THE CLINICAL PICTURE IS COMPATIBLE WITH CONTACT DERMATITIS? OCCURS IN THE WORKPLACE EXPOSURE TO pdf

1: Contact dermatitis | American Academy of Dermatology

Exposure in the workplace is responsible for a wide range of cutaneous problems, as summarized in Table www.enganchecubano.comt dermatitis, however, accounts for 90% of all cases of occupational dermatoses [1, 2].

It occurs in a minority of individuals and is caused by chemical or biological agents that are otherwise innocuous to the vast majority of people. The sequence of events that generate visible dermatitis is a biphasic process. These chemicals are incomplete antigens, or haptens, that must be captured by APCs, internalized, bound to proteins of the major histocompatibility complex, and reexpressed at the cell surface to become complete antigens. APCs migrate to local lymph nodes, where they present the newly formed allergens to naive T cells. These lymphocytes subsequently undergo clonal proliferation and differentiation into CD4 and CD8 effector, suppressor, and memory cells that are liberated in the bloodstream and home for the skin. This process takes place over 10 to 15 days and rarely gives rise to visible skin lesions. These lymphokines induce proliferation of cytotoxic T cells and recruitment of macrophages. Within 8 to 48 hours, these effector cells and their proinflammatory cytokines will attack the epidermis and generate the clinical picture of dermatitis. Untreated, this process may go on for days or weeks, until suppressor cells that secrete mainly IL-4 and IL take over and inhibit the reaction [4]. Although clinical signs of ICD and ACD often overlap and cannot always be distinguished, ACD tends to manifest as acute to subacute dermatitis, with pruritus as its cardinal symptom. Acute lesions begin as pruritic erythematous and edematous, urticarial-looking plaques that become rapidly studded with vesicles and sometimes tense bullae. A clear serous exudate escapes when these blisters rupture. Erythema and edema are still present in the subacute stages, but vesiculation becomes less visible, replaced by erosions, oozing, crusting, and desquamation. In long-standing, chronic cases, the skin appears dry and rough, fissured, grayish, and thickened with increased skin lines, a process called lichenification. In rare cases, the morphology of the eruption may be different. Contact urticaria, as exemplified by natural rubber latex hypersensitivity, is an immediate, immunoglobulin E-mediated reaction characterized by transient edematous wheals without epidermal changes. Protein contact dermatitis, sometimes seen in food handlers, bakers, and veterinarians, begins as an urticarial reaction and is followed in a few days by an eczematous phase. Hypersensitivity reactions to strong allergens such as poison ivy or exotic woods sometimes present as widespread erythema multiforme with target lesions. Exposure to colour film developer is known to induce lichen planus-like lesions, characterized by flat-topped, slightly scaly, violaceous, and polygonal papules that coalesce to form irregular plaques. ICD from liquids such as water and detergents affects the fingertips and the web spaces. Allergy to rubber chemicals in gloves presents as dermatitis of the dorsal hand, whereas the palm is more often affected by allergy to solid objects. The hands may transfer irritants and allergens to distant sites such as the face. Airborne exposure to particulate matter, such as sawdust and fibreglass, or the smoke, fumes, and vapours of volatile chemicals causes lesions on the face, upper eyelids, ears, scalp, neck, and other exposed areas, sometimes infiltrating clothes. In general, involvement of covered areas, genitals, or feet is not suggestive of occupational origin, but exceptions do occur: The very fine sawdust generated by sanding exotic woods is very pervasive and can cause lesions that are more prominent in areas of friction from clothes such as body folds and genitals. At times, the pattern of the dermatitis suggests the cause. Linear streaks of papules and vesicles are characteristic of phytodermatitis, whereas photocontact dermatitis will affect areas exposed to light, sparing the upper eyelids and submental and retroauricular areas. ICD tends to remain localized to the area of contact, whereas ACD has a propensity to spread to more distant sites, either by a process known as autoeczematization or through the phenomenon of systemic contact dermatitis. The latter occurs when an individual previously sensitized by cutaneous exposure is exposed to the allergen orally or parenterally: The Offenders Irritants The vast majority of irritants are chemicals. Strong acids and alkalis, concentrated solutions of sodium hypochlorite, isothiazolinone biocides, the agricultural fungicide chlorothalonil, and aliphatic amine epoxy catalysts will cause immediate burns on skin contact. Weaker agents, such as soap, detergents, solvents,

and water, will slowly damage the epidermal barrier and cause dermatitis only after cumulative exposure. Fine or coarse particles of sand, sawdust, metal filings, or plastic may be blown on exposed surfaces and cause mechanical irritation. Tiny fibreglass needles penetrate deeply in the skin and create an intensely itchy dermatitis that mimics scabies. Plants have husks, thorns, and spines that produce foreign body granulomas. Other plants, such as dieffenbachias, philodendrons, agaves, and daffodils, contain high levels of oxalic acid responsible for the epidemic of dermatitis in gardeners and florists. Plants of the Apiaceae eg, celery, carrot, parsnip, fennel and Rutaceae citrus fruits families contain phototoxic psoralens. Skin contact with the sap or juice of these plants, followed by sunlight exposure, will cause an erythematous or bullous burn that heals with intense pigmentation [5]. Hexavalent chromium is present in cement, corrosion-inhibiting primer paints, and coolants and is used to tan leather. Cobalt and nickel, the most common contact sensitizer, are ubiquitous in the metalworking industry. Mercury from amalgam is a hazard in the dental profession. Gold allergy, once thought to be rare, is now detected with increasing frequency among jewellers, dentists, and electronic technicians.

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2: Skin Allergies | www.enganchecubano.com

The suspicion that dermatitis has been caused by exposure to an exogenous trigger is formed on the basis of allergen and/or toxin exposure and the clinical picture. In irritant contact dermatitis, the trigger is usually exposure of the skin to an irritant, such as frequent or prolonged contact with water, solvents and cleaning agents, dust, etc.

