Aquatic Skin Diseases from Chemical and Physical Causes

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Apart from the diseases due to biotic noxae described up to now, there are various other dermatological conditions connected in some way with salt- or freshwater contact or with aquatic activities (Table 13.1) [1, 2]. No analysis will here be made of clinical forms favored, induced or aggravated by exposure to the sun, that is of course inevitable in subjects involved in aquatic activities for long and even short periods, nor of those caused by non aquatic biotic agents.

13.1 Aquagenic Urticaria

This form of urticaria, first described by Shelley and Rawnsley in 1964 [3], is induced simply by skin contact with the water, regardless of its physical and chemical properties (source, salt content, temperature) [4–11]. The affliction is often misdiagnosed. The onset is generally observed in young adults, with a mean age of 18 years, and is five-fold more frequent in the female sex [7, 12–14]. Sometimes, several members of the same family are affected [3, 15–17]. There are no data available on the evolution and duration of the disease, although many patients have referred a very long course, even 20 years.

The onset of the urticarial affection occurs 3–10 min after any type of skin contact with water; it reaches a peak in about 30 min and dies down again after a further

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Table 13.1 Some skin diseases of aquatic origin

General
Sunburn
Aquagenic urticaria
Aquagenic pruritus
Cold urticaria
Contact dermatitis
Swimming costume
Diving equipment
Dry skin (swimmer's xerosis)
Aquagenic skin wrinkling
Seawater
Immersion syndrome
Abrasive effect
Surfer's nodules
Otitis externa
Aquagenic urticaria
Freshwater
Swimming pools
Mycosis
Verrucae
Chlorine irritation
Chapping in atopic subjects
Aquagenic acne
Greenish hair tinge
Hair bleaching
Chemical conjunctivitis
Otitis externa
Swimming pool granuloma
Jacuzzi/hot tubs
Folliculitis induced by *Pseudomonas*
Sauna
Miliaria
Tinea versicolor
Shower
Aquagenic pruritus
Sub-aqua activities
Otitis externa
Intertrigo
Staphylococcal infections
Burns
Linear abrasions from wetsuit folds
Pruritus and erythema from decompression
"Napkin rash" type dermatitis

30–60 min. The exposed skin sites are generally refractory to stimuli for several hours. The raised lesions are not clinically distinguishable, as regards shape and distribution, from those of cholinergic urticaria (with which they can coexist), being punctiform, with a diameter of 2–3 mm, surrounding the hair follicles (Fig. 13.1). These small wheals, that are intensely itchy, appear on the areas in contact with the water and if the whole body is exposed, mainly on the neck and trunk, and to a lesser

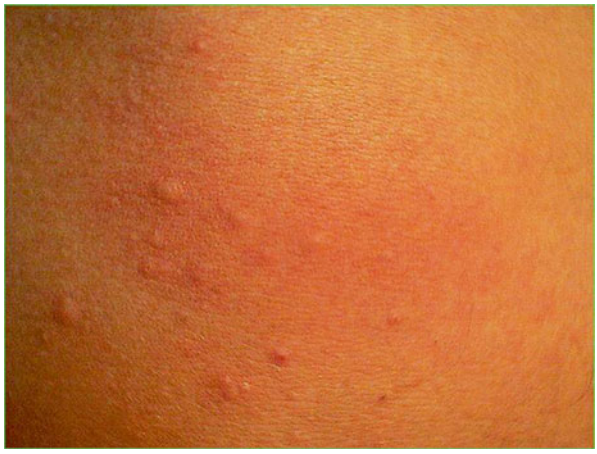

Fig. 13.1 Aquagenic urticaria: erythemato-oedematous punctiform wheals

degree, the shoulders and sides. The palms of the hands and soles of the feet are not affected. Alcohol and other organic solvents applied to the skin do not cause wheal formation [17]. Systemic reactions are rarely reported [18, 19].

The pathogenic mechanism is unknown. Several mechanisms seem most likely to be implicated. An interaction of water with a component within or on the stratum corneum or sebum, that may generate a toxic compound, has been suggested: absorption of this substance would lead to degranulation of the perifollicular mast cells and histamine release [3]. However, removal of the stratum corneum seems to aggravate the reaction, while pretreatment with organic solvents enhances wheal formation in contact with water [6]. Czarnetzki et al. have hypothesized the existence of a water-soluble antigen at the epidermal layer that, when absorbed in the derma, causes the release of histamine by mast cells [7]. Other authors have recently reported that 5 % saline was more effective than distilled water in eliciting the urticarial eruption. They proposed that the salt concentration and/or water osmolarity may influence the pathogenic mechanism of aquagenic urticaria, possibly by enhancing the solubilization and penetration of a hypothetical epidermal antigen, in the same way as has been postulated for the enhancement of organic solvents [20]. As regards the ionic concentration of water, Gallo and Coll. reported six young women with urticarial rashes triggered mostly by sea bathing, characteristically localized on the inferior facial contours and neck. The patients underwent challenge tests with tap water and with a hypertonic NaCl water solution (3.5 %, iso-osmolar with seawater) at room temperature, by means of applying soaked compresses for 20 min to the mandibular region or neck, and a positive response was elicited [21, 22]. The authors concluded that what they observed may be a distinct subtype of aquagenic urticaria with a peculiar, sometimes exclusive, localization [21, 22]. Another proposed chemical mediator in aquagenic urticaria is acetylcholine, in view of the ability of the acetylcholine antagonist, scopolamine, to suppress wheal formation when applied to the skin before water contact [6]. Methacholine injection testing is negative in patients with aquagenic urticaria, while it is often positive in cholinergic urticaria [16]. Serum histamine levels will differ from case to case [16].

Table 13.2 Provocation tests of some types of physical urticaria

Physical urticaria	Diagnostic test
Aquagenic urticaria	Wet compresses (35 °C) at room temperature to the upper body for 30 min
Cold urticaria	An ice-cube in a thin plastic bag applied to the forearm for 5–10 min
Cholinergic urticaria	Exercise test until sweating Warm bath (43 °C)
Heat urticaria	Heated cylinder (50–55 °C) applied to the upper trunk for 30 min

Table 13.3 Skin reactions directly (*) or indirectly (°) (pseudo-aquagenic reactions) due to contact with water

Reaction	Stimulus
Aquagenic pruritus (*)	Water
Aquagenic urticaria (*)	Water
Cholinergic urticaria (°)	Bathing or swimming in hot water
Acquired cold urticaria (°)	Cold water
Localized heat urticaria (°)	Hot water
Symptomatic dermographism (°)	Shower jet, towel friction
Polycythaemia rubra vera (*)	Water

The standard diagnostic test is application of 35 °C water compresses to the patient's chest for 20–40 min. It is best to use a thermometer on the site of the test to make sure of the correct temperature, in order to be able to differentiate between aquagenic, cholinergic, cold and heat urticaria (Table 13.2). A possible association with dermographism, another type of physical urticaria, should be taken into account. If only itching develops, without skin lesions, then a diagnosis of aquagenic pruritus can be made.

