid Mazurek

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References

1. Massey H, Eglin C, Tipton M. Making waves in cold-water immersion/swimming research: towards a balanced assessment of therapeutic efficacy and water safety. Lifestyle Med. 2026;7(1):e70046. https://doi.org/10.1002/lim2.70046

2. Knechtle B, Waśkiewicz Z, Sousa CV, Hill L, Nikolaidis PT. Cold water swimming – benefits and risks: a narrative review. Int J Environ Res Public Health. 2020;17(23):8984. https://doi.org/10.3390/ijerph17238984

3. Tipton MJ, Collier N, Massey H, Corbett J, Harper M. Cold water immersion: kill or cure? Exp Physiol. 2017;102(11):1335-1355. https://doi.org/10.1113/EP086283

4. Cain T, Brinsley J, Bennett H, Nelson M, Maher C, Singh B. Effects of cold-water immersion on health and wellbeing: a systematic review and meta-analysis. PLoS One. 2025;20(1):e0317615. https://doi.org/10.1371/journal.pone.0317615

5. National Water Safety Forum. WAID UK 2024 summary for the National Water Safety Forum [Internet]. Birmingham: Royal Society for the Prevention of Accidents; 2025 [cited 2026 Oct 16]. https://nationalwatersafety.org.uk/media/1444/waid-uk-2024-summary-may25-final.pdf

6. Maltseva N, Borzova E, Fomina D, Bizjak M, Terhorst-Molawi D, Košnik M, Kulthanan K, Meshkova R, Thomsen SF, Maurer M; COLD-CE Steering Committee. Cold urticaria – What we know and what we do not know. Allergy. 2021;76(4):1077-1094. https://doi.org/10.1111/all.14674

7. Zuberbier T, Ansari ZA, Abdul Latiff AH, Abuzakouk MM, Agcaoili-De Jesus MS, Agondi RC, Al-Ahmad M, Alangari AA, Alhameli H, Alonso Bello CD, Alshareef S, Al-Tamemi S, Altrichter S, Al Wahshi H, Aquilina S, Araújo M, Arnaout R, Asero R, Ballmer-Weber B, Bangert C, Bauer A, Ben-Shoshan M, Bernstein JA, Bindslev-Jensen C, Bizjak M, Boccon-Gibod I, Bonnekoh H, Bouillet L, Brockow K, Brzoza Z, Bulatović Ćalasan M, Bulkhi A, Buttgereit T, Bygum A, Caballero T, Calderon O, Campos R, Cancian M, Carne E, Castor MA, Cerecedo I, Çetinarslan T, Cherrez-Ojeda I, Chkhikvadze N, Chong-Neto HJ, Choo K, Christoff G, Chu CY, Ciupka K, Conlon N, Costa C, Craig TJ, Criado P, Danilycheva I, Darlenski R, De Arruda Chaves E, de Montjoye L, Doutre MS, Du-Thanh A, Ebo D, Elkhalifa S, Elmariah S, El-Shanawany T, Ensina LF, Ertaş R, Fachini Jardim Criado R, Ferrer M, Ferrucci S, Fok JS, Fomina D, Fonacier L, Fouda G, Francescantonio I, Fukunaga A, Galvan Calle CA, Garcia E, Gáspár K, Gelincik A, Geng S, Godse K, Gonçalo M, Gotua M, Grattan C, Grosber M, Guidos Fogelbach G, Guilarte M, Guillod R, Hamelmann E, Hawkes J, Hayama K, Heuer R, Hide M, Hoetzenecker W, Inomata N, Kang HR, Kaplan AP, Kapp A, Karam M, Kasperska-Zajac A, Katelaris CH, Kessel A, Khoshkhui M, Kim B, Kinaciyan T, Kocatürk E, Kolacinska-Flont M, Kolkhir P, Konstantinou GN, Košnik M, Krasowska D, Kulthanan K, Kumaran MS, Kuprys-Lipinska I, Labrador M, Larco JI, Larenas-Linnemann D, Latysheva E,