Dermatitis is a localized inflammation of the skin. In general, inflammation refers to a condition in the body when it is trying to react to a localized injury of tissues. Signs of inflammation include some or all of the following: Occupational irritant contact dermatitis is an inflammation caused by substances found in the workplace that come in direct contact with the skin. Signs of irritant contact dermatitis include redness of the skin, blisters, scales or crusts. These symptoms do not necessarily occur at the same time or in all cases. This kind of dermatitis is caused by chemicals that are irritating e. Another kind of contact dermatitis, allergic contact dermatitis , is different because it is an allergic response to skin contact with some allergy-causing material e. Another difference is that allergic dermatitis can occur in other places on the body that did not come in contact with the allergy-causing material. How does irritant contact dermatitis develop? In the workplace, irritant contact dermatitis can develop after a short, heavy exposure or a repeated or prolonged, low exposure to a substance. The appearance of irritant contact dermatitis varies considerably according to the conditions of exposure. For example, an accidental contact with a strong irritant causes immediate blisters. Contact with a mild irritant may only produce redness of the skin. However, if the irritation continues, small lesions or sores appear on the reddened area; afterwards crusts and scales form. The skin damage usually heals a few weeks after exposure ends if no complications have arisen e. The irritant action of a substance depends on its ability to change some properties of the outer layer of the skin that acts as a protective barrier against toxic substances. Among other changes, some substances can remove skin oils and moisture from the outer layer of the skin. This reduces the protective action of the skin and increases the ability of irritants to enter or infiltrate the skin. The removal of fat or fat-like material from the skin is also responsible for the dryness, cracking and whitening of the skin. To produce the damage, the irritant substance must infiltrate the outer layer of the skin. Following infiltration, the substance comes into contact with cells and tissues. The substance also reacts with certain chemicals naturally present endogenous in cells and tissues. These reactions produce skin damage. The cells and tissues try to repair the damage and set up a defensive response to remove the invading material causing the damage. Minimal skin damage, as in the thickening of the inner layer of the skin, will not be visible. However, when the damage is severe, the skin shows signs of chapping, scaling, and blistering. Some skin cells also die. Typically, an irritant reaction develops within a few hours from exposure and is at its worst after approximately twenty-four hours. What are factors contributing to irritant contact dermatitis? Factors contributing to irritation include the chemical properties of the substance for example, is it an acid, an alkali, or a salt , the amount and concentration of chemical coming in contact with the skin, and the length and frequency of the exposure. Factors peculiar to individual workers are also important. Hereditary factors influence the variety of reactions seen in different persons when exposed to the same irritant. The part of the body that comes in contact with an irritant substance is another factor to remember. The penetration of substances varies over different body regions. For example, some substances penetrate the face and the upper back more quickly than the arms. Environmental factors play a significant role. For example, hot, humid workplaces cause workers to sweat. Sweat can dissolve some types of industrial chemical powders that may come in contact with the skin. This increases their toxic or irritant effects of the chemicals because solutions penetrate the skin more readily than solids. On the other hand, sweating may also have a protective function because it may dilute or wash out substances. Working where the air humidity is low or where the skin is wet for prolonged and repeated periods can cause chapping of the skin that, in turn, can increase the possibility of irritation. Friction against the skin, occurring while operating grinding machines and other equipment can scrape away the skin, reducing its protective action against irritants. Clothing soaked with irritants is another

important factor. Cuts or skin injuries enable irritant substances to penetrate the skin more readily. Table 1 summarizes some factors that contribute to skin irritation.

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3: CDC - Skin Exposures and Effects - NIOSH Workplace Safety and Health Topic

4/7 of these need to be met 1. Is the clinical picture consistent with an occupational dermatosis? 2. Is there an exposure at the workplace to an agent capable of causing the dermatosis?

In , for example, the NACDG carried out a retrospective study on the relationship of occupation to contact dermatitis. Therefore, ACD has an important public health effect, based on its frequency, and its significant economic, social, and emotional impact for the affected patient. Notice the positive patch-test reaction to colophonly rosin. Relevant positive reactions to acrylates where noted during patch testing. Age Over the past decade, multiple studies have recognized contact dermatitis as an important cause of childhood dermatitis, and a common diagnosis among children; being equally as likely in childhood as in adulthood, 21 , 22 although the most common allergens identified differ between the age groups. On the other hand, although fragrance mix allergy is an important sensitizer in all ages, certain studies, such as the Augsburg study, which was based on adults aged 28â€”75 years, have shown a significant increase in fragrance mix allergy with increasing age. Similarly, a recent Danish study demonstrated the prevalence allergy to preservatives being higher among those aged 41â€”60 years. When the human repeat-insult patch testing method was used to assess induction rates for ten common allergens, women were more often sensitized to seven of the ten allergens studied. When looking specifically at nickel sensitivity, the same study showed that the prevalence was much higher among women than men This might be due to the fact that numerous studies have demonstrated that pierced ears are a significant risk factor for development of nickel allergy. The role of race, if any, in the development of ACD to some potent allergens such as para-phenylenediamine PPD , remains controversial. With regard to the patch-test protocol, the evaluation of positive reactions may be slightly more difficult in darker skin types Fitzpatrick types V and VI , as erythema may not be as obvious, posing the risk of overlooking a mild positive allergic reaction. Finally, the darker the skin, the more difficult it is to mark the patch-test site after removal. Such immunological reaction, results from exposure and subsequent sensitization of a genetically susceptible host, to an environmental allergen, which on reexposure triggers a complex inflammatory reaction. The resulting clinical picture is that of erythema, edema, and papulo-vesiculation, usually in the distribution of contact with the instigating allergen, and with pruritus as a major symptom Fig. This is an important distinction to irritant contact dermatitis ICD in which no sensitization reaction takes place, and the intensity of the irritant inflammatory reaction is proportional to the doseâ€”concentration and amount of the irritant. In ACD, only minute quantities of an allergen are necessary to elicit overt allergic reactions. There are two distinct phases in the development of ACD: The unprocessed allergen is more correctly referred to as a hapten. Once the hapten penetrates the skin, it binds with epidermal carrier proteins to form a haptenâ€”protein complex, which produces a complete antigen. The antigen-presenting cell then migrates via the lymphatics to regional lymph nodes where it presents the HLA-DRâ€”antigen complex to naive antigen-specific T cells that express both a CD4 molecule that recognizes the HLA-DR and more specifically a T-cell receptor CD3 complex that recognizes the processed antigen. The antigen can also be presented in the context of the MHC class I molecules, in which case it would be recognized by CD8 cells. Subsequently, the naive T cells are primed and differentiate into memory also referred to as effector T cells which undergo clonal expansion, acquire skin-specific homing antigens, and emigrate out of the lymph node into the circulation. Such rechallenge can occur via multiple routes, including transepidermal, subcutaneous, intravenous, intramuscular, inhalation, and oral ingestion. This localized proinflammatory state results in the classical clinical picture of spongiotic inflammation redness, edema, papules and vesicles, and warmth. Recent advances in the knowledge of the pathophysiology of ACD have demonstrated the important role of the skin innate immunity in the sensitization process; have revisited the dogma that Langerhans cells are mandatory for ACD; and have addressed the nature, mode, and site of action of the regulatory T cells that control the skin inflammation Box see also Chapter Recent studies suggest that Langerhans cells LC that have been credited with an