Differential diagnosis must also be made with other complaints induced by water, even if not always directly (Table 13.3). Apart from aquagenic urticaria, other cases of urticaria from physical causes develop only after contact with certain types of water, which is not the true cause. Pre-treatment of the skin with vaseline or lanolin prevents the eruption, perhaps by preventing the formation of the causal molecule [23].

The treatment of choice is antihistamines, although variable responses are obtained [16, 19]. Refractory cases can treated with ultraviolet (UV) radiation (psoralen plus UVA and UVB therapy), either alone or in combination with antihistamines. It has been hypothesized that the effect of UV therapy is mediated by inducing a thickening of the epidermis thereby preventing water penetration, or by interaction with dendritic cells, or a decreased mast cell response [16, 24].

13.2 Cold Urticaria

This consists of the appearance of pomphoid lesions on skin or mucosal sites after contact with cold objects, water, or air, or after the ingestion of cold drinks or foods. Among the forms of physical urticaria, cold urticaria can present in various guises: familial or acquired, immediate or delayed, localized or systemic, primitive or

Table 13.4 The various forms of cold urticaria

1. Familial
Localized (delayed, after 9–18 h)
Systemic (immediate)
2. Acquired
Localized
Immediate
Delayed
Cholinergic cold dermographism
Due to localized reflex
Systemic
Due to cold
Due to generalized reflex
Cholinergic cold urticaria
Idiopathic
Secondary
Viral infections (respiratory virosis, HIV, infectious mononucleosis)
Bacterial infections (borreliosis, hepatitis, *Helicobacter pylori* colonization, acute toxoplasmosis)
Insect stings
Drug intolerance or allergy (penicillin, griseofulvin, oral anticoagulants, diazepam, alprazolam)
Atopy
Coeliac disease
Diseases featuring cryoglobulins, cryofibrinogen or haemolysins due to the cold
Autoimmune diseases (connectivitis, thyroiditis, erythema nodosum)
Cold paroxystic haemoglobinuria
Myeloproliferative diseases (myeloma)

secondary (Table 13.4). The incidence ranges from 1 to 7 % of all physical forms, depending on the various case series [8–10].

The acquired idiopathic form is the most common and the familial form the rarest. Rare forms of atypical contact urticaria also include delayed cold urticaria, where localized whealing appears 12–48 h after the cold exposure, and cold cholinergic forms (in which punctiform pomphoid lesions, 1–2 mm wide, develop after physical exercise in a cold environment, whereas no clinical lesions appear after physical exercise in a warm environment or after exposure to a cold environment but no physical exercise). Another rare form is cholinergic cold dermographism (linear pomphoid lesions appear if the patient is exposed to the cold during or immediately after applying appropriate stimulation; exposure to the cold without such stimulation, or alternatively the application of stimulation in a warm environment, do not elicit any response) (Table 13.5).

Acquired cold urticaria may be secondary to various conditions: viral infections (mononucleosis), infections such as HIV, syphilis, hepatitis, parasites and bacterial infections. In addition, cryoglobulinemia with or without malignancies, reactions to insects, intolerance or allergy to drugs (penicillin, griseofulvin, diazepam, alprazolam),

Table 13.5 Differential diagnosis of the various types of cold urticaria

Clinical forms	Diagnostic
Acquired cold urticarial	A melting ice cube in a thin plastic bag
Atypical acquired cold urticaria	Negative immediate contact stimulation test
Delayed cold urticaria	Delayed urticarial lesions up to 24 h after testing
Cold-dependent dermographism	Urticarial lesions after stroking pre-cooled skin
Cold-induced cholinergic urticaria	Urticarial symptoms by exercise in cold environments
Hereditary subtipes of cold urticaria	
Delayed cold urticaria	Negative immediate contact stimulation test; delayed urticarial lesions after 9–18 h; the lesions typically resolve into hyperpigmentation
Familial cold auto-inflammatory syndrome	Episodic urticarial-like lesions associated with conjunctival injection, fever and other systemic inflammatory symptoms; often delayed lesions (1–2 h)

Modified by Magerl and Coll. [25]

coeliac disease, hypothyroidism, and leukocytoclasic vasculitis may contribute to a similar clinical presentation [26–29].

The onset of acquired idiopathic cold urticaria can occur at any age but it particularly affects young adults, especially the female sex. The classic picture is the development of pomphoid lesions in sites exposed to contact with cold objects, foods, air conditioning or to sudden changes of temperature. Uncovered skin zones seem to be much more sensitive to stimuli. The skin wheals usually develop within a few minutes and persist for 1–2 h. The oral mucosa and tongue may also be affected. In more severe cases with diffuse manifestations, systemic symptoms such as weakness, breathlessness, headache, tachycardia and vertigo can develop. Sometimes even shock symptoms can develop immediately after swimming and it is very important to warn patients suffering from cold urticaria of the dangers of swimming [28, 30–34].

Pathogenic considerations should be made bearing in mind that cold urticaria can also be passively transferred to healthy subjects by means of the Prausnitz-Küstner test; this passive sensitisation has to do with serum IgE, and sometimes IgM, IgG or IgA [35]. It has been suggested that subjects with cold urticaria develop autoantibodies of IgE type, but also IgG, against skin antigens prevalently associated with the skin mast-cells. Anti-nuclear serum autoantibodies (acting against the B laminar fraction) have occasionally been demonstrated [36]. The efficacy of antibiotic treatment, demonstrated in a high percentage of patients, has recently given rise to the suggestion that acquired forms may have a microbial origin, possibly of a subclinical nature [37].

Histamine, a chemotactic neutrophilic factor, and some chemotactic eosinophilic factors, are among the main mediators of cold urticaria. In the acquired idiopathic form, tests of immersion in cold water have, instead, elicited a decreased chemotactic neutrophilic index; this event, described as "the granulocytic inactivation phenomenon" seems to be highly specific and is not present either in chronic urticaria or in other forms of cold urticaria [9]. Quinines and some derivatives of arachidonic acid (PGD2, LTE4) sometimes appear to be increased; they may amplify the biological effect of other mediators [38–41].

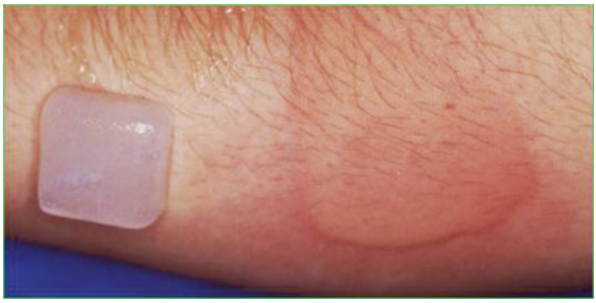

Fig. 13.2 Itchy skin wheal on the site of ice cube test

The observation that topical capsaicin, an antagonist of substance P released at the level of the nerve terminals, can inhibit local reactions to contact with the cold implies that this neuropeptide may play an important role in triggering the complaint. On the basis of these data, a defective thermal and/or vasomotor regulation of central origin may be postulated [9]. During severe episodes of cold urticaria, associated with systemic symptoms, high serum levels of TNF-α, a powerful pro-inflammatory cytokine, have been demonstrated [42].

Histologically, the inflammatory dermal infiltrate features two different cellular patterns, one with a predominance of neutrophils and the other of lymphocytes. This is likely the result of two different stages of the same process: the neutrophils may prevail at first, and then the lymphocytes take over [43].

