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Contact dermatitis: relevant differential diagnoses, simulators, and variants

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Summary

Similar to all eczematous disorders, irritant and allergic contact dermatitis are primarily defined by their morphology. In addition, the diagnosis of this specific reaction pattern on the skin (and mucous membranes) also requires prior exposure (direct or indirect) to an allergen or irritant. While it is quite easy to give a textbook description of the typical features of eczema, its clinical manifestations in daily practice are diverse and frequently uncharacteristic. Contact reactions may present as lichenoid, lymphomatoid, granulomatous, pigmented, purpuric, and erythema multiforme-like lesions, thus lacking the typical eczematous appearance and broadening the spectrum of differential diagnoses to be considered. Moreover, a considerable number of agents, acting by mechanisms other than contact, may trigger adverse reactions mimicking the eczematous morphology and distribution pattern of contact dermatitis. Increasingly used in oncology, this is especially true for drugs such as kinase inhibitors. Knowledge of these associations is crucial for the adequate diagnostic and therapeutic care of patients with supposed or actual contact dermatitis.

Introduction

Marked by its variable clinical appearance and etiology, eczema ranks among the most common skin diseases in the history of dermatology (at least since the Industrial Revolution), with prevalence rates of 15–20% in Germany.

Allergic contact dermatitis alone is thought to be equally prevalent as diabetes mellitus.

Marked by its variable clinical appearance and etiology, eczema ranks among the most common skin diseases in the history of dermatology (at least since the Industrial Revolution), with prevalence rates of 15–20% in Germany. Allergic contact dermatitis alone is thought to be equally prevalent as diabetes mellitus [1].

Moreover, its occurrence in all age groups, its high socioeconomic relevance as one of the most common reasons for occupational disease reporting, and the individual personal burden caused by pruritus and esthetic problems justify its special status. Given that the avoidance of triggering factors is crucial for successful therapy as well as the prognosis in all clinical variants of contact dermatitis, accurate diagnostic workup and correct etiological classification have to be considered essential.

Given that the avoidance of triggering factors is crucial for successful therapy as well as the prognosis in all clinical variants of contact dermatitis, accurate diagnostic workup and correct etiological classification are indispensable. A complicating aspect in this context is the fact that other disease entities – with a fundamentally different pathogenesis – may mimic the clinical appearance of contact dermatitis, thus requiring different therapeutic strategies.

Eczema is primarily defined by its morphology.

The disease entity “eczema” stands out among all dermatologic diseases because all primary as well as secondary lesions may appear in the course of this disorder.

But no matter how straightforward and simple textbook descriptions of the typical clinical features of contact dermatitis are, the clinical manifestations encountered in daily practice are frequently diverse and uncharacteristic. A complicating aspect in this context is the fact that other disease entities – with a fundamentally different pathogenesis – may mimic the clinical appearance of contact dermatitis, thus requiring different diagnostic and therapeutic measures [2]. The aim of this article is to draw attention to clinically significant variants of contact dermatitis and point out relevant differential diagnoses as well as simulators.

Definition

Eczema is primarily defined by its morphology: the skin shows a characteristic inflammatory intolerance reaction with consecutive and concurrent appearance of erythema, vesicles, exudation, papules, and scales, with the acuteness of the disease determining their respective proportions. The term “eczema” is sometimes replaced by the broader term “dermatitis”, with acute forms often designated as “dermatitis” and chronic forms as “eczema”. From a practical standpoint, however, the two terms may be used interchangeably [1].

While the aforementioned definition, firmly anchored in the literature as well as among practicing dermatologists, may sound simple and plausible for physicians and patients alike, it does carry an enormous implication for clinical practice: the disease entity “eczema” stands out among all dermatologic diseases precisely because all lesions – primary (erythema, macule, papule, plaque, nodus, vesicle, bulla, pustule, wheal) and secondary (erosion or excoriation, rhagades, ulcer, lichenification, scale, crust, scar, hyper- and hypopigmentation) – may appear in the course of this disorder.

In this context, it must be noted that the morphological diversity mentioned does not only depend on the acuteness (i.e. the course of time), but also on the body region affected as well as the immunological and physical/chemical quality of the triggering agent.

By nature and definition, contact dermatitis is primarily induced by external, noninfectious, immunologically, chemically or physically acting noxious agents. The mere clinical picture, however, frequently does not allow for an unambiguous diagnosis of allergic versus irritant contact dermatitis [1].