indispensable role in ACD may not be essential for the development of contact hypersensitivity. Loss of T-reg cell activity may play a role in chronic inflammation. Mast cells appear to be pivotal in determining the magnitude of the inflammatory reaction. Clinical Approach An algorithmic approach to the patient is described in the following sections. Consideration of the Diagnosis The character and distribution of the dermatitis should raise the index of suspicion for ACD. Therefore, any patient who presents with an eczematous dermatitis should be regarded as possibly having ACD Fig. Additionally, one must also consider contact allergy in patients with other types of dermatitis e. For that reason, ACD should always be in the differential of eczematous lesions surrounding leg ulcers. These were described by Marks and DeLeo and include the following: Figure Typical appearance of eczematous dermatitis compatible with ACD. Note the erythematous scaly plaques with some fissuring on the hands. Erythematous papules, scattered on the extensor forearms. This is a typical picture of contact allergy to a skin care product ingredient. Figure Stasis dermatitis is a risk factor for the development of contact allergy. This is likely because of more frequent application of products that contain contact allergens to this area. Products without high frequency positive allergens are preferred in this area. ACD is not always bilateral even when the antigen exposure is bilateral i. Even when exposure to an allergen is uniform e. ACD can and does affect the palms and the soles. History Taking The first step in the diagnosis of ACD is a careful medical and environmental exposure history. History taking should begin with a discussion of the present illness focusing on the site of onset of theproblem and the topical agents used to treat the problem including over the counter and prescription medications. A past history of skin disease, atopy, and general health should be routinely investigated. This is followed by a detailed history of the usage of personal care products soap, shampoo, conditioner, deodorant, lotions, creams, medications, hair styling products, etc. The occupation should be ascertained as well, and if it appears contributory, or there are potential allergenic exposures, then a thorough occupational history should be taken. Occupations requiring frequent hand washing, glove use, or frequent chemical exposure should be prime suspects, among others. Clinical Manifestations Cutaneous Findings The classic presentation of ACD is a pruritic, eczematous dermatitis initially localized to the primary site of allergen exposure. Geometric or linear patterns or involvement of focal skin areas, may also be suggestive of an exogenous etiology Fig. A linear or streaky array on the extremities, for example, often represents ACD from poison ivy, poison oak, or poison sumac. Occasionally, the actual sensitizing substance in these plants, an oleoresin named urushiol may be aerolized when the plants are burned, leading to a more generalized and severe eruption on exposed areas such as the face and arms. Transfer of the resin from sources other than directly from the plant such as clothes, pets, or hands may result in rashes on unexpected sites i. Thus, relevant historic data gathered from thoughtful questioning may prove as useful as the distribution of the lesions. It is important to note that lesions of ACD will vary morphologically depending on the stage of the disease. For example, during the acute phase, lesions are marked by edema, erythema, and vesicle formation. As the vesicles rupture, oozing ensues and papules and plaques appear. Stronger allergens often result in vesicle formation, whereas weaker allergens often lead to papular lesion morphology, with surrounding erythema and edema. Subacute ACD on the other hand, will present with erythema, scaly juicy papules and weeping; whereas chronic ACD can present with scaling, fissuring, and lichenification. A key symptom for allergy is pruritus, which seems to occur more typically with allergy, than a complaint of burning. Lichenoid ACD is considered a rare variant. Clinical features mimic lichen planus and has been associated with metallic dyes in tattoos. Oral lichenoid ACD from dental amalgams can resemble typical oral lichen planus. Pigmented ACD has been mainly described in populations from Asian ethnicity. Lymphomatoid ACD is based only on histopathological criteria presence of significant dermal infiltrate displaying features of pseudolymphoma. Clinical signs which are nonspecific include erythematous plaques, sometimes very infiltrated, at the site of application of the contact allergen. Some examples include allergy to metal, allergy to hair dye, and to dimethylfumarate, a mold inhibitor found in sachets within some furniture implicated in causing a severe epidemic of ACD. Topographic Approach Dermatitis distribution is usually the single most important clue to the diagnosis of ACD. Typically, the area

