

Protein Contact Dermatitis: Allergens, Pathogenesis, and Management

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Protein contact dermatitis is an allergic skin reaction induced principally by proteins of either animal or plant origin. The clinical presentation is that of a chronic dermatitis, and it is often difficult to differentiate between allergic contact dermatitis and other eczematous dermatoses. One distinguishing clinical feature is that acute flares of pruritus, urticaria, edema, or vesiculation are noted minutes after contact with the causative substances. Additionally, the patch-test result is typically negative, and the scratch- or prick-test result is positive. The pathogenesis of protein contact dermatitis is unclear but may involve a type I (immunoglobulin E [IgE], immediate) hypersensitivity reaction, type IV (cell-mediated delayed) hypersensitivity reaction, and/or a delayed reaction due to IgE-bearing Langerhans' cells. Management involves avoidance of the allergen.

THE TERM "PROTEIN CONTACT DERMATITIS" was coined by Hjorth and Roed-Petersen in 1976¹ to describe the condition of several food handlers with hand and forearm eczema. The responsible allergen in these patients was not a low-molecular-weight substance, as in classic allergic contact allergy, but a protein of greater molecular weight. Additionally, these patients had positive scratch-test results and (in some cases) specific immunoglobulin E (IgE) antibodies to the food they handled, which included meat, fish, cheese, vegetables, and spices, whereas patch-test results were often negative. Of 33 patients evaluated, only 6 exhibited delayed hypersensitivity; in 10 patients, scratch tests revealed the only explanation for their eczema. Most patients did not have respiratory or mucosal symptoms. The authors postulated that protein contact dermatitis was a combined type I- and type IV-mediated allergic reaction to "proteins."

In 1983, Veien and colleagues² further characterized protein contact dermatitis. They defined specific criteria, including (1) a chronic recurrent dermatitis caused by contact with proteinaceous material, (2) an acute urticarial or vesicular eruption occurring minutes after contact with the causative protein, (3) immediate testing results that are usually positive, and (4) patch-test results that are often negative.

Because there is an immediate wheal and flare response, some authors have classified protein contact dermatitis as part of the "contact urticaria syndrome," a term that encompasses immunologic, nonimmunologic, and uncertain mechanism-mediated contact urticaria. In this view, protein contact dermatitis is not a unique entity and may be caused by both nonimmunologic (irritant) and immunologic (allergic) mechanisms.^{3,4} This article will refer to protein contact dermatitis as that which is immunologically mediated.

Because protein contact dermatitis is relatively rare, little is known about its epidemiology. Information from case series indicates that protein contact dermatitis is primarily seen in chefs, veterinarians, dairy workers, and workers in other occupations in which there is significant protein exposure.⁵ The purpose of this review is to summarize the current literature on the clinical presentation, pathogenesis, and management of protein contact dermatitis.

Clinical Presentation

Protein contact dermatitis describes a distinct type of chronic recurrent dermatitis that differs from the wheal and flare reaction of contact urticaria.⁶ The dermatitis usually affects the hands and forearms, and these areas are typically diffusely involved.⁷ Sometimes, only the fingertips are involved. Other and less common sites include facial areas.⁸ Among food handlers, chronic paronychia is a not uncommon presentation, with the proximal nail fold demonstrating severe swelling and itching.⁹ This pattern has also been recently described as occurring with latex

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allergy.¹⁰ Vesicular, urticarial, or edematous papules and plaques may occur within a few minutes after contact with the causative allergen. Pruritus, burning, stinging, and/or pain are not uncommon.^{8,11} Extracutaneous reactions, including angioedema, gastrointestinal symptoms, rhinoconjunctivitis, and bronchial asthma, may accompany the skin reactions if the allergen is ingested.^{12–14}

Contributory Proteins

The proteins that induce protein contact dermatitis are traditionally classified into four groups.⁵ These include fruits/vegetables/spices/plants (group 1), animal proteins (group 2), grains (group 3), and enzymes (group 4). In 1995, Janssens and colleagues⁵ summarized proteins that had been reported to cause protein contact dermatitis (Table 1). Since that review, many more reports have been published; they are summarized by protein group in Tables 2 to 4. Interestingly, there are very few reports of latex-associated protein contact dermatitis, although immediate and delayed sensitivities to latex have been known to exist for some time and are suspected to be relatively common.¹⁵ It is possible that all type I–mediated allergy to latex is assumed to be contact urticaria even if the clinical picture (ie, an eczematous response) suggests protein contact dermatitis.¹⁰