Cold urticaria can be associated with chronic urticaria or other urticarial forms with physical causes, especially dermographism and cholinergic urticaria. The diagnosis is often confirmed by the ice-cube test: a melting ice cube in a thin plastic bag (to avoid cold damage to the skin). When applied to the flexural surface of the forearm for 5–10 min, an itchy skin wheal will develop on the site of the test, that should be assessed 10 min after removing the ice cube (Fig. 13.2).

A positive ice cube test confirms the presence of cold urticaria and should prompt further tests to determine individual temperature and/or stimulation time thresholds [25, 28, 29, 44]. For this purpose, a Peltier element-based electronic provocation device (TemptTest®, Emo Systems GmbH, Berlin, Germany) has been designed, that allows simultaneous skin exposure to 12 different temperatures ranging from 4 to 42 °C, in a standardized and reproducible manner [45]. Critical temperature threshold tests enable patients to be better instructed on how to avoid situations that will cause them urticaria. They also gauge how effectively patients are protected by therapy, and allow individualized treatment optimization [46]. Laboratory tests are of limited value in most cases of cold urticaria [47]: therefore additional tests should be limited to those required to exclude underlying diseases and identify associated diseases suggested by the clinical history. The ice cube test is negative in familial and cholinergic cold urticaria forms (Table 13.4).

The clinical course of acquired idiopathic cold urticaria is long, ranging from 2 to 10 years.

As regards treatment approaches, the patient must first of all be carefully informed about all the possible triggering factors and especially the danger of

swimming in cold water (in any case, such patients must never swim unaccompanied). Threshold testing (e.g. using TempTest ®) can help patients to recognize and control cold exposure in their daily life [20]. Treatment with antihistamines is the most common and most efficacious symptomatic therapy [48, 49]. In many patients, however, very high dosage antihistamines, up to four times the daily recommended dose, are needed to obtain a satisfactory response [49, 50]. In such cases unsuccessful treatment can severely harm the patient's quality of life, and indeed these patients are also at risk of developing life-threatening complications, including suffocation from pharyngeal angioedema induced by cold foods or beverages, and drowning after experiencing shock-like symptoms during aquatic activities. For these reasons it is essential to establish an optimal antihistamine treatment regimen for each patient. Other therapeutic options in severe cases or those with a poor response to antihistamines include leukotriene antagonists [51], cyclosporin [52], anti-IgE [53], and corticosteroids [54]. Successful treatment has recently been reported with some TNF-alpha inhibitors, such as etanercept [55] and omalizumab [56]. Other drugs, such as cyproheptadine, ketotifen, oral cromoglycate and H2-blockers, are not effective [57].

Patients suffering from severe forms of cold urticaria (at risk of oropharyngeal edema or shock-like reactions) must carry an emergency medication kit containing corticosteroids, antihistamines and epinephrine (adrenaline) injector, and be properly instructed as to how to use it.

In some cases antibiotic treatment may have to be considered, as this has sometimes proven useful even in the absence of a manifest infection. This will consist of high doses of penicillin (e.g. oral phenoxymethylpenicillin 1 MU/day for 2–4 weeks, or intramuscular benzylpenicillin 1 MU/day for 20 days) and tetracyclines over 2–4 weeks (e.g. doxycycline 200 mg/day for 3 weeks) [44, 58].

Patients must also protect themselves against cold air by wearing appropriate clothing, including gloves and woolen socks. It may be possible to induce tolerance (hardening) to the cold [59]. Clearly, the induction of tolerance must be done very cautiously at the beginning, under supervision, because of the risk of systemic reactions, and the patient should be hospitalized. A very good patient compliance is needed (involving as it does cold showers several times daily on an increasing body surface and at decreasing temperatures; the initial temperature of the cold water must be 5 °C above provocation), and the patient should know that discontinuation will cause the re-presentation of the symptoms [59]. Treatment with topical capsaicin, the principle ingredient of chili peppers, has been shown to prevent symptoms [59]. Capsaicin induces a depletion of neuropeptides from sensory nerve fibers, that might contribute to the onset of the symptoms, although a pathogenic role in cold urticaria has still to clarified [60].

Familial cold urticaria has a dominant autosomal transmission. Pomphoid lesions appear, associated with burning rather than itching, between 30 min and 3 h after exposure to cold winds. Atmospheric cold is a typical trigger, but handling cold objects or ingesting cold food or beverages can also bring on symptoms. The ice-cube test is negative. General symptoms often develop: shivering, fever, muscle and joint pain, headache. The symptoms are present

from birth and persist throughout life. The pathogenic mechanism [61–64] is not clear, and the passive transmission test is negative. Leukocytosis is generally present and skin biopsy reveals a polymorphonuclear infiltrate. Diagnosis is on the basis of a positive family history, onset at birth, a negative ice-cube test and the presence of systemic signs. Daily antihistamines have been reported to decrease symptom severity. In some cases, stanozolol, an attenuated androgen, can help [65].

13.3 Aquagenic Pruritus

Showering or bathing are very enjoyable daily interludes, essential for hygiene and sometimes as a form of treatment. However, some subjects suffer acute pruritus as a direct consequence of bathing. In some of them, contact with water is an indirect stimulus (acute pseudo-aquagenic reactions) (Table 13.3), while in others the pruritus is a direct local effect of skin contact with water, as in aquagenic urticaria (featuring objective signs), and aquagenic pruritus (no objective signs).

The latter, in turn, may be classified as:

1. true aquagenic pruritus (Table 13.6);
2. senile aquagenic pruritus (Table 13.7);
3. aquagenic pruritus, observed in 40–50 % of patients with polycythaemia rubra vera (Table 13.8).

True aquagenic pruritus, first reported in 1970 by Shelley [66], features intense pricking or burning pruritus, that develops after contact with water,

Table 13.6 Diagnostic criteria for aquagenic pruritus

1. Intense, recurrent itching after contact with water, regardless of temperature
2. Itching onsets within a few minutes after contact with water and may persist for up to 2 h
3. No visible skin signs
4. Cold aquagenic cholinergic heat and cold localized heat urticaria forms and symptomatic dermographism have been excluded
5. Polycythaemia rubra vera has been excluded

Table 13.7 Diagnostic criteria for senile aquagenic pruritus

1. Especially in the female sex (75 %), in subjects over 60 with pale skin
2. Excessively dry skin
3. Intense itching after drying
4. The intensity of the itching sensations is proportional to the duration of exposure to water and the degree of skin dryness
5. Triggering factors: contact with water and consequent dryness of the skin, variations in temperature, friction
6. The itching starts on the legs or forearms, then spreads and persists for 10–60 min
7. The intensity of the itching increases with age and during the winter

Table 13.8 Diagnostic criteria for aquagenic pruritus associated with polycythaemia rubra vera

1. Only subjective signs as in aquagenic pruritus
2. Specific symptoms of the disease
3. More frequent onset of spontaneous itching
4. Itching generally depends on the temperature of the water
5. Hot baths elicit worse itching than cool baths
6. Cooling of the skin provokes itching
7. The intensity of the itching is not correlated with the severity of the disease

Table 13.9 Diseases which may cause aquagenic pruritus

Dermatological diseases	Juvenile xanthogranulomas, urticaria factitia
Infectious diseases	Hepatitis C
Intestinal diseases	Lactose intolerance
Solid neoplasms	Uterine cancer
Haematological and lymphoproliferative diseases	Polycythemia vera, haemochromatosis, acute lymphoblastic leukemia, essential thrombocythemia, myeloblastic syndrome, T-cell non-Hodkin's lymphoma
Drugs	Antimalarials (chloroquine, hydroxychloroquine), bupropion, clomipramine

regardless of temperature, with no apparent objective signs. It lasts between 10 and 120 min. In some subjects it manifests during bathing, while in others the onset occurs immediately after they come out of the water. In almost all cases the legs and thighs are mainly affected, although the trunk and arms may also be involved [8, 67].