of greatest eczematous dermatitis is the area of greatest contact with the offending allergen s. Notably, facial, eyelid, lip, and neck patterns of dermatitis should always raise suspicion of a cosmetic-related contact allergy. However, for all of these presentations, correct identification of the culprit chemical s will still require patch testing, since even the most astute and experienced clinician is, for the most part, unable to properly surmise the positive allergen s prior to testing. The pattern of dermatitis should be mainly used in determining whether or not to patch test, and which allergens and screening series to test. Figure ACD to para-phenylenediamine. Notice the eczema on the distribution of the hairline and behind the ears. Dermatitis on the forehead where the bangs came in contact with the skin of the same patient. It will darken the patch-test site. Occasionally, the topographic approach does not hold, and the distribution can actually be misleading. Ectopic ACD can follow two circumstances: Auto transfer, in which the allergen is inconspicuously transferred to other body sites by the fingersâ€”the classical example being nail lacquer dermatitis located on the eyelids or lateral aspects of the neck; and heterotransfer, in which the offending allergen is transferred to the patient by someone else spouse, parent, etc. A discussion of allergens in the context of common patterns of presentation is briefly detailed below. Face The face is a common site for ACD. Among patients with facial dermatitis, women are more commonly affected than men, particularly by cosmetic-associated allergens such as fragrances, PPD, preservatives, and lanolin alcohols eFig. In addition to allergens found as ingredients in cosmetics, products used to apply themâ€”such as cosmetic sponges, have also been reported to produce facial dermatitis in rubber-sensitive patients. Nevertheless, patients exquisitely sensitive to certain ingredients in hair products such as PPD or glyceryl monothioglycolate may show a marked scalp reaction with edema and crusting. PPD is one of the most potent sensitizers known and is widely used as an ingredient in hair dyes. In general, PPD sensitization manifests on the face and scalp of female adult patients who had contact with a hair dye. Allergic sensitivity to GMT can manifest as intense scalp reactions with scaling, edema, and crusting. Transfer of small amounts of allergens used on the scalp, face, or hands can be enough to cause an eczematous reaction of the eyelids, while the primary sites of contact remain unaltered eFig. Similarly, volatile agents may affect the eyelids first and exclusively, causing airborne eyelid contact dermatitis. Sources of contact dermatitis of the eyelids include cosmetics such as mascara, eyeliners and eye shadows, adhesive in fake eyelashes, and nickel and rubber in eyelash curlers. Furthermore, marked edema of the eyelids is often a feature of hair-dye dermatitis. Topical antibiotics like bacitracin and neomycin and certain metals such as gold 57 can also cause eyelid contact dermatitis. In fact, in the NACDG analysis of contact allergens associated with eyelid dermatitis, 58 gold was the most common allergen accounting for pure eyelid dermatitis. Notably, it has been observed that upon contact with hard particles such as titanium dioxide used to opacify facial cosmetics, and in sunscreens as a physical blocker of ultraviolet light , gold found in jewelry may abrade, resulting in the release of gold particles that can then make contact with facial and eyelid skin, causing dermatitis. Culprit allergens may include gold, fragrances, and preservatives. Lips According to an NACDG study, approximately one-third of patients with cheilitisâ€”without other areas of dermatitisâ€”are typically found to have an allergen as a contributing factor.

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4: Occupational Skin Disease - - American Family Physician

Occupational contact dermatitis accounts for 90% of all cases of work-related cutaneous disorders. It can be divided into irritant contact dermatitis, which occurs in 80% of cases, and allergic contact dermatitis. In most cases, both types will present as eczematous lesions on exposed parts of the.

Open in a separate window Histological analysis of a skin biopsy is indicated in all cases showing atypical symptoms or clinical course. Patient history includes questions relating to the development of the dermatitis and allergen exposure, as well as an assessment of causality. Once patch test results are available, questions relating to allergen exposure often need to be repeated in a second patient history. Due to the complexity of possible types of exposure, supplementary questionnaires to aid patient history taking have been developed for a number of occupations [43 , 44 , 45 , 46 , 47 , 48 , 49 , 50]. In irritant contact dermatitis, the trigger is usually exposure of the skin to an irritant, such as frequent or prolonged contact with water, solvents and cleaning agents, dust, etc. The diagnosis of allergic contact dermatitis is made by detecting contact sensitization to causative allergens by means of patch testing. A detailed description of how to perform patch tests and evaluate their relevance is given in the relevant DDG guidelines [51]. It is essential to: If no plausible result is achieved using a conventional patch test despite suspected contact allergy, modified patch testing methods are considered [52 , 53 , 54 , 55 , 56 , 57 , 58] Tab. For certain substances e. Delayed readings over several days are necessary Prick testing or intracutaneous testing can also be helpful in the case of suspected protein contact allergy; again, delayed readings are required Open in a separate window The methods described in Tab. The diagnosis of contact dermatitis is based on patient history, clinical examination, and skin testing. Additional investigations may be necessary. Performing these tests is technically challenging and the methods are poorly standardized; thus, LTTs should remain the reserve of specialist laboratories that have particular experience with these test methods and the interpretation of their results. In the absence of a critical evaluation of LTT results in comparison with patch test results, possibly also a repeated open application test ROAT or exposed control person, their relevance is questionable and should not form the basis for prophylactic or therapeutic measures [60 , 61 , 62]. In exceptional cases involving very strong patch test reactions to para-phenylenediamine PPD , LTTs can be helpful in preventing reactions due to cross-sensitization in further testing [61 , 63]. Other in vitro methods for the diagnosis of contact allergies are not validated. Whether the often poor specificity of LST for the analysis of metal compounds can be attributed to non-optimized conditions is unclear. Especially good correspondence between LST and patch testing is achieved for nickel sulfate in particular. However, from a dermatological point of view, there is no clinical indication to favor the complex in vitro test that is not validated for most allergens over the patch test, thereby leaving the real value of the LST in relation to contact allergens squarely in the domain of scientific investigations and further development of the test system. LTT LST is indicated in scientific, however generally not in clinical investigations of contact allergies. There is currently no useful diagnostic test for the direct identification of irritant contact dermatitis [64]. Alkaline resistance testing, the Nitrazine yellow swab test, or measuring transepidermal water loss do not represent reliable diagnostic aids. Thus, the diagnosis of irritant contact dermatitis is made on the basis of patient history and clinical picture â€” once possible causal contact sensitization has been excluded â€” and can be indirectly confirmed by subsequent resolution following cessation of toxin exposure. Treatment Patient information The successful treatment of contact dermatitis requires patient cooperation. The information provided to the patient and their mastery of the treatment, as well as care and protection measures, can contribute significantly both in terms of treatment and prophylaxis, particularly where occupation-related dermatitis triggers are relevant [65 , 66 , 67]. Avoiding the noxa Contact dermatitis is triggered by exogenous toxins in the vast majority of cases. The most important therapeutic approach, therefore, is to cease causal exposure â€” no form of symptomatic treatment can substitute for this approach. Attempts to induce tolerance to contact allergens by means of