Lazaridou E, Li PH, Lima H, Lippert U, Magerl M, Makris M, Alves Marcelino J, Marzano AV, Medina I, Meshkova R, Micallef D, Mohammed Ali R, Mortz CG, Munoz M, Oude Elberink HNG, Nakonechna A, Nasr I, Nast A, Netchiporouk E, Nettis E, Nieto S, Ogueta Canales I, Okas TL, Orfali RL, Özkaya E, Parisi C, Pennitz A, Pawankar R, Pereira MP, Peter J, Petkova E, Pigatto PD, Podder I, Popov T, Porebski G, Pyatilova P, Ramon GD, Ratti Sisa HA, Recto MS, Ress K, Ridge K, Riedl M, Ritchie C, Rosario Filho N, Rosmaninho I, Rudenko M, Rukhadze M, Rutkowski K, Sabato V, Sahiner UM, Saini S, Saleh Al Sabbagh F, Salman A, Salvo F, Sanchez J, Santucci A, Schliemann S, Schmid-Grendelmeier P, Sekerel BE, Serpa F, Sheikh F, Sheikh J, Shendi H, Siebenhaar F, Sonomjamts M, Soria A, Sousa Pinto B, Staevska M, Staubach P, Stephan M, Stevanovic K, Stingeni L, Stobiecki M, Su Küçük Ö, Sussman G, Szegedi A, Takahagi S, Tanaka A, Teovska Mitrevska N, Thomsen SF, Toubi E, Tsatsou F, Turk M, Vadasz Z, Valerieva A, Valle S, Doorn MV, Veleiro Perez B, Vera Ayala CE, Vestergaard C, Vieira RJ, Maruta CW, Wedi B, Werner RN, Yap EWY, Xepapadaki P, Xiang YK, Ye YM, Yong P, Yosipovitch G, Zalewska-Janowska A, Zeyen C, Zhao Z, Metz M, Giménez-Arnau AM. The international guideline for the definition, classification, diagnosis and management of urticaria. Allergy. 2026;81(8):2582-2632. https://doi.org/10.1111/all.70210

8. Bizjak M, Košnik M, Dinevski D, Thomsen SF, Fomina D, Borzova E, Kulthanan K, Meshkova R, Ahsan DM, Al-Ahmad M, Altrichter S, Bauer A, Brockstädt M, Costa C, Demir S, Fachini Criado R, Ensina LF, Gelincik A, Giménez-Arnau AM, Gonçalo M, Gotua M, Holm JG, Inomata N, Kasperska-Zajac A, Khoshkhui M, Klyucharova A, Kocatürk E, Lu R, Makris M, Maltseva N, Miljković J, Pasali M, Paulino M, Pesqué D, Peter J, Ramón GD, Ritchie C, Rodrigues Valle SO, Rudenko M, Sikora A, de Souza Lima EM, Wagner N, Xepapadaki P, Xue X, Zhao Z, Terhorst-Molawi D, Maurer M. Risk factors for systemic reactions in typical cold urticaria: results from the COLD-CE study. Allergy. 2022;77(7):2185-2199. https://doi.org/10.1111/all.15194

9. Prosty C, Gabrielli S, Le M, Ensina LF, Zhang X, Netchiporouk E, Ben-Shoshan M. Prevalence, management, and anaphylaxis risk of cold urticaria: a systematic review and meta-analysis. J Allergy Clin Immunol Pract. 2022;10(2):586-596.e4. https://doi.org/10.1016/j.jaip.2021.10.012

10. Alladin A, Guillemin I, Chuang CC, Chao J, Krol M, Zikos E, Martincova R, Brookes E. A qualitative interview study exploring the lived experiences of adults, adolescents, and children with chronic inducible cold urticaria. J Patient Rep Outcomes. 2026;10:5. https://doi.org/10.1186/s41687-025-00970-6

11. Terhorst-Molawi D, Ahsan DM, Fomina D, Salameh P, Altrichter S, Andrenova G, Bauer A, Borzova E, Dinevski D, Giménez-Arnau AM, Gonçalo M, Gutsche A, Holm JG, Khoshkhui M, Klyucharova A, Kocatürk E, Košnik M, Kulthanan K, Makris M, Meshkova R, Metz M, Pasali M, Pesqué D, Peter JG, Thomsen SF, Xepapadaki P, Zuberbier T, Bizjak-Suran M. Cold urticaria with negative standard cold stimulation testing: results from the COLD-CE study. J Allergy Clin Immunol Pract. 2026;14(10):2397-2407.e4. https://doi.org/10.1016/j.jaip.2026.08.022