There are few studies of the prevalence and incidence of aquagenic pruritus: in small patients cohorts the incidence ranged from 1.4 % [68] to 4.5 % [69], and up to 23.8 % in a cohort of young Nigerians [70]. The affliction is often reported as an accompanying or premonitory symptom in various underlying systemic diseases (Table 13.9) [71–74]. The onset of aquagenic pruritus is observed above all in subjects with no underlying disorder; these forms, of uncertain origin, have been subdivided by some authors into two subgroups depending on the patients age, young or elderly [68, 75].

The most common association is with polycythemia rubra vera (PcV), first reported in 1985 [76, 77]. Pruritis, that may precede the onset of PcV by as many as 13 years [72], is present in 40–50 % of patients with PcV. A mutation of the janus kinase 2 enzyme (JAK2) has been found to be strongly associated with aquagenic pruritus in PcV patients; the mutation affects a valine-phenylalanine exchange at position 617 (JAK2617 V>F) [78]. Subjects with a homozygotic mutation have a significantly higher rate of aquagenic pruritus than those with a heterozygotic mutation [78].

Drug-induced aquagenic pruritus seems to be rare; some antimalarial drugs (chloroquine, hydroxychloroquine) and anti-depressants (bupropion, clomipramine) have been implicated [74, 79, 80].

The mechanism of cutaneous induction of aquagenic pruritus is not well understood. Although both the release of histamine and mast-cell degranulation have been demonstrated, histamine does not seem to be the main mediator. Intradermal injections of acetylcholine do not reproduce the itching symptoms [74, 81, 82]. An increased fibrinolytic cutaneous activity has recently been shown, although the plasma fibrinolytic activity was within normal limits [81]. This increased fibrinolytic cutaneous activity can be blocked by ε-aminocaproic acid, suggesting that the increased activity may be due to an inherently increased plasminogen activity. Intradermal histamine and acetylcholine injections trigger an increased cutaneous fibrinolytic activity, which implies that the phenomenon may be secondary to histamine and acetylcholine release.

Aquagenic pruritus does not respond well to H1 and H2 antihistamines, that only partially relieve the symptoms. The most efficacious treatment, that can reduce symptoms up to 100 % in 50 % of patients, is UV therapy [67, 73, 83, 84], which acts by minimizing the blood levels of eosinophils. Both systemic and bath PUVA (also reducing PUVA to one to two times a week), and the combination of UVA/UVB, as well as a narrow band and broadband UVB therapy, can reduce pruritus. Other drug options include anticonvulsant drugs (pregabalin) [74], propranolol, a beta-receptor antagonist of adrenaline [85], and atenolol, a long acting beta-1 selective adrenergic receptor blocker [82]. Emollients and alkalization of bath water (pH 8), by adding sodium bicarbonate (0.1–0.5 kg/bath), reduce the itching symptoms in some cases.

13.4 Contact Dermatitis

Contact dermatitis caused by the swimming costume is rare but possible, due to sensitisation to the elastic (rubber additives) or dyes.

Contact dermatitis from diving equipment can be observed, due to professional or sports activities, and induced by the mask (Figs. 13.3 and 13.4), goggles, snorkel, fins and rubber wetsuits [86–89]. The sensitising substances responsible are mercaptobenzothiazole, thiourams, dithiocarbamates, paraphenylenediamine, thiourea compounds, formaldehyde, butylphenolformaldehyde resin, isopropyl-phenyl-paraphenylenediamine, and carbamates.

In sensitized subjects, snorkels can also cause inflammation of the mouth, that generally starts with an intermittent, mild burning sensation associated with ingesting hot drinks or spicy foods.

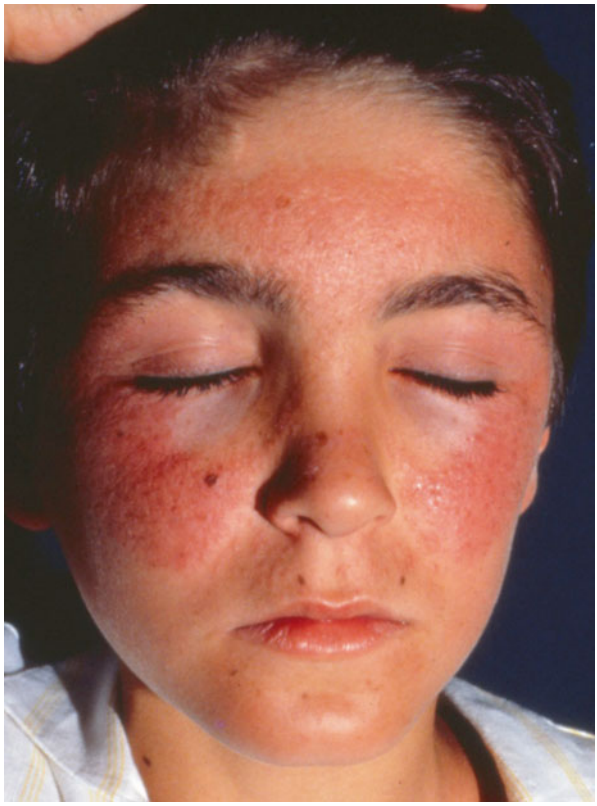

Fig. 13.3 Allergic contact dermatitis to rubber mask

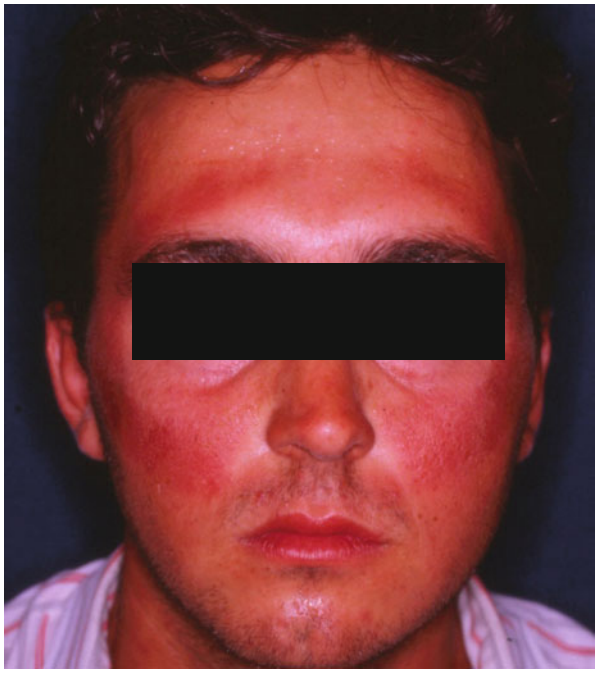

Fig. 13.4 Allergic contact dermatitis to rubber mask

13.5 Saltwater Dermatitis

Prolonged immersion in seawater causes electrolytic alterations due to percutaneous absorption (immersion syndrome). Occasionally, a skin peeling effect may appear where the swimming costume hugs closely, that may even evolve into ulceration due to the combination of friction and the abrasive effect of the salt.