immunotherapy have been hitherto unsuccessful [7 , 68]. These measures need to be tailored to the individual situation toxic substance, type of exposure. Prolonged use of gloves should be avoided due to their occlusive effect, although these effects are apparently milder than originally assumed [71]. Adjuvant use of suitable skin barrier creams can be helpful [72 , 73]. The selection of gloves and barrier creams should be made on the basis of their efficacy against the relevant toxins [74]. Dietary measures can be helpful in cases where a systemic hematogenous triggering of contact dermatitis in the setting of high-grade sensitization to an orally-ingested contact allergen is diagnosed as evidenced by patient history, patch testing, exclusion diet, and diagnostic provocation. Under this premise, a low-nickel diet may improve symptoms in individuals allergic to nickel [75 , 76 , 77 , 78], whilst chelating agents have also been described as helpful [79 , 80]. Avoiding the diagnostically determined noxa e is crucial. Symptomatic treatment of contact dermatitis Topical treatment is generally sufficient. As with other inflammatory dermatoses, the base in which the active substance is applied must be tailored to the severity of the dermatitis. Acute dermatitis is generally moist and needs to be treated with a hydrophilic preparation gel, lotion, cream , whereas chronic disease is more likely to require a water-in-oil-based preparation ointment. Needless to say, the base should not contain any allergens that may be relevant to the patient. Corticosteroids The efficacy of topical treatment with class-II or -III corticosteroids in acute allergic contact dermatitis is undisputed [81]; stronger preparations are required only in exceptional cases. However, weaker preparations at least do not always produce any detectable effect in irritant contact dermatitis [53]. The selection of a suitable corticosteroid with the appropriate efficacy should be made on the basis of the localization of skin lesions, as well as the severity and acuteness of the dermatitis, whilst bearing the therapeutic index in mind [82 , 83]. Where long-term therapy is indicated, preparations bearing low risk of atrophy e. The general principles governing the use of corticosteroids apply equally to their use in the treatment of contact dermatitis. The known side effects of topical treatment must be borne in mind when deciding upon the type and duration of treatment. Hence, topical corticosteroids represent the medication of first choice for the symptomatic treatment of contact dermatitis. Calcineurin antagonists In Germany, Austria, and Switzerland, calcineurin antagonists are only approved for the treatment of atopical dermatitis. They are less effective than strong corticosteroids in manifest contact dermatitis [87 , 88 , 89 , 90 , 91 , 92 , 96]. However, if long-term use is indicated, topical calcineurin antagonists may be beneficial in contact dermatitis compared to corticosteroids, particularly in sensitive areas of the skin e. With regard to safety, the reader is referred to the AWMF guidelines of the DDG on topical calcineurin antagonists and neurodermatitis [85 , 94]. In some forms of hand dermatitis, topical application of psoralens is advisable in the context of PUVA therapy in order to intensify the therapeutic effect. Positive data are also available on the use of UVA1 and narrow-band UVB, particularly in hand dermatitis [, ,]. Other external agents Due to its antiphlogistic and antiproliferative effects, the use of coal tar as a follow-up treatment is still reasonable today in cases where other external agents are ineffective or declined by the patient. There is no evidence to support the fear that local treatment with coal tar is carcinogenic [, , , ,]. However, the known side effects of coal tar treatment skin irritation and discoloration, acnegenic effect, photosensitization must be borne in mind. Antiseptic agents such as triclosan, polyhexanide, octenidine, etc. Iontophoresis can be beneficial in dyshidrotic dermatitis [ ]. Soft X-ray therapy and Grenz ray therapy have proven to be helpful in the treatment of dermatitis [, , ,]. However, due to the harmful cumulative effects of X-rays to the skin, these methods are in principal contraindicated today and only justified in exceptional cases. The efficacy of topical non-steroidal antiphlogistic agents in contact dermatitis has not been sufficiently proven; in addition, there is a relevant risk of contact sensitization to these substances when used topically in dermatitis [,]. Although Bufexamac has had its approval withdrawn by the European Medicines Agency due to its sensitization potential, it is still available in Switzerland and outside Europe. Moreover, many other substances for which no published data on efficacy are available are nevertheless used and recommended for the treatment of dermatitis. The same is true for antihistamines. Systemic treatment Systemic treatment may become necessary in cases where local treatment is insufficiently effective. It is essential to take the specific side-effects profile of the agents used