12. World Anti-Doping Agency. World Anti-Doping Code International Standard: Prohibited List 2026. Montreal: WADA; 2025 [cited 2026 Oct 12]. https://www.wada-ama.org/en/prohibited-list

13. Yee CSK, El Khoury K, Albuhairi S, Broyles A, Schneider L, Rachid R. Acquired cold-induced urticaria in pediatric patients: a 22-year experience in a tertiary care center (1996-2017). J Allergy Clin Immunol Pract. 2019;7(3):1024-1031.e3. https://doi.org/10.1016/j.jaip.2018.10.025

14. Prosty C, Gabrielli S, Mule P, Le Gallee M, Miles LM, Le M, Zhang X, Ensina LF, Netchiporouk E, Ben-Shoshan M. Cold urticaria in a pediatric cohort: clinical characteristics, management, and natural history. Pediatr Allergy Immunol. 2022;33(3):e13751. https://doi.org/10.1111/pai.13751

15. Karabag Citlak H, Azkur D, Kavas Yildiz Y, Demirel AC, Kot H, Vezir E, Kilic M, Usta Guc B, Kilic M, Yakici N, Kocabas CN, Dibek Misirlioglu E, Civelek E, Orhan F. Cold-induced urticaria in children: a multicenter, retrospective cohort study. Allergy Asthma Proc. 2023;44(6):e36-e43. https://doi.org/10.2500/aap.2023.44.230050

16. Diaz VL, Gribbons KB, Yazdi-Nejad K, Kuemmerle-Deschner J, Wanderer AA, Broderick L, Hoffman HM. Cold urticaria syndromes: diagnosis and management. J Allergy Clin Immunol Pract. 2023;11(8):2275-2285. https://doi.org/10.1016/j.jaip.2023.05.040

17. Ahsan DM, Elieh-Ali-Komi D, Pereira MP, Sürmeli S, Bizjak M, Brockstaedt M, Metz M, Altrichter S, Terhorst-Molawi D. Subtypes of atypical cold urticaria and recommendations for their diagnostic workup. J Allergy Clin Immunol Pract. 2025;13(9):2370-2380.e2. https://doi.org/10.1016/j.jaip.2025.07.019

18. Jain SV, Mullins RJ. Cold urticaria: a 20-year follow-up study. J Eur Acad Dermatol Venereol. 2016;30(12):2066-2071. https://doi.org/10.1111/jdv.13841

19. Ginter K, Ahsan DM, Bizjak M, Krause K, Maurer M, Altrichter S, Terhorst-Molawi D. Cryoglobulins, cryofibrinogens, and cold agglutinins in cold urticaria: literature review, retrospective patient analysis, and observational study in 49 patients. Front Immunol. 2021;12:675451. https://doi.org/10.3389/fimmu.2021.675451

20. Deza G, Brasileiro A, Bertolín-Colilla M, Curto-Barredo L, Pujol RM, Giménez-Arnau AM. Acquired cold urticaria: clinical features, particular phenotypes, and disease course in a tertiary care center cohort. J Am Acad Dermatol. 2016;75(5):918-924.e2. https://doi.org/10.1016/j.jaad.2016.06.017

21. Bizjak-Suran M, Fležar M, Pereira MP. Pathophysiology and emerging treatments for dermographic, cholinergic and cold urticaria. J Eur Acad Dermatol Venereol. 2026. Online ahead of print. PMID: 42299718. https://doi.org/10.1111/jdv.70559

22. Metz M, Schütz A, Weller K, Gorczyza M, Zimmer S, Staubach P, Merk HF, Maurer M. Omalizumab is effective in cold urticaria – results of a randomized placebo-controlled trial. J Allergy Clin Immunol. 2017;140(3):864-867.e5. https://doi.org/10.1016/j.jaci.2017.01.043