Risk Factors

Because protein contact dermatitis is relatively rare, there have been no formal epidemiologic studies. Anecdotally, risk factors for the development of protein contact dermatitis include atopy, chronic irritant dermatitis, and an occupation or hobby involving exposure to protein allergens. Approximately 50% of cases are associated with atopy.⁵ The reasons for this are unclear. It is possible that the immune mechanisms in atopic dermatitis and protein contact dermatitis are similar (see “Pathogenesis,” below) or that high-molecular proteins are more likely to penetrate the impaired skin barrier in atopic individuals.¹⁶ Chronic irritant dermatitis may represent a separate risk factor.⁵ As shown in Tables 2 to 4, there does not appear to be a gender predilection.

Protein contact dermatitis from fruits, vegetables, spices, and plants is most common in kitchen workers, caterers, food vendors, food packers, and gardeners.^{1,2,6} Commonly, these patients have involvement limited to the hands, fingers, and wrists although one case report described a patient with worsening facial rosacea papules and pustules.¹⁷ Occasionally, ingestion of the raw fruits or

vegetables will induce coughing, dysphagia, dyspnea, rhinitis, asthma, or gastrointestinal symptoms.^{18–20} Systemic symptoms are most often observed in atopic and birch pollen–sensitive individuals.²¹

Animal protein contact dermatitis is most often observed in slaughterhouse workers, butchers, fisherman, cooks, farmers, and veterinarians. Butchers and slaughterhouse workers are particularly susceptible owing to contact with animal guts.²² Bovine amniotic fluid and blood are common triggers in veterinarians,^{23,24} as is raw fishing bait in fishermen.²⁵

Flour-associated protein contact dermatitis has been reported primarily among bakers and home helpers, who often present with chronic hand dermatitis but sometimes present with a more generalized distribution often involving the face. Rhinoconjunctivitis and asthma may occur in this population. The most common culprits are wheat and rye.^{5,26,27}

Proteolytic enzyme–associated protein contact dermatitis is most common among pharmaceutical workers, soap makers, and chemical enzyme factory workers. In addition, bakers may react to enzymes used as dough enhancers, such as α -amylase.¹³ Respiratory symptoms are more common in protein contact dermatitis from enzymes.^{5,28}

Methods of Diagnostic Testing

Several methods are currently used to test for protein contact dermatitis from a particular allergen. The most common methods include the open application test, prick tests, scratch tests, patch tests, and the radioallergosorbent test (RAST). Prick and scratch tests are most sensitive for detecting protein contact dermatitis. Patch-test results are usually negative, and RAST results are sometimes positive. While prick tests and scratch tests with serially diluted antigens are generally considered safe, systemic symptoms and anaphylaxis may occur.²⁹ Therefore, these tests should be performed only in settings where resuscitation equipment and trained personnel are available. The use of diluted allergens is especially important for patients with a history of systemic symptoms.³⁰ Because testing with commercial food allergens often yields negative results, the actual raw and cooked foods should be used for testing.³¹ The methodologies of these immediate tests are briefly described below.³²

Open Application Test

Similar to the skin application food test used to diagnose food allergy in atopic children,³¹ an open application test

Table 1. Food Products and Constituents Causing Protein Contact Dermatitis as Reported before 1995

<i>Fruits, Vegetables, Spices, Plants</i>		<i>Animal Proteins</i>	<i>Grains</i>	<i>Enzymes</i>
Almond	Blood (pig, cow, lamb)		Barley	α -amylase
Banana	Amphibian serum		Rye	
Bean	Cheese (cheddar, cheese products, Emmenthal)		Wheat	
Caraway	Egg yolk		Oat flour	
Carrot	Epithelium/dander (cow, pork)			
Castor bean	Fish (cod, cuttlefish, lobster, mackerel, plaice, shellfish, shrimp, whitefish)			
Cauliflower	Gut (pig)			
Celery	Liver (chicken)			
Chicory	Meat (beef, pork, chicken, horse, lamb)			
Chrysanthemum	Mesenteric fat			
Cress	Milk (cow's, milk products)			
Cucumber	Saliva (cow)			
Curry	Skin (chicken, turkey)			
Dill	Worms (<i>Nereis diversicolor</i>)			
Eggplant				
Endive				
Fig				
Garlic				
Hazelnut				
Horseradish				
Kiwi fruit				
Lemon				
Mushroom				
Onion				
Paprika				
Parsley				
Parsnip				
Peanut				
Pineapple				
Tomato				