Surfer's nodules are hard and indolent and onset at the level of the anterior tibial region. They are reversible and caused by continual contact with the board. These pretibial fibrotic masses may be dermal or hypodermal, and deformity of the underlying bone and calcifications can also ensue [90].

External otitis is an acute bacterial infection of the external ear fostered by the macerating effect of the water and the persistent humidity of this part. The clinical symptoms are pain, exudation, pruritus and sometimes fever and impaired hearing. The most common bacterial cause is *Pseudomonas aeruginosa*.

13.6 Freshwater Dermatitis

Swimming in chlorinated pools has a dehydrating effect on the skin and hair (anti-oil action) that is more evident in atopic subjects. Depending on the concentration, chlorine has a bleaching effect on the hair, which is most apparent in blonde subjects and in the summer months in combination with the sunrays. A greenish tinge may develop in blonde subjects who often swim in strongly chlorinated pools; shampooing the hair immediately after swimming is the best prophylaxis. Temporary chemical conjunctivitis (so-called "red eyes") is observed in subjects who swim with their eyes open.

Various dermatological diseases (Table 13.10) can be caused, via different pathogenic mechanisms, by chemical irritants [91, 92], allergens [92–96] and infectious agents present in swimming pool water [97–102], as well as by the irritant effect of water itself [103]. In particular, these diseases can be of occupational type (in professional swimmers, hydrotherapists, physiotherapists, and swimming pool workers such as cleaners and attendants) [104, 105].

Chlorine and bromine-based compounds are widely used to disinfect swimming pool water, destroy microorganisms and oxidate organic waste originating in the bather's body. The weak hypochlorous acid formed in the reaction between chlorine-based compounds and water is the main active compound in such disinfection processes. The undissociated form of this acid reacts with the cellular component of the microorganism and destroys it, while the hypochlorite anion assists in the oxidation of organic waste followed by formation of chloramines [104]. Both irritant and allergic contact dermatitis, induced by the chemical disinfectants, has been observed among pool users [96, 104–108]. Since the 1980s, owing to their potent action against waterborne pseudomonas, bromine derivatives have largely replaced chlorine derivatives as disinfectants in many swimming pools, resulting in a higher incidence of skin irritations [105]. Other symptoms include hair discoloration, reported in 30 % of swimmers [101], changes in the fingernails and toenails, and irritation and drying of the oral and genital mucosa.

Table 13.10 Dermatological diseases from swimming pools

Irritant contact dermatitis
Allergic contact dermatitis
Folliculitis
Hot foot syndrome
Swimming pool granuloma
Warts
Molluscum contagiosum
Xerotic skin
Hair discoloration
Fingernails changes
Aquagenic wrinkling
Chemical conjunctivitis
Otitis externa

Aquagenic skin wrinkling, a classic manifestation in patients with cystic fibrosis, presents in the form of white, oedematous, poorly delineated papules and plaques of the palms of the hands and soles of the feet, following water exposure [109, 110]. The lesions appear within 2 min of exposure, are transient and resolve within a few hours after the end of exposure. Other signs include discomfort, pruritus, tingling and hyperhydrosis. Histologically, there is hyperkeratosis and dilation of the eccrine ostia [111, 112]. This phenomenon has been given various names, when observed in absence of cystic fibrosis, such as aquagenic keratoderma, aquagenic syringeal acrokeratoderma, aquagenic palmoplantar keratoderma, and transient reactive papulotranslucent acrokeratoderma.

13.7 Dermatitis Associated with Deep Sea Diving

Notoriously, both occupational and amateur scuba divers are exposed to an enormous variety of risks, including skin problems [113].

Staphylococcal skin infections are relatively frequent and also difficult to treat [114]. The pressurized environment, with a high partial pressure of oxygen, high temperatures and sometimes humidity exceeding 90%, are conditions that favor *Pseudomonas* external otitis. This problem can be preventable by good prophylactic hygiene and by use of aluminum acetate ear drops [115]. Overheating inside the wetsuit can cause local burns. Underwater welding procedures can induce erythema and telangiectasia. The skin folds trapped in the wetsuit can present linear abrasions.

During decompression, divers may notice itching, with or without an urticarial eruption, mainly localized on the back or trunk [116]. If they stay underwater for long they may develop a form of "napkin rash", due to having to attend to physiological needs.

13.8 Occupational Chronic Traumatic Scleroedema

Diffuse delayed reactions of the backs of the hands may manifest as a particular form of chronic scleroedema. This traumatic lymphoedema is an occupational complaint and we have often observed it in fishermen, caused by repeated penetration of

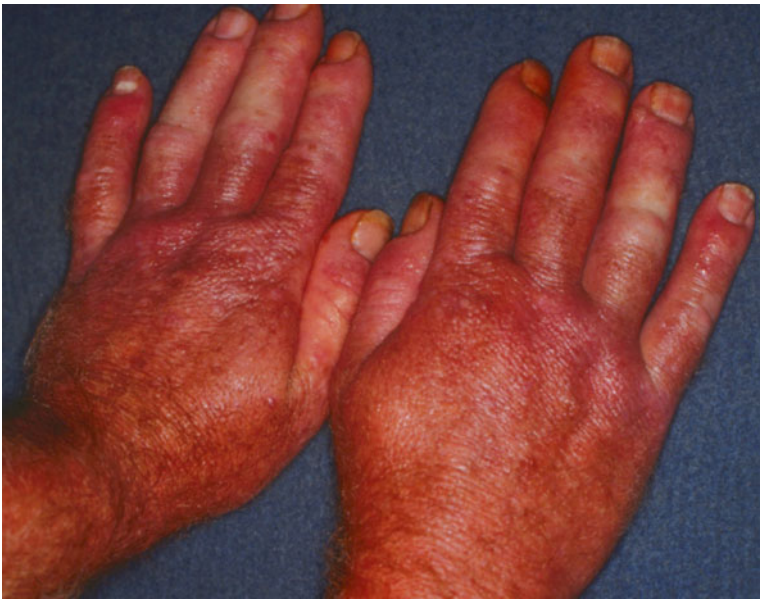

Fig. 13.5 Chronic traumatic scleroedema of the hands in a sea urchin fisherman. Acrocyanosis and skin atrophy are also evident

sea urchin spines, together with the constriction of the wrists caused by the wetsuit and the low temperature of the water. It manifests with hard, persistent oedema of the backs of the hands and sometimes also of the forearms (Figs. 13.5, 13.6, and 13.7) [116, 119].