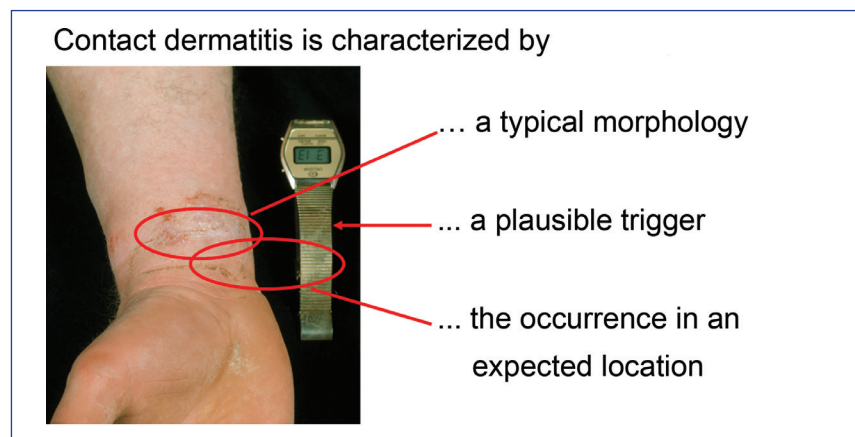


Figure 1 Clinical characteristics and prerequisites for the diagnosis of typical contact dermatitis.

Table 1 Important and frequent predilection sites of contact dermatitis relevant for differential diagnostic considerations (not intended to be exhaustive).

Differential diagnoses	Locations (predilection sites of the mentioned differential diagnoses; in case of suspected contact dermatitis in these areas, they should be considered)
All other types of eczema:	
▶ Atopic dermatitis	Flexor aspects, trunk, head
▶ Dyshidrotic eczema	Hands (esp. fingers), feet
▶ Asteatotic eczema	(Lower) extremities, trunk
▶ Seborrheic dermatitis	Face, decolleté, interscapular region
▶ Nummular (discoïd) eczema	Trunk, extremities
▶ Stasis dermatitis	Lower legs
Psoriasis and its variants	Scalp, elbows, fingers, trunk
Pityriasis rosea	Trunk
Erythrasma	Axillae and groin
Lichen planus (verruccous)	Lower legs, feet
Lichen simplex chronicus	Lower legs, forearms, hands, trunk
Bowen's disease, Erythroplasia of Queyrat	Face, neck, extremities, anogenital region
Paget's disease	Breast (mamilla)
Hailey-Hailey disease	Axillae and groin
Mycoses, tinea versicolor	Extremities, trunk
Cutaneous T-cell lymphomas, parapsoriasis en plaque	Trunk
Perioral dermatitis, rosacea-like dermatitis	Face (perioral, paranasal, forehead)
Polymorphic light eruption	UV-exposed areas
Scabies (eczematized)	Anogenital region, hands (interdigital), mamilla
Systemic lupus erythematosus, subacute cutaneous lupus erythematosus	Face, extremities, trunk
Delusions of parasitosis, dermatitis artefacta	Mostly extremities, also face

In summary, it is therefore almost astounding that the literature describes allergic and irritant contact dermatitis as examples of easily identifiable entities – particularly in relation to the various chronological stages. Even in well-established textbooks, interested readers may find passages such as “the nature of eczema is readily recognized by any physician”. Such an assessment, however, may lead to long diagnostic and therapeutic detours in the daily care of dermatologic patients. Figure 1 lists the fundamental characteristics of typical contact dermatitis.

Differential diagnoses of contact dermatitis

The basis of differential diagnostic considerations is the presence and recognition of an “eczematous appearance” in affected patients.

The basis of differential diagnostic considerations is the presence and recognition of an “eczematous appearance” in affected patients. Thus, in a first step, the morphology “eczema” leads to those differential diagnostic entities that may also be characterized by an “eczema-like” appearance (Figure 2). The most important,



Figure 2 Suspected occupational contact dermatitis in a right-handed female patient treated with topical corticosteroids for weeks. Eventually, the diagnosis dermatomycosis was confirmed (tinea incognito caused by *Trichophyton rubrum*).

epidemiologically significant entities are listed in Table 1 (modified according to [3]). The list is not intended to be exhaustive.

An important factor in the context of the differential diagnostic workup of eczema is time. Thus, if a reliably (!) acute onset (within hours to a few days) of eczematous lesions has been established, the following diseases listed in Table 1 are (rather) unlikely: verrucous lichen planus, lichen simplex chronicus, Bowen's disease, Paget's disease, cutaneous T-cell lymphomas, parapsoriasis, (systemic) lupus erythematosus.