into consideration. The usual rules on systemic administration of corticosteroids apply here. Systemic administration of alitretinoin may be helpful in chronic hand eczema [, , ,]. In this regard, the reader is referred to the guidelines on hand dermatitis [1]. Insufficient data is available to date on the long-term effects [ ]. Cyclosporine is currently the drug of first choice in the treatment of severe, therapy-resistant atopic dermatitis in adults, an indication for which it is approved. Long-term oral administration of cyclosporine A can be helpful in patients with therapy-resistant hand dermatitis [,]. Other immunomodulators, such as azathioprine, mycophenolate mofetil, or methotrexate are also used for atopic dermatitis off-label but only if cyclosporine is ineffective or contraindicated , and can also be considered for contact dermatitis [94 , ,]. Basic therapy and skin protection Follow-up treatment with basic moisturizing agents to promote skin barrier regeneration and protect against recurrence, combined with the use of skin protection creams, is beneficial when individually tailored to skin status and skin exposure [, ,]. On the other hand, preparations containing unsuitable levels of water and fat or allergenic components may delay the resolution of dermatitis or even intensify the effect of substances harmful to the skin [ ]. Although skin protection training is beneficial in the case of hazardous occupational exposure [ ], the effectiveness of skin protection creams alone under working conditions has not be unequivocally proven [ ]. Complete restoration of barrier function is not expected until several weeks after the clinical resolution of contact dermatitis. However, the beneficial effect of moisturizers is measurable [ ]. Evidence of therapeutic efficacy Only a small number of prospective, randomized, double-blind, controlled studies meeting current criteria have proven the efficacy of the contact dermatitis treatments mentioned here in sufficiently large patient populations. Relevant data supporting efficacy is only available for the use of topical corticosteroids and systemic administration of alitretinoin in hand dermatitis [84 , ,]. However, this does not mean by implication that the other treatment forms discussed here are ineffective. Although studies on conventional therapy methods in dermatitis may be lacking for many reasons, the long-term clinical experience of experts in terms of efficacy is undisputed. Individually tailored systemic therapy should be considered when topical therapy is either ineffective or unfeasible. Reporting dermatitis It is generally necessary to establish whether a case of contact dermatitis has been triggered by occupational exposure. However, if the reasonable suspicion of an occupational disease has already been confirmed, i.

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5: Occupational Contact Dermatitis

dermatitis, which occurs in 80% of cases, and allergic contact dermatitis. In most cases, both types will present as eczematous lesions on exposed parts of the body, notably the hands.

Diagnosis and treatment How dermatologists diagnose contact dermatitis To diagnose this common skin condition, dermatologists: Examine your skin, paying close attention to the rash. Ask about current and past health issues. Ask questions to help them determine what is causing the rash. Finding the cause can require a bit of detective work. Your dermatologist may ask you questions about your work, free time, pets, and skin care products. Be sure to tell your dermatologist about all cosmetics you use. If you or a close family member uses a skin lightener, be sure to mention this, too. If your dermatologist suspects that you have an allergy, patch testing may be recommended. This offers patients a safe and effective way to find out if your skin has developed an allergic reaction to anything. What happens during patch testing? If patch testing is recommended, the following will happen: Patches containing small amounts of substances to which you may be allergic will be applied to your skin, usually on your back. You keep the substances on your skin for a specific amount of time, usually 2 days. You may need to keep some patches on your skin for a longer time and see your doctor again in a few days. To find out if the allergen is causing your rash, you will need to avoid that substance. For example, if the test shows that you have a nickel allergy, you may need to: Stop wearing jewelry and clothing zippers, fasteners that contains nickel. Cover your cell phone with a case to avoid touching the metal. Get a pair of eyeglasses made without nickel. If your skin clears when you avoid the allergen, it is likely the cause of your rash. How dermatologist treat contact dermatitis Treatment is the same for both types of contact dermatitis. Avoid what is causing your rash. If avoiding the cause will be difficult, ask your dermatologist for help. For example, if you are allergic to latex but must wear exam gloves, your dermatologist can recommend another type of glove that you can wear. If you must work outdoors where poison ivy grows, your dermatologist can recommend a protective barrier cream and clothing that can help. Once you can avoid the cause, your rash should clear. To relieve your symptoms, a dermatologist may recommend the following: Antihistamine pills, moisturizer, and a corticosteroid that you can apply to your skin. Most patients apply the medicine twice a day for 1 week and once a day for 1 to 2 weeks. Oatmeal baths can relieve discomfort. If you have a lot of swelling, your face swells, or the rash covers much of your body, you may need a strong medication. Your dermatologist may prescribe prednisone. It is important to take this medication exactly as directed to avoid another flare. Wet dressings can help soothe skin that has lots of oozing and crusting. If you have an infection, your dermatologist may prescribe an antibiotic. Some patients need light therapy to calm their immune system. Your dermatologist may refer to this as phototherapy. If you avoid what caused the rash, your skin will clear. Most people see clear skin within 1 to 3 weeks. Clearing may take longer if poison ivy, poison oak, or poison sumac caused your rash. The first time you have an allergic reaction to one of these plants, the rash may linger for 6 weeks. If you get another rash, your skin should clear within 10 to 21 days. Once your skin clears, you must continue to avoid what caused your rash. If your rash does not clear, you should tell your dermatologist. You may need extra help. Systemic contact dermatitis Some people develop a rash even when they avoid touching the allergen. A rash can appear when the substance to which you have an allergy gets inside your body. The medical term for this condition is systemic contact dermatitis. The allergen can get inside your body in different ways. Eat food that contains the allergen Inhale or inject a medicine that contains the allergen Use birth control IUD or spermicide that contains the allergen While rare, some people develop a rash because the fillings in their mouth contain mercury. They could only get rid of the rash when a dentist replaced their fillings with fillings that did not contain mercury. Trying to find everything that contains the allergen can be a challenge. Your dermatologist may be able to help you create a list of things you need to avoid. The list often varies from region to region. Outcome for patients with contact dermatitis By avoiding what caused the rash, most people can avoid flare-ups. If you work with

substances that caused the rash, you can still avoid a rash. Your dermatologist can recommend ways to work and products to use. Image used with permission of Journal of the American Academy of Dermatology: American Academy of Dermatology. Last update July Hamann CR, Boonchai W. Katta R, Schlichte M. Nguyen JC, Chesnut G, et al. Saary J, Qureshi R.