The oedema is firstly recurrent but within a few years it becomes persistent, very hard and clearly distinct, ending in a sharp line at the wrists. It can persist for many years even after abandonment of the working activity and may be associated with "sea urchin granulomas", functional impairment of the wrists and fingers, dystrophic alterations of the nails and sometimes acrocyanosis, "cigarette-paper" atrophy of the affected skin and morphological alterations of the joint. In one case with intense scleroedema and granulomas, lymphography of the upper limb showed an irregular spread and distribution of the contrast medium on the back of the hand (Figs. 13.8 and 13.9) [117–120].

This picture of hard scleroedema closely resembles Secrétan's syndrome, a cutaneous artefact due to self-infliction of various repeated mechanical stimuli (haemostatic ligatures, occlusive bandaging, traumas) for financial gain (generally to obtain a pension) or psychiatric reasons [121].

Spontaneous, chronic professional scleroedema of the hands must thus be differentiated from self-inflicted complaints (Table 13.11) and other acute or chronic lymphedemas, such as lymphatic aplasia, recurrent erysipelas, deep thrombophlebitis, angioedema, chilblains, urticaria due to the cold, filariasis, venous obstruction, complications of surgical operations and radiotherapy for breast cancer or other tumors.

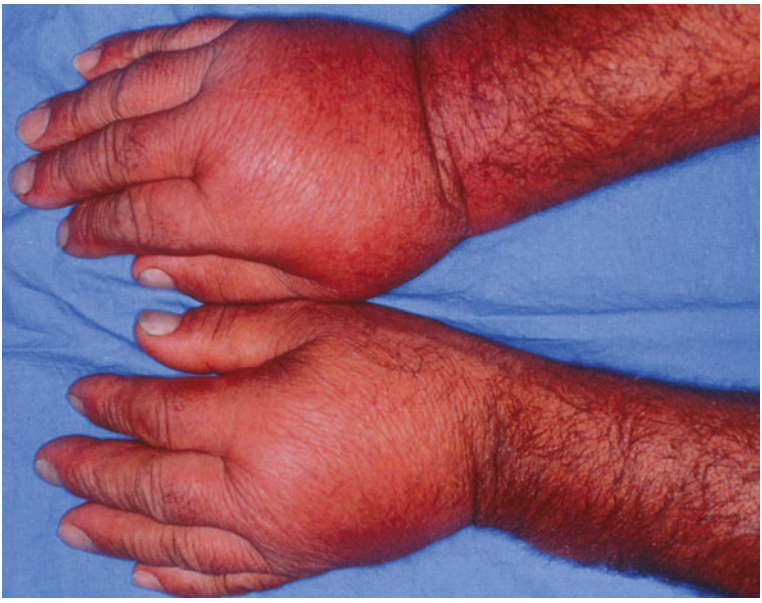

Fig. 13.6 Intense chronic traumatic scleroedema of the hands and forearms. The fisherman had stopped this working activity 15 years before. The joint function is impaired (Reproduced with permission from Bonamonte and Angelini [117])

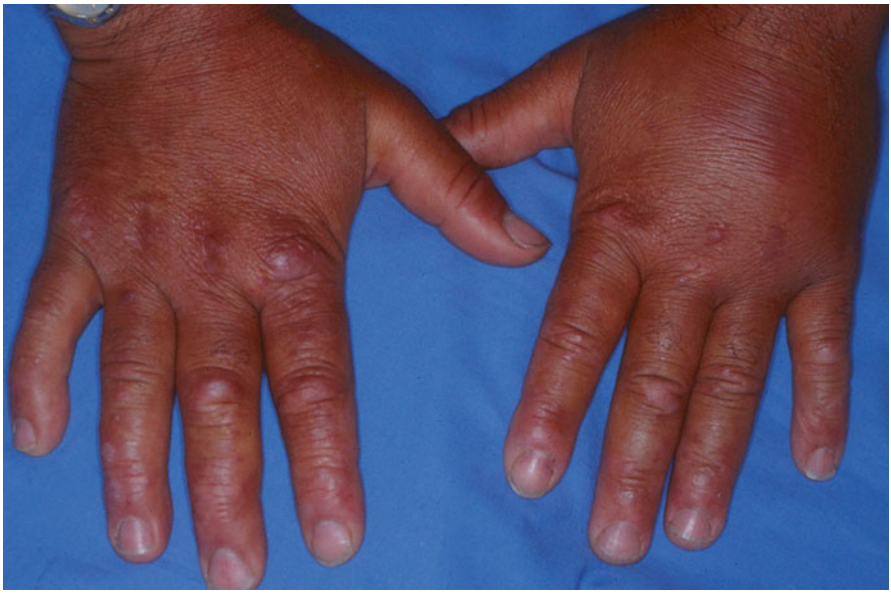

Fig. 13.7 Chronic traumatic scleroedema of the hands and granulomas from sea urchins (Reproduced with permission from Cassano and Coll. [118])

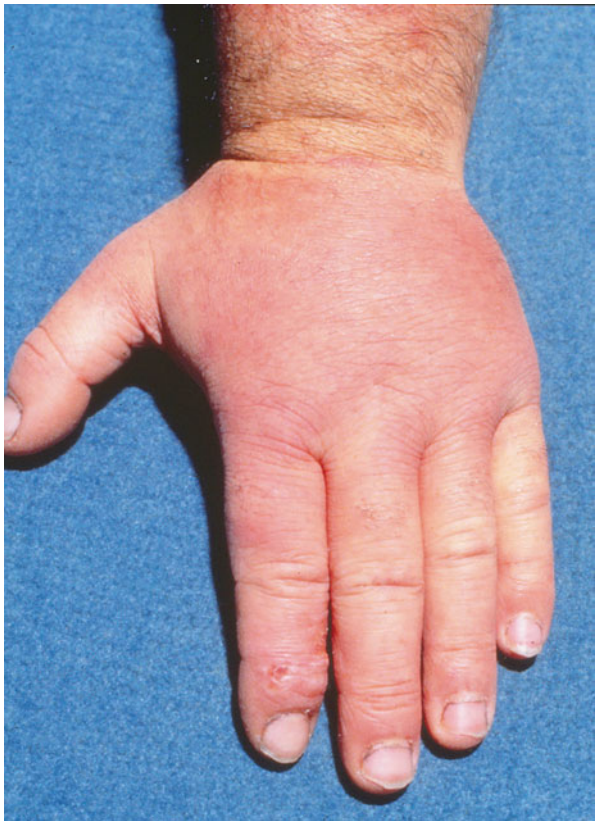

Fig. 13.8 Irreversible chronic traumatic scleroedema and granulomas from sea urchins in a subaqua diver