23. Bizjak M. Cold urticaria: from wheals to anaphylaxis. Allergy Asthma Immunol Res. 2025;17(5):547-562. https://doi.org/10.4168/aair.2025.17.5.547

24. Bizjak M, Maurer M, Košnik M, Terhorst-Molawi D, Zver S, Burmeister T, Siebenhaar F. Severe cold urticaria can point to an underlying clonal mast cell disorder. Allergy. 2021;76(8):2609-2613. https://doi.org/10.1111/all.14844

25. Bizjak M, Košnik M, Terhorst-Molawi D, Dinevski D, Maurer M. Cold agglutinins and cryoglobulins associate with clinical and laboratory parameters of cold urticaria. Front Immunol. 2021;12:665491. https://doi.org/10.3389/fimmu.2021.665491

26. Deza G, Mensa-Vilaró A, March-Rodriguez A, Sánchez S, Pujol RM, Aróstegui JI, Giménez-Arnau AM. Acquired cold urticaria vs. autoinflammatory diseases, genetic and clinical profile and differential diagnosis: study

of a cohort of patients in a tertiary reference centre. Acta Derm Venereol. 2019;99(12):1071-1077. https://doi.org/10.2340/00015555-3292

27. Bonnekoh H, Butze M, Spittler S, Staubach P, Weller K, Scheffel J, Maurer M, Krause K. Inhibition of interleukin-1 with rilonacept is not effective in cold urticaria – results of a randomized, placebo-controlled study. Clin Transl Allergy. 2023;13(3):e12226. https://doi.org/10.1002/clt2.12226

28. Metz M, Saini SS, Yagami A, Gagnon R, Giménez-Arnau AM, Luo XD, Robinson LB, Martinčová R, Radin A, Makhija M. Dupilumab in patients with cold urticaria: results from a phase 3 trial. J Allergy Clin Immunol Glob. 2026;5(3):100673. https://doi.org/10.1016/j.jacig.2026.100673

29. Mlynek A, Magerl M, Siebenhaar F, Weller K, Vieira dos Santos R, Zuberbier T, Zalewska-Janowska A, Maurer M. Results and relevance of critical temperature threshold testing in patients with acquired cold urticaria. Br J Dermatol. 2010;162(1):198-200. https://doi.org/10.1111/j.1365-2133.2009.09441.x

30. Popa DA, Winn AR, Masters TC, Logue C. Cold urticaria preventing clearance for scientific diving. Undersea Hyperb Med. 2025;52(4):621-625. PMID: 41429040. https://pubmed.ncbi.nlm.nih.gov/41429040/

31. Bierens JJ, Lunetta P, Tipton M, Warner DS. Physiology of drowning: a review. Physiology (Bethesda). 2016;31(2):147-166. https://doi.org/10.1152/physiol.00002.2015

32. Shattock MJ, Tipton MJ. 'Autonomic conflict': a different way to die during cold water immersion? J Physiol. 2012;590(14):3219-3230. https://doi.org/10.1113/jphysiol.2012.229864

33. Leuci L, Melau J, Messina A, Tipton M, Carenzo L. Beyond hypothermia: mechanisms of death, rescue, and prevention in cold water immersion. J Appl Physiol. 2026;141(3):760-769. https://doi.org/10.1152/japplphysiol.00578.2026

34. Kulthanan K, Tuchinda P, Chularojanamontri L, Kiratiwongwan R. Cold urticaria: clinical features and natural course in a tropical country. Allergy Asthma Immunol Res. 2019;11(4):538-547. https://doi.org/10.4168/aair.2019.11.4.538

35. Bizjak M, Fomina D, Peter J, Giménez-Arnau AM, Pesqué D, Gonçalo M, Aarestrup FM, Ahsan DM, Al-Ahmad M, Altrichter S, Andrenova G, Bauer A, Borzova E, Costa C, Fachini Criado R, Demir S, Dinevski D, Ensina LF, Gelincik A, Gotua M, Holm JG, Inomata N, Khoshkhui M, Klyucharova A, Kocatürk E, Košnik M, Kulthanan K, de Souza Lima EM, Lu R, Makris M, Meshkova R, Pasali M, Paulino M, Ramón GD, Ritchie C, Rudenko M, Thomsen SF, Rodrigues Valle SO, Wagner N, Xepapadaki P, Xue X, Kasperska-Zajac A, Zhao Z, Terhorst-Molawi D. Mucosal angioedema involving the oropharynx signals severe cold urticaria: COLD-CE study insights. J Eur Acad Dermatol Venereol. 2026;40(1):e65-e70. https://doi.org/10.1111/jdv.20845