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6: Contact and Occupational Dermatitis information. Patient | Patient

contact dermatitis, there is contiguous spread beyond the site of contact. Less frequently, there is a non-contiguous spread, and only very rarely is there a generalised spread of the allergic reaction (Fig. and).

Clinical Features Rash appears in areas exposed to the sensitizing agent, usually with an asymmetric or unilateral distribution. Sensitizing agent on the hands or clothes is often transferred to other body parts. The rash is characterized by erythema, vesicles, and severe edema. Pruritus is the overriding symptom. Latex allergic reactions range from pruritus to erythematous, weeping lesions. Exposure to latex, as well as some other substances, can proceed to anaphylaxis. In photoallergic dermatitis, sunlight and exposure to the offending substance usually affect the face and arms, but the sun-shaded area under the chin is generally spared. For example, allergic contact dermatitis caused by exposure to a member of the Rhus genus may be suspected in a patient who presents with vesicles or bullae arrayed in a linear fashion on the forearm two to three days after performing outdoor tasks in an area that contains poison oak or poison ivy. Direct patch skin testing is recommended for more definitive diagnosis and identification of the sensitizing agent. Skin testing can also be used to screen for an allergic cause of a dermatitis of long duration or unknown etiology, or to identify a suspected allergic component to irritant contact dermatitis. Photopatch testing with ultraviolet light should be used to diagnose photoallergic dermatitis. RAST measures specific immunoglobulin antibodies to sensitizing substances e. Controversy exists regarding the sensitivity and specificity of RAST compared with direct patch or scratch skin testing. The previously described general principles of treatment should be followed. Desensitization to agents such as Rhus antigens provides protection that is incomplete and lasts for no more than a few months; furthermore, desensitization must be repeated each year to maintain partial resistance. Clinical Features Comedones, pustules, and papules may be present. A key feature is the occurrence of these lesions in areas with exposure to oil-soaked clothing e. Occupational acne may also present as aggravation of existing acne, usually in the face or neck area. Secondary infection from bacterial folliculitis is common. Treatment and Prevention Patients with occupational acne should be advised to avoid contact with oils and greases. Frequent routine cleansing of the skin and daily washing of work clothes are necessary. If preventive measures are ineffective, routine acne therapy is indicated. Frequently, the skin tumors do not appear until two or three decades after the exposure. All patients should be provided with information on how to prevent skin cancer. Get immediate access, anytime, anywhere. Choose a single article, issue, or full-access subscription. Earn up to 6 CME credits per issue.

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7: Guideline contact dermatitis

The two commonest forms of occupational skin disease are irritant and allergic contact dermatitis. Morphology and history are used to establish the diagnosis and the relation of the disorder to work exposure, respectively.

References What is contact dermatitis? Occupationally related contact dermatitis can develop from frequent and repeated use of hand hygiene products, exposure to chemicals, and glove use. Contact dermatitis is classified as either irritant or allergic. Irritant contact dermatitis is common, nonallergic, and develops as dry, itchy, irritated areas on the skin around the area of contact. By comparison, allergic contact dermatitis type IV hypersensitivity can result from exposure to accelerators and other chemicals used in the manufacture of rubber gloves as well as from exposure to other chemicals found in the dental practice setting. Allergic contact dermatitis often manifests as a rash beginning hours after contact and, like irritant dermatitis, is usually confined to the areas of contact. Top of Page What is latex allergy? Latex allergy type I hypersensitivity to latex proteins can be a more serious systemic allergic reaction. It usually begins within minutes of exposure but can sometimes occur hours later. It produces varied symptoms, which commonly include runny nose, sneezing, itchy eyes, scratchy throat, hives, and itchy burning sensations. However, it can involve more severe symptoms including asthma marked by difficult breathing, coughing spells, and wheezing; cardiovascular and gastrointestinal ailments; and in rare cases, anaphylaxis and death. What are the categories of glove-associated skin reactions? Red, dry, itchy irritated areas Acute: Itchy, red rash, small blisters Acute: Hives, swelling, runny nose, nausea, abdominal cramps, dizziness, low blood pressure, bronchospasm, anaphylaxis shock Chronic: Dry, thickened skin, crusting, deep painful cracking, scabbing sores, peeling Chronic: Dry thickened skin, crusting, scabbing sores, vesicles, peeling appears 4â€”96 hours after exposure Chronic: Dental health care personnel experiencing contact dermatitis or latex allergy symptoms should seek a definitive diagnosis by an experienced health care professional e. Top of Page What are some considerations if dental health care personnel are allergic to latex? Dental health care personnel who are allergic to latex will need to take precautions at work and outside the workplace since latex is used in a variety of other common products in addition to gloves. If definitively diagnosed with allergy to natural rubber latex NRL protein: Avoid, as far as feasible, subsequent exposure to the protein and only use nonlatex e. Make sure that other staff members in the dental practice wear either nonlatex or reduced protein, powder-free latex gloves. Use only synthetic or powder-free rubber dams. Dental personnel can further reduce occupational exposure to NRL protein by taking the following steps: Using reduced protein, powder-free latex gloves. Frequently changing ventilation filters and vacuum bags used in latex contaminated areas. Checking ventilation systems to ensure they provide adequate fresh or recirculating air. Frequently cleaning all work areas contaminated with latex dust. Educating dental staff on the signs and symptoms of latex allergies. Top of Page Why are powder-free gloves recommended? Proteins responsible for latex allergies are attached to glove powder. When powdered gloves are worn, more latex protein reaches the skin. Also, when gloves are put on or removed, particles of latex protein powder become aerosolized and can be inhaled, contacting mucous membranes. As a result, allergic dental health care personnel and patients can experience symptoms related to cutaneous, respiratory, and conjunctival exposure. Dental health care personnel can become sensitized to latex proteins after repeated exposure. Work areas where only powder-free, low-allergen i. Top of Page What are some considerations for providing dental treatment to patients with latex allergy? Patients with a latex allergy should not have direct contact with latex-containing materials and should be treated in a "latex safe" environment. Such patients also may be allergic to the chemicals used in manufacturing natural rubber latex gloves, as well as to metals, plastics, or other materials used to provide dental care. By obtaining thorough patient health histories and preventing patients from having contact with potential allergens, dental health care professionals can minimize the possibility of patients having adverse reactions. Considerations in providing safe treatment for patients with possible or documented latex allergy include but are not limited to the following: Screen all patients for