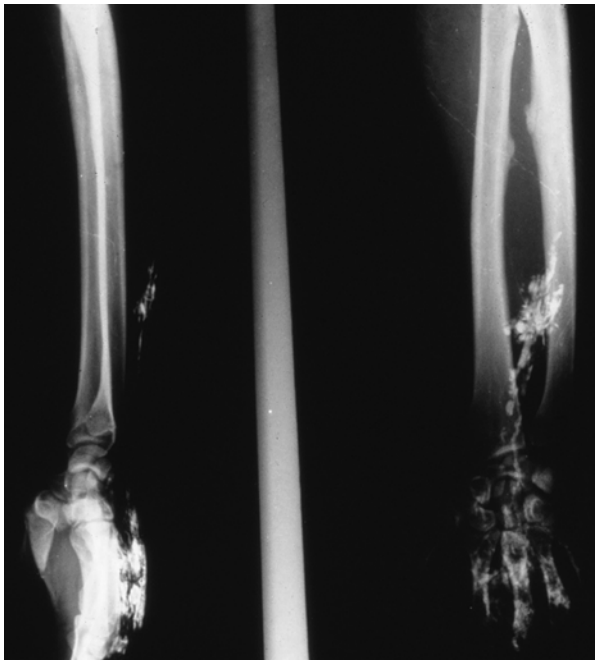

Fig. 13.9 The same case as in Fig. 13.8. Lymphography shows irregular flow and distribution of the contrast medium on the back of the hand

Table 13.11 Differential diagnosis between spontaneous professional scleroedema (SPS) of the hands and non spontaneous Secrétan's syndrome (NSSS)

	SPS	NSSS
Acrocyanosis	++−−	++−−
Lesions		
Monolateral	+−−−	++++
Bilateral	++++	+−−−
In association with		
Artefact dermatosis	−−−−	++−−
Sea urchin granulomas	++−−	−−−−
Age and sex		
Young women	−−−−	++−−
Young or elderly men	++++	++−−
Worse in winter	++++	−−−−
Lesions persist after abandonment of working activity	++−−	−−−−
Psychiatric problems	−−−−	++−−

13.9 Cutaneo-Systemic Complaints in Fishermen

Deep-sea fishermen can be victims of rare, practically unthinkable events nowadays such as a possible encounter with a bomb containing mustard gas. From 1970 till now, we have observed 12 fishermen with dermatitis whose onset occurred 6–10 h after fishing in the open sea outside Molfetta, a city 30 km to the North of Bari [122, 123]. All these patients presented an intensely erythemato-oedematous dermatitis featuring widespread blistering lesions with a clear liquid content (Figs. 13.10, 13.11, and 13.12). The affliction particularly involved the hands, forearms and face (where the erythema and oedema were more marked, being especially severe on the eye-lids), while in three cases the genitals were also affected (Fig. 13.13), with intense erythema in two cases and erythema, oedema and blisters in 1. All the cases were associated with severe conjunctivitis, lachrymation and photophobia. There was intense burning and itching at the affected sites. In six patients the skin symptoms were associated with headache, vomiting and nausea.

The fishermen referred that when they pulled their nets on board they had found bombs mingled in with the fish (Fig. 13.14). A few hours after handling the nets contaminated with the liquid gas contained in the bombs, they suffered the above symptoms. The dermatitis of the hands and forearms was obviously induced by direct irritant contact with the contaminated bombs and nets, while the other skin and mucosal lesions were caused by evaporation of the gas and hence airborne cutaneo-mucosal dermatitis (irritant airborne contact dermatitis) [123]. In all the cases, the symptoms resolved after 10–15 days, leaving dark skin patches. The conjunctivitis was treated with eye-baths containing 2 % sodium bicarbonate and antibiotic eye-drops. The other symptoms regressed rapidly with symptomatic treatment. Controls after 20–30 days excluded any re-presentation of the dermatitis.

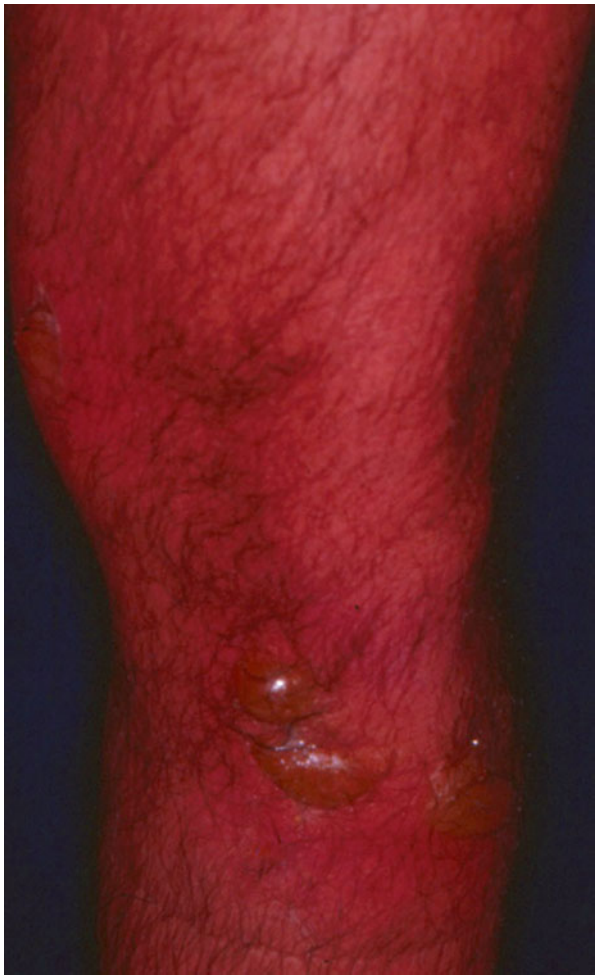

Fig. 13.10 Blistering dermatitis from mustard gas

The risk of fishing bombs is well known to fishermen and the harbor authorities in the area. These authorities report that more than 100 cases of intoxication from mustard gas have been observed over the years. This gas (2,2′-dichlorodiethyl sulfide: $C_4H_8CL_2S$), one of the most aggressive gases used in chemical warfare, is also known as yperite after the city of Ypres (Belgium), where it was first used in July 1917. The English and Americans call it mustard gas because of its characteristic odor. In the pure state it is an odorless, colorless oily liquid, and the characteristic yellowish-brown color and mustard-like smell are due to impurities (ethylsulphides). It is poorly soluble in water but dissolves rapidly in organic solvents or fats; this facilitates penetration of the cells, where it has a toxic effect. It evaporates

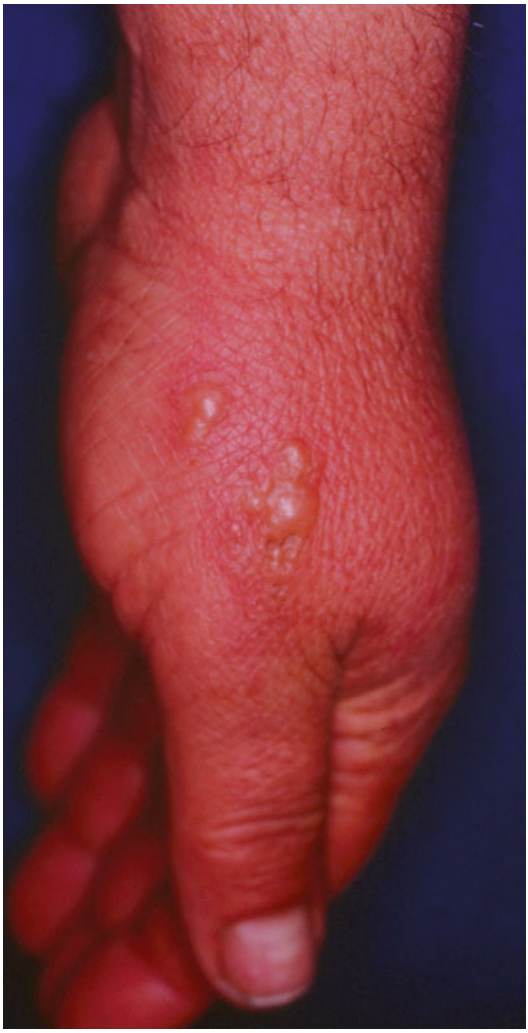

Fig. 13.11 Blistering dermatitis from mustard gas

slowly because of its low vapor pressure, although this increases at higher temperatures. It is toxic in both liquid and vapor form: in the former cases it damages the skin and in the latter, the skin, conjunctiva and respiratory mucosa. Its toxic effects manifest after 4–24 h from exposure [124–126].