Adapted from Janssens V et al.⁵

involves placing or rubbing the food on intact skin. Because test results on normal skin are often negative, it is important also to test the substance on damaged or eczematous skin. The open application test is generally thought to be less hazardous than invasive methods (prick tests and scratch tests). Therefore, some authors have recommended open skin application testing prior to the performance of more invasive tests.³³

Prick Test

In the prick test,³² one drop each of diluted test allergen, vehicle (negative control), and histamine (positive control) are applied to the volar aspects of the patient's forearms. The test sites are pierced with lancets to introduce the allergen into the skin. The test sites are read after 15 to 30

minutes. A positive reaction is a wheal of at least 3 mm in diameter and at least half the size of the histamine control in the absence of such a reaction in the vehicle control.

Scratch Test

One drop each of diluted test allergen, vehicle, and histamine are applied to the volar aspects of the forearms or back, and needles are used to scratch the skin slightly at these sites. Modified scratch tests involve scratching the skin first and then applying food items (Figs 1, 2 and 3). The test sites are read within 30 minutes. An edematous reaction that is at least half the diameter of the histamine control is considered positive in the absence of such a reaction in the vehicle control.

Table 2. Summary of Published Reports of Protein Contact Dermatitis from Fruits, Vegetables, Spices and Plants, 1995 to 2008

<i>Allergen</i>	<i>Patient Sex</i>	<i>Patient Occupation</i>	<i>Clinical Description</i>	<i>Diagnostic Test</i>	<i>Reference</i>
Asparagus	M	Cook	Itchy dyshidrosiform plaques on hands; dysphagia and dyspnea	Prick test(+) Patch test (+) Total IgE (high) Specific IgE (high)	50
Carrot, coriander, potato	M	Chef	Itchy hand (occasionally face) dermatitis	Prick test (+) Patch test (–) Total IgE (nl)	51
Cornstarch	F	Housewife	Wrists, forearms, neck, shoulders	Prick test (+)	52
Mugwort, paprika, curry, camomile, coriander, cumin	M	Butcher	Hand eczema; stomachache after eating curry	Prick test (+) Scratch test (+ for coriander) Total IgE (high) Specific IgE (high for mugwort, curry, paprika)	19
Natural rubber latex	M	General property maintenance worker	Itchy dermatitis of fingers; paronychia	Prick test (+)	10
Spathe flower, mugwort, yucca, weeping fig	M	Gardener	Dermatitis and wheals on hands, forearms; rhino-conjunctivitis	Prick test (+) Patch test (–) Specific IgE (high for spathe flower)	20
Apple, pear, peach, melon, potato, orange, garlic, onion	M	Kitchen assistant	Hand, finger, wrist eczema	Prick test (+) Patch test (–) Rub test (+ for peach) Specific IgE (+ for melon, pear, peach, potato, tomato, profilin)	53
Melon, curry, <i>Cynodon</i>	F	NR	Hand dermatitis	Prick test (+) Patch test (–) Specific IgE (+ for melon)	54
Shiitake mushroom, mugwort pollen	F	Shiitake mushroom plant worker	Vesicular dermatitis on palms, fingers; fever; dry cough	Prick test (+) Specific IgE (+)	55
Natural rubber latex, wheat, rye, barley, oat	F	Home helper	Fingertip dermatitis	Prick test (+) Total IgE (+) Specific IgE (+ for latex, wheat, rye, barley)	56
Soy	F	NR	Aggravation of rosacea (facial pustules, erythema, papules)	Prick test (+) Patch test (–)	17

F = female; IgE = immunoglobulin E; M = male; nl = normal; NR = not reported.

RAST

A RAST is an in vitro test that measures the amount of circulating antibody present in serum. Serum is incubated with the likely allergen, which is bonded to a solid polymer. If allergen-specific IgE is present in the patient's serum, it binds to the allergen. The amount of bound antibody can then be measured, most commonly with

radioactive or fluorescent techniques. Importantly, RASTs do not measure tissue-bound antibodies, only circulating antibodies; therefore, a negative RAST result does not rule out protein contact dermatitis or immediate hypersensitivity. In addition, many of the allergenic proteins responsible for protein contact dermatitis are unknown. Therefore, RASTs may be of little value. RAST results should also be carefully interpreted in the setting of high levels of total IgE.^{5,33,34}

Table 3. Summary of Published Reports of Protein Contact Dermatitis from Animal Proteins, 1995 to 2008