latex allergy e. Be aware of some common predisposing conditions e. Be familiar with the different types of hypersensitivityâ€”immediate and delayedâ€”and the risks that these pose for patients and staff. Consider sources of latex other than gloves. Dental patients with a history of latex allergy may be at risk from a variety of dental products including, but not limited to, prophylaxis cups, rubber dams, and orthodontic elastics. Provide an alternative treatment area free of materials containing latex. Ensure a latex-safe environment or one in which no personnel use latex gloves and no patient contact occurs with other latex devices, materials, and products. Be aware that latent allergens in the ambient air can cause respiratory and or anaphylactic symptoms in people with latex hypersensitivity. Therefore, to minimize inadvertent exposure to airborne latex particles among patients with latex allergy, try to give them the first appointments of the day. Frequently change ventilation filters and vacuum bags used in latex-contaminated areas. Have latex-free kits e. Be aware that allergic reactions can be provoked from indirect contact as well as direct contact e. Hand hygiene, therefore, is essential. Communicate latex allergy procedures e. If latex-related complications occur during or after the procedure, manage the reaction and seek emergency assistance as indicated. Follow current medical emergency response recommendations for management of anaphylaxis. Reduction of latex aeroallergens and latex-specific IgE antibodies in sensitized workers after removal of powdered natural rubber latex gloves in a hospital. J Allergy Clin Immunol ; Allergy to Latex Rubber. The dental team and latex hypersensitivity. J Am Dent Assn ; Can a threshold limit value for natural rubber latex airborne allergens be defined? Journal of Allergy and Clinical Immunology ; Baur X, Jager D. Airborne antigens from latex gloves. Allergic reactions to latex among health-care workers. Mayo Clin Proc ; Preventing allergic reactions to natural rubber latex in the workplace. Questions and Answers about identifying and preventing latex allergy. Natural Rubber Latex Hypersensitivity: Management of dental patients with allergies to natural rubber latex. Gen Dent ;50 6: Allergic contact dermatitis in dental professionals: J Am Dent Assn ; 2: A prospective, controlled study showing that rubber gloves are the major contributor to latex aeroallergen levels in the operating room. Effect of powder-free latex examination glove use on airborne powder levels in a dental school clinic. J Dent Educ ; An epidemic of occupational allergy to latex involving health care workers. J Occup Environ Med ; How infection control procedures are affecting dental practice today. Its prevalence among dental professionals. Allergic reactions to rubber gloves in dental patients: Report of three cases. Br Dent J ; Snyder H, Settle S. Implications for the dentist. Quantification of occupational latex aeroallergens in a medical center. Control of airborne latex by use of powder-free latex gloves. Latex gloves use and symptoms in health care workers 1 year after implementation of a policy restricting the use of powdered gloves. Am J Infect Control ; Allergens in latex surgical gloves and glove powder. Latex allergy in hospital employees. Latex sensitivity among perioperative nurses.

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8: Allergic Contact Dermatitis | Plastic Surgery Key

Contact dermatitis is a rash that occurs at the site of exposure to a substance capable of producing an allegic or irritant skin response.; Contact dermatitis can be caused by noxious, irritating substances or substances to which the patient has developed a skin allergy.

Through the appendages Figure 3 Figure 1: Intercellular lipid pathway As shown in Figure 1, the stratum corneum consists of cells known as corneocytes. The spaces between the corneocytes are filled with substances such as fats, oils, or waxes known as lipids. Some chemicals can penetrate through these lipid-filled intercellular spaces through diffusion. Transcellular permeation As shown in Figure 2, another pathway for chemicals to be absorbed into and through the skin is transcellular, or cell-to-cell, permeation whereby molecules diffuse directly through the corneocytes. Through the appendages hair follicles, glands As shown in Figure 3, the third pathway for diffusion of chemicals into and through the skin is skin appendages i. This pathway is usually insignificant because the surface area of the appendages is very small compared to the total skin area. However, very slowly permeating chemicals may employ this pathway during the initial stage of absorption. Contact Dermatitis Contact dermatitis, also called eczema, is defined as an inflammation of the skin resulting from exposure to a hazardous agent. It is the most common form of reported OSD, and represents an overwhelming burden for workers in developed nations. Common symptoms of dermatitis include: Itching Redness Swelling The formation of small blisters or wheals itchy, red circles with a white centre on the skin Dry, flaking, scaly skin that may develop cracks Occupational contact dermatitis is frequently divided into two categories: Irritant contact dermatitis ICD is a non-immunologic reaction that manifests as an inflammation of the skin caused by direct damage to the skin following exposure to a hazardous agent. The reaction is typically localized to the site of contact. ICD may be caused by phototoxic responses e. Allergic contact dermatitis ACD is an inflammation of the skin caused by an immunologic reaction triggered by dermal contact to a skin allergen. For ACD to occur, a worker must be first sensitized to the allergen. Subsequent exposures of the skin to the allergenic agent may elicit an immunologic reaction resulting in inflammation of the skin. The reaction is not confined to the site of contact and may result in systemic responses. ACD may be caused by industrial compounds i. Because the symptoms and presentation of ICD and ACD are so similar, it is extremely difficult to distinguish between the two forms of contact dermatitis without clinical testing e. The severity of contact dermatitis is highly variable and depends on many factors including:

9: Dermatitis, Irritant Contact : OSH Answers

dermatitis are irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). The mechanisms and common causative agents vary for both ICD and ACD, but the clinical picture is often similar.

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