Since the First World War, intoxication from mustard gas has been caused only by occupational contact, except for cases arising due to its widespread use during the Iran-Iraq war (1980–1988) [127]. The cases we observed were attributable to the previous presence of factories loading and unloading mustard gas bombs in Molfetta. After the Second World War, the bombs were thrown into the sea about three miles from the coast. The bombs are therefore still fished up sometimes, especially in the summer season when drag-nets are used.

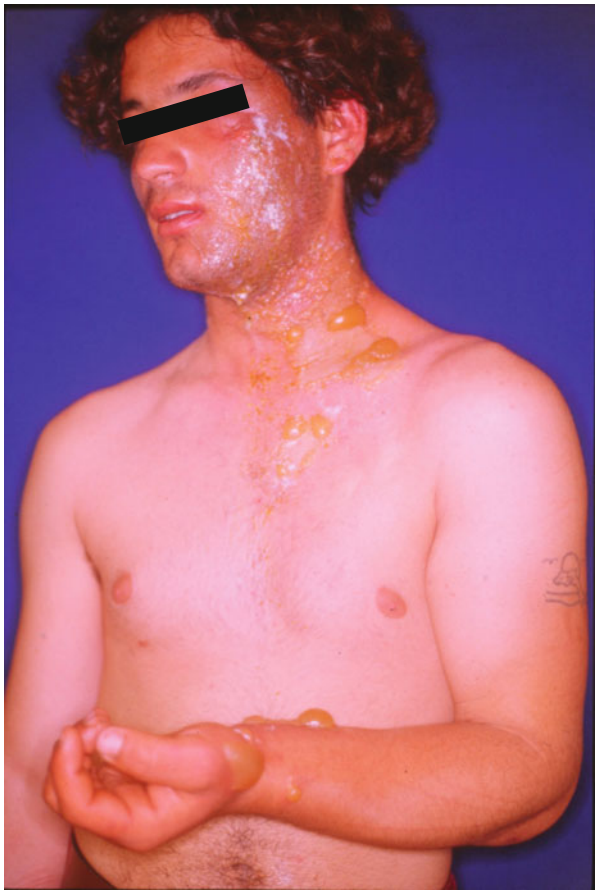

Fig. 13.12 Blistering dermatitis from mustard gas

Although chemical bombs are present in all European seas, similar cases of dermatitis from mustard gas have only occasionally been reported [128–130], probably because it is practically impossible to connect the disease with contamination by fishing nets unless the bombs are actually seen in them. Otherwise, the skin symptoms may be attributed to the harmful action of some marine flora and fauna.

Fishermen should be informed of the risks of fishing bombs in particular areas, and must be instructed to throw them straight back into the water without opening them and in cases of inadvertent contamination, to go straight to hospital. All the contaminated areas of the boat must be thoroughly cleaned and the fishermen's clothes and personal effects must be destroyed. Mustard gas can impregnate clothes and leather objects and persist for a long time. In fact, we have also observed cases of contamination of members of the family due to contact with the fisherman's clothing.

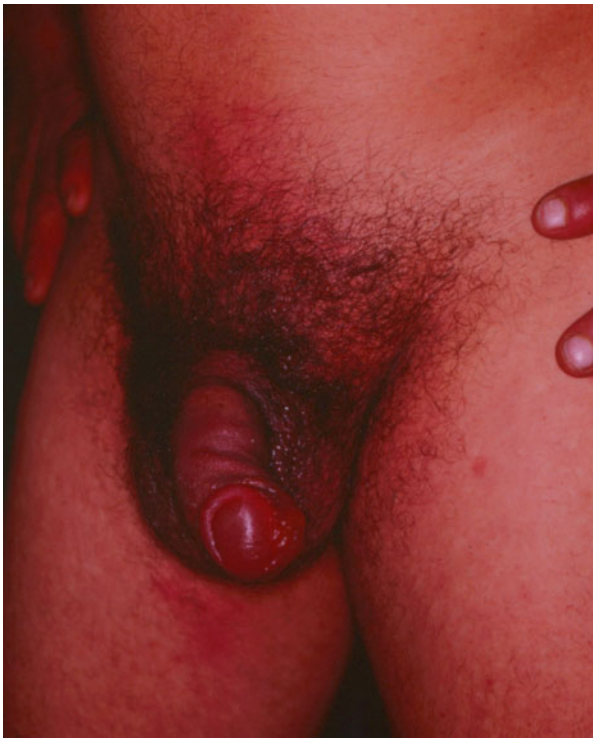

Fig. 13.13 Intensely erythemato-oedemato-exudative dermatitis from mustard gas

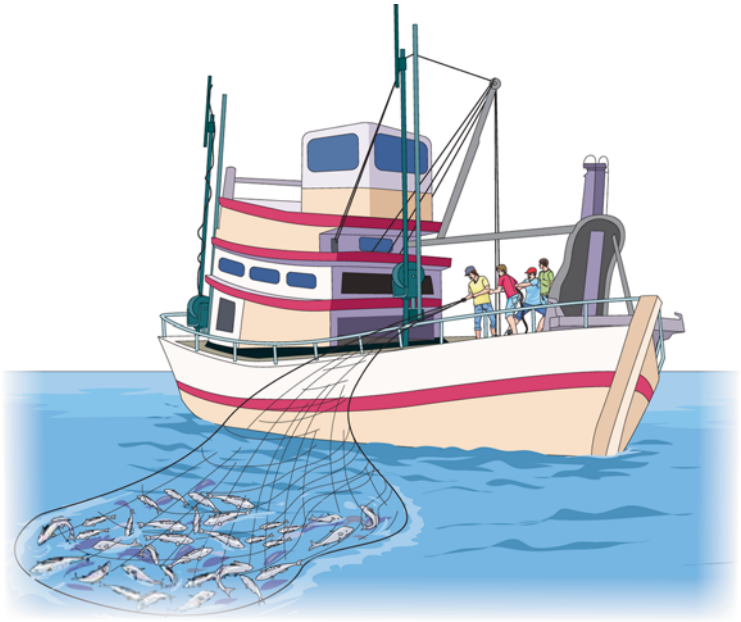

Fig. 13.14 Bombs containing mustard gas can be pulled in together with the fish in dragnet fishing

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