<i>Allergen</i>	<i>Patient Sex</i>	<i>Patient Occupation</i>	<i>Clinical Description</i>	<i>Diagnostic Test</i>	<i>Reference</i>
<i>Calliphoria vomitoria</i> maggots	M	Fishing bait factory worker	Vesicular hand dermatitis	Prick test (+) Patch test (–)	25
Milk proteins	M	Cheese factory worker	Erythema of hands, forearms, thighs, abdomen	Prick test (+) Scratch test (+)	57
Pork	M	Slaughterman	Exfoliative hand dermatitis	Prick test (–) Patch test (–) Specific IgE (+)	58
Meat (beef, pork, lamb, horse, fowl, goose, duck, turkey) wheat flour, rye flour, pasta, potato	M	Cook	Dyshidrotic dermatitis	Prick test (+) Scratch test (+)	59
Cow dander	F	Farmer	Eczema of face, hands, forearms, neck, elbows	Prick test (–) Scratch test (+) Patch test (–)	60
<i>Anisakis simplex</i> *	F	Housewife	Eczema of palms	Prick test, fish (–) Prick test, nematode (+) Rub test, nematode (+) Patch test (–)	61
Rainbow trout, whitefish, perch	M	Chef	Purulent hand dermatitis	Prick test (+) Specific IgE (+ for rainbow trout)	35
Chicken	F	Butcher	Dermatitis; urticaria	Prick test (+) Specific IgE (+) Basophil activation test (+)	62
Beef	M	Butcher	Papules at site of contact; dizziness; asthma; angioedema when eating	Prick test (+) Specific IgE (+) Open application test (+)	63
Salmon	M	Restaurant manager	Papules and wheals in area of contact	Prick test (+) Patch test (–) Open application test (–) Rub test (+) Specific IgE (+)	63
Beef, pork	F	Butcher	Fissured eczema	Prick test, immediate (–) Prick test, delayed (+) Patch test (–) Specific IgE (–) Rub test (–)	64
Baby squid	F	Fishmonger	Dermatitis of hands, forearms	Prick test (+) Patch test (–) Specific IgE (–)	65
Fishing bait (sea worm)	M	Building worker	Eczema of hands, palms	Prick test (+) Specific IgE (+) Patch test (–)	66
Meat	M	Cook	Hand eczema	Prick test (+) Specific IgE (+) Patch test (–)	67
Midge larvae	M	Painter (with fishing hobby)	Palmar eczema	Prick test (+) Specific IgE (+)	68

Table 3. Continued

<i>Allergen</i>	<i>Patient Sex</i>	<i>Patient Occupation</i>	<i>Clinical Description</i>	<i>Diagnostic Test</i>	<i>Reference</i>
Flesh fly maggots	M	Fisherman	Hyperkeratotic desquamative dermatitis	Patch test (–) Rubbing (+) Handling (+)	69
Bovine amniotic fluid	M	Veterinarian	Erythematous, edematous papules; plaques on hands, face	Prick test (–) Patch test (+ at 4 days) Open application test (–) Intradermal (+) Specific IgE (+)	70
Cuttlefish	F	Pizza maker	Edematous, erythematous papules and vesicles	Prick test (+)	35
Chicken viscera, gizzard	F	Poultry slaughter-house worker	Itchy dermatitis on dorsal hands, volar forearms	Prick test (+) Patch test (+ for nickel)	49
Halibut	F	Frozen-fish plant worker	Itchy erythematous plaques of hands, forearms	Prick test (+) Specific IgE (+ for hake, tuna)	49
Shrimp	M	Cook	Vesicular hand eczema; pruritus after oral ingestion	Prick test (+) Scratch test (+) Patch test (+) Specific IgE (+)	24

F = female; IgE = immunoglobulin E; M = male.

*Nematode in marine animals.

Pathogenesis

The pathogenesis of protein contact dermatitis is not entirely understood. Although the clinical presentation resembles allergic contact dermatitis, patch-test results are usually negative, whereas skin-prick or scratch-test results

are usually positive. Additionally, the histologic characteristics of protein contact dermatitis may resemble those of allergic contact dermatitis³¹ or urticaria.³⁵

Several theories regarding the pathogenesis of protein contact dermatitis have been proposed. First, it is possible that protein contact dermatitis is primarily a type I

Table 4. Summary of Published Reports of Protein Contact Dermatitis from Grains and Enzymes: 1995 to 2008