36. Magerl M, Altrichter S, Borzova E, Giménez-Arnau A, Grattan CEH, Lawlor F, Mathelier-Fusade P, Meshkova RY, Zuberbier T, Metz M, Maurer M. The definition, diagnostic testing, and management of chronic inducible urticarias – the EAACI/GA²LEN/EDF/UNEV consensus recommendations 2016 update and revision. Allergy. 2016;71(6):780-802. https://doi.org/10.1111/all.12884

37. Magerl M, Abajian M, Krause K, Altrichter S, Siebenhaar F, Church MK. An improved Peltier effect-based instrument for critical temperature threshold measurement in cold- and heat-induced urticaria. J Eur Acad Dermatol Venereol. 2015;29(10):2043-2045. https://doi.org/10.1111/jdv.12739

38. Ahsan DM, Altrichter S, Gutsche A, Bernstein JA, Altunergil T, Brockstaedt M, Maurer M, Weller K, Terhorst-Molawi D. Development of the Cold Urticaria Activity Score. Allergy. 2022;77(8):2509-2519. https://doi.org/10.1111/all.15310

39. Proença M, Marcelino J, Vieira J, Tomaz E. Cold urticaria patients can be susceptible to anaphylaxis during diagnostic cold provocation tests. BMJ Case Rep. 2025;18(6):e261489. https://doi.org/10.1136/bcr-2024-261489

40. Feldweg AM. Food-dependent, exercise-induced anaphylaxis: diagnosis and management in the outpatient setting. J Allergy Clin Immunol Pract. 2017;5(2):283-288. https://doi.org/10.1016/j.jaip.2016.11.022

41. Moon RE, Martina SD, Peacher DF, Kraus WE. Deaths in triathletes: immersion pulmonary oedema as a possible cause. BMJ Open Sport Exerc Med. 2016;2(1):e000146. https://doi.org/10.1136/bmjsem-2016-000146

42. Muraro A, Worm M, Alviani C, Cardona V, DunnGalvin A, Garvey LH, Riggioni C, de Silva D, Angier E, Arasi S, Bellou A, Beyer K, Bijlhout D, Bilò MB, Bindslev-Jensen C, Brockow K, Fernandez-Rivas M, Halken S, Jensen B, Khaleva E, Michaelis LJ, Oude Elberink HNG, Regent L, Sanchez A, Vlieg-Boerstra BJ, Roberts G. EAACI guidelines: anaphylaxis (2021 update). Allergy. 2022;77(2):357-377. https://doi.org/10.1111/all.15032

43. Cardona V, Ansotegui IJ, Ebisawa M, El-Gamal Y, Fernandez Rivas M, Fineman S, Geller M, Gonzalez-Estrada A, Greenberger PA, Sanchez Borges M, Senna G, Sheikh A, Tanno LK, Thong BY, Turner PJ, Worm M. World Allergy Organization anaphylaxis guidance 2020. World Allergy Organ J. 2020;13(10):100472. https://doi.org/10.1016/j.waojou.2020.100472

44. Bizjak M, Košnik M, Dinevski D, Thomsen SF, Fomina D, Borzova E, Kulthanan K, Meshkova R, Aarestrup FM, Ahsan DM, Al-Ahmad M, Altrichter S, Bauer A, Brockstädt M, Costa C, Demir S, Criado RF, Ensina LF, Gelincik A, Giménez-Arnau AM, Gonçalo M, Gotua M, Holm JG, Inomata N, Kasperska-Zajac A, Khoshkhui M, Klyucharova A, Kocatürk E, Lu R, Makris M, Maltseva N, Pasali M, Paulino M, Pesqué D, Peter J, Ramón GD, Ritchie C, Rodrigues Valle SO, Rudenko M, Sikora A, Wagner N, Xepapadaki P, Xue X, Zhao Z, Terhorst-Molawi D, Maurer M. Adrenaline autoinjector is underprescribed in typical cold urticaria patients. Allergy. 2022;77(7):2224-2229. https://doi.org/10.1111/all.15274