<i>Allergen</i>	<i>Patient Sex</i>	<i>Patient Occupation</i>	<i>Clinical Description</i>	<i>Diagnostic Test</i>	<i>Reference</i>
Flour (wheat, rye)	F	Pizza maker	Edematous, erythematous papules and vesicles	Prick test (+) Patch test (–) Specific IgE (+)	71
Corn flour	M	Corn flour plant worker	Hand dermatitis	Prick test (+) Scratch test (+) Specific IgE (+)	26
Natural rubber latex, wheat, rye, barley, oats	F	Home helper	Fingertip dermatitis	Prick test (+) Total IgE (+) Specific IgE (+ for latex, wheat, rye, barley)	56
Glucoamylase	F	Chemical enzyme factory worker	Dermatitis of dorsal finger	Prick test (+) Patch test (–)	27
Lactase	F	Pharmaceutical worker	Urticaria, eczema of hands, arms, face; rhinoconjunctivitis; throat irritation; cough	Prick test (+) Patch test (slight +) Specific IgE (+)	28

F = female; IgE = immunoglobulin E; M = male.



Figure 1. Modified scratch testing of a cook with hand and forearm dermatitis. Site one is the positive histamine control.

immediate hypersensitivity reaction with superimposed irritant and/or allergic contact dermatitis. Protein contact dermatitis is often accompanied by flares of urticarial eruptions shortly after contact with the causative material, and this phenomenon may represent type I IgE reactivity. The “eczema” noted in most individuals with protein contact dermatitis may be irritant dermatitis (preexisting or secondary to excoriation) or coexisting allergic contact dermatitis.

Second, it is possible that the allergens causing protein contact dermatitis induce a combined type I and type IV hypersensitivity reaction. If this is the case, then one must explain why patch-test results are so often negative in these patients. Some experts believe that patch-test results may be falsely negative, and that these reactions may become positive if allergens are occluded for longer periods.³⁶ Others suggest that a higher concentration of antigen may



Figure 2. Positive wheal and flare from shrimp.



Figure 3. Positive wheal and flare from green pepper.

be needed to penetrate skin to produce a positive delayed patch-test reaction than to produce the urticarial wheal in prick or scratch testing.⁵ It is also plausible that the patch-test results are negative because the protein-based molecules are too large to penetrate intact normal uninvolved skin.^{1,37} Therefore, stripping of the skin prior to patch testing has also been recommended.³⁶ This hypothesis is supported by the fact that most cases of protein contact dermatitis develop after chronic skin irritation. This may also explain why the result of standard patch testing is often negative, whereas scratch or prick tests yield positive results. It is also plausible that the positive type I histamine response may prevent the detection of a type IV hypersensitivity reaction. In an experiment involving chronic dermatophytosis, testing with *Trichophyton mentagrophytes* antigen revealed an immediate wheal and flare (type I) reaction with no subsequent delayed type IV response in eight patients. However, when the antihistamine chlorpheniramine was injected along with *Trichophyton*, the immediate reaction was partially blocked, and a positive delayed type IV response was uncovered in 3 of 8 patients tested.³⁷ The authors of this experiment posited that the IgE (T1-H1) antibody either depletes the available antigen in forming antigen-antibody complexes or that the resulting wheal and flare reaction nonspecifically antagonizes the delayed reaction.

Finally, others have suggested that the pathogenesis of protein contact dermatitis involves IgE-presenting Langerhans' cells. These cells are also thought to be important in atopic dermatitis.^{5,38,39} The acute inflammatory response in atopic dermatitis skin is facilitated by IgE receptor-bearing Langerhans' cells that promote allergen

Table 5. Comparison of Cutaneous Allergic Contact Reactions

	<i>Protein Contact Dermatitis</i>	<i>Allergic Contact Dermatitis</i>	<i>Contact Urticaria</i>
Clinical description	Eczematous dermatitis; rapid onset; sometimes urticarial plaques	Eczematous dermatitis	Urticarial plaques
Pathophysiology	Hypothetical (see text)	Type IV delayed-type cell-mediated hypersensitivity	Type I immediate IgE-mediated hypersensitivity
Allergen size	High molecular weight	Low molecular weight	Low molecular weight, high molecular weight ⁷²
Histology	Perivascular lymphocytic infiltrate with eosinophils; spongiosis with lymphocyte exocytosis ⁷³	Spongiotic dermatitis with lymphocyte exocytosis; perivascular lymphocytic infiltrate ⁵⁸	Dermal edema; perivascular lymphocytic and eosinophilic infiltrate ⁷⁴
Patch-test result	Negative	Positive	Negative
Prick/scratch-test result (early reading)	Positive	Negative	Positive
Prick/scratch-test result (delayed reading)	Sometimes positive	Negative	Negative
RAST result	Sometimes positive	Negative	Sometimes positive

IgE = immunoglobulin E; RAST = radioallergosorbent test.

capture, and the systemic expansion of Th2 memory T cells. Homing cutaneous lymphocyte antigen (CLA+) receptors on the surface of the Th2 cells induce the influx of inflammatory cells into the skin. There is an increase in cytokines interleukin (IL)-5, IL-4, IL-13, and IL-3; this causes eosinophilia, increases IgE, and increases the growth and development of mast cells.^{40–44} There is also a corresponding decrease in Th1 cytokines, mainly interferon- γ and IL-2.

Recently, a mouse model of protein contact dermatitis induced by natural rubber latex (NRL) provided some evidence that protein contact dermatitis may have a pathogenesis similar to that of atopic dermatitis. In this study, repeated application of NRL in mice caused a skin reaction similar to that of protein contact dermatitis. Immunologic analyses revealed an increase in the number of CD4⁺ CD3⁺ T cells and mast cells produced in this mouse model. Additionally, the epicutaneous sensitization with NRL favored a Th2-type response with the development of a strong IgE-mediated response. Others have shown that the application of the protein ovalbumin on nonsensitized animal integument results in the generation of specific IgE. When the application was repeated, a contact dermatitis that histologically resembled atopic dermatitis developed, which included the generation of T cells, the infiltration of eosinophils, and the production of IL-4 and IL-5.⁴⁵ In all, it is certainly plausible that protein contact dermatitis is a result of an IgE-mediated delayed hypersensitivity reaction similar to that of atopic dermatitis.⁴⁶

Differential Diagnosis

The clinical differential diagnosis of protein contact dermatitis includes allergic contact dermatitis, contact urticaria (immunologic or nonimmunologic), atopic dermatitis, and irritant contact dermatitis (Table 5). When the hands are involved, other types of hand dermatitis should also be considered.⁴⁷ In distinguishing protein contact dermatitis from other types of hand eczema, it is important to ask whether patients have associated immediate symptoms of burning, stinging, or itching. Certainly, patients with protein contact dermatitis may have more than one condition, and preexisting irritant or atopic dermatitis may be common. Tanaka and colleagues described simultaneous type I and type IV allergies to *Chrysanthemum*; low-molecular-weight sesquiterpene lactones were postulated to be the responsible allergens.⁴⁸ Patch testing should be performed to exclude allergic contact dermatitis. Immediate testing (prick test, scratch test, and RAST) with appropriate controls will identify type I hypersensitivity.

Treatment

As with all forms of allergic reactions, the first step in treatment is avoidance of the particular allergen. However, this may not always be practical or economically feasible for patients. Topical agents such as high-potency corticosteroids may decrease inflammation and accelerate the healing process. But side effects (including skin atrophy, purpura,

and possible alteration of intrinsic adrenocortical production) make high-potency steroids a poor long-term option. Topical tacrolimus 0.1% ointment is reported to have been used to successfully treat one individual with protein contact dermatitis from chicken viscera and another patient with protein contact dermatitis from halibut.⁴⁹ Neither patient responded to 2 months of topical clobetasol propionate, but both achieved clearing with topical tacrolimus within 1 week. The mechanism of action in these patients was attributed to (1) decreased expression of high-affinity Langerhans' cells and epidermic dendritic cells and (2) inhibited stimulation of Langerhans' cells.

Summary

Protein contact dermatitis is now recognized as being distinct from contact urticaria and allergic contact dermatitis. It most often presents as a chronic hand dermatitis that gives positive results in open application tests, prick tests, and scratch tests, and negative results in patch tests. Common allergens include raw meat, fish, vegetables, plants, and spices. Butchers, chefs, fishermen, and veterinarians are most often affected. A history of atopy is observed in about 50% of affected individuals. Respiratory symptoms and rhino-conjunctivitis are more common in patients with protein contact dermatitis from grains or enzymes. The pathogenesis of protein contact dermatitis is unclear, but it may be a combined type I and IV hypersensitivity reaction or perhaps be due to IgE-bearing Langerhans' cells, similar to what is observed in atopic dermatitis. Further immunologic studies and a better characterization of the specific proteins causing protein contact dermatitis will provide greater insight into the pathogenesis of this condition. It is important for dermatologists to consider this entity in the clinical differential diagnosis of chronic hand dermatitis, especially in patients with high-risk occupations.

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