45. Pumphrey RS. Fatal posture in anaphylactic shock. J Allergy Clin Immunol. 2003;112(2):451-452. https://doi.org/10.1067/mai.2003.1614

46. Lott C, Karageorgos V, Abelairas-Gomez C, Alfonzo A, Bierens J, Cantellow S, Debaty G, Einav S, Fischer M, González-Salvado V, Greif R, Metelmann B, Metelmann C, Meyer T, Paal P, Peran D, Scapigliati A, Spartinou A, Thies K, Truhlar A, Deakin CD. European Resuscitation Council Guidelines 2025 Special Circumstances in Resuscitation. Resuscitation. 2025;215(Suppl 1):110753. https://doi.org/10.1016/j.resuscitation.2025.110753

47. Kulthanan K, Hunnangkul S, Tuchinda P, Chularojanamontri L, Weerasubpong P, Subchookul C, Maurer M. Treatments of cold urticaria: a systematic review. J Allergy Clin Immunol. 2019;143(4):1311-1331. https://doi.org/10.1016/j.jaci.2019.02.005

48. Siebenhaar F, Degener F, Zuberbier T, Martus P, Maurer M. High-dose desloratadine decreases wheal volume and improves cold provocation thresholds compared with standard-dose treatment in patients with acquired cold urticaria. J Allergy Clin Immunol. 2009;123(3):672-679. https://doi.org/10.1016/j.jaci.2008.12.008

49. Abajian M, Curto-Barredo L, Krause K, Santamaría E, Izquierdo I, Church MK, Maurer M, Giménez-Arnau A. Rupatadine 20 mg and 40 mg are effective in reducing the symptoms of chronic cold urticaria. Acta Derm Venereol. 2016;96(1):56-59. https://doi.org/10.2340/00015555-2150

50. Terhorst-Molawi D, Hawro T, Grekowitz E, Kiefer L, Merchant K, Alvarado D, Thomas LJ, Hawthorne T, Crowley E, Heath-Chiozzi M, Metz M, Maurer M. Anti-KIT antibody, barzolvolimab, reduces skin mast cells and disease activity in chronic inducible urticaria. Allergy. 2023;78(5):1269-1279. https://doi.org/10.1111/all.15585

51. World Anti-Doping Agency. World Anti-Doping Code International Standard: Therapeutic Use Exemptions 2023. Montreal: WADA; 2022 [cited 2026 Oct 12]. https://www.wada-ama.org/en/resources/world-anti-doping-code-and-international-standards/international-standard-therapeutic-use

52. World Aquatics. Science supports safety: new research reinforces 16°C World Aquatics threshold for open water swimming [Internet]. Lausanne: World Aquatics; 2026 Apr 2 [cited 2026 Oct 16]. https://www.worldaquatics.com/news/4479872/science-supports-safety-new-research-reinforces-16c-world-aquatics-threshold-open-water-swimming

53. Tipton MJ, Vincent MJ. Protection provided against the initial responses to cold immersion by a partial coverage wet suit. Aviat Space Environ Med. 1989;60(8):769-773. PMID: 2775133.

54. Viatris Products Limited. EpiPen Adrenaline (Epinephrine) 0.3 mg Auto-Injector: summary of product characteristics. Potters Bar: Viatris; 2026 [updated 2026 Aug 10; cited 2026 Oct 12]. https://www.medicines.org.uk/emc/product/4289/smpc

55. Tipton M, Massey H, Mayhew A, Morgan P. Cold water therapies: minimising risks. Br J Sports Med. 2022;56(23):1332-1334. https://doi.org/10.1136/bjsports-2022-105953

56. Chmiela M, Baran WP, Świątko A, Lachowska E, Dembowska J, Bartkiewicz PA. Cholinergic urticaria – clinical significance in athletes and physically active individuals: a narrative review. Qual Sport. 2026;60:72740. https://doi.org/10.12775/QS.2026.60.72740