However, to complicate matters even further, contact reactions may also take a noneczematous course. In such cases, differential diagnostic considerations are based on the prevailing morphology (lichenoid, granulomatous, pigmented, and others), as will be explained below.

The second fundamental prerequisite for a skin reaction to be regarded as contact dermatitis includes – based on the distribution pattern of the eczematous lesions – the plausibility, or at least possibility, of contact-induced lesions.

The second fundamental prerequisite for a skin reaction to be regarded as contact dermatitis includes – based on the distribution pattern of the eczematous lesions – the plausibility, or at least possibility, of contact-induced lesions. A complicating aspect in this context is the fact that allergic contact dermatitis may possibly not be induced by direct contact with the triggering allergen but via hematogenous or airborne contact [1].

As potential airborne allergens have been discussed in comprehensive studies and reviews in the literature, only a few – those most relevant for daily clinical practice – shall be mentioned here [4, 5]: acrylates, epoxy resins, isothiazolinones (particularly methylchloroisothiazolinone), formaldehyde and formaldehyde releasers, glutaraldehyde, Compositae plant extracts, rosin, primin, propolis.

Primarily hematogenously acting contact allergens include nickel sulfate, chromium compounds, parabens, balsam of Peru, and a variety of pharmaceutical agents [6–10].

The importance of taking a thorough patient history (including allergological and occupational aspects) cannot be overemphasized here [11].



Figure 3 Erythema multiforme-like contact reaction with typical target lesions on the upper arm. This reaction was caused by the antiinflammatory agent bufexamac, which was taken off the EU market by the EMA in 2010 because of the agent's high potential for contact dermatitis (note: bufexamac is still commercially available in Switzerland).

Unusual manifestations of contact reactions

Apart from the above-mentioned typical eczematous morphology and clinical presentation, both irritant and allergic triggers may cause various other cutaneous reaction patterns. The current S1 guidelines “Contact Dermatitis” lists the clinically most important noneczematous manifestations of contact allergic reactions [1]:

- ▶ Erythema multiforme-like reactions after contact with topical pharmaceutical agents, herbal allergens, or textile dyes. Among these drugs, topical antiinflammatory agents are particularly important, including bufexamac, which was taken off the EU market by the EMA in 2010 (Figure 3) [12–14]. With regard to this substance, it should be noted that it is still commercially available in Switzerland. This is well known in relevant Internet forums. Thus, there are still patients presenting with contact reactions to this substance supposedly nonexistent in the EU.
- ▶ Pigmented purpura, pigmented contact dermatitis due to dyes (especially disperse colors), and rubber allergens [15–17].
- ▶ Lichen planus-like and lichenoid contact reactions of the mucous membranes to dental allergens, for example, amalgam metals [18].
- ▶ Bullous, papulonodular and pustular reactions in particular to metals or metal salts [2].
- ▶ Granulomatous reactions, for example, to metal salts in tattoo dyes. Recent evidence suggests that a histological examination be done, as histologically proven sarcoidal granulomas in such reactions may be indicative of existing or evolving sarcoidosis [19].
- ▶ Lymphomatoid reactions, for example, to tattoo pigments [20].



Figure 4 Clinical picture resembling mechanically induced skin trauma. In this case, however, the diagnosis was irritant contact dermatitis caused by body fluids. The report by EMS read: “The physician (called by police) found the patient lying in bed... must have been there for about 20 days ... Bed linen and mattress were soaked with approximately 4 liters of foul-smelling brownish fluid ...”

Atypical manifestation patterns of contact dermatitis include erythema multiforme-like reactions, bullous, nodular, granulomatous reactions, lichen planus-like reactions, lymphomatoid reactions, pigmented reactions.

- ▶ Primarily edematous reactions caused by, for example, PPD or azo dyes; sometimes to be considered as differential diagnosis in angioedema [21].

If any one of these reaction patterns is present, the differential diagnoses considered on the basis of the prevailing morphology should be worked up or ruled out. However, not only allergic but also irritant (toxic) reactions may be noneczematous. Depending on the chemical properties of the triggering substance, its exposure time, the mode of exposure (occlusive, nonocclusive), and the site of the body where contact takes place, clinical manifestations may result that are not immediately suggestive of contact dermatitis (Figures 4, 5). Via an irritant-inflammatory mechanism (that is considered essential for the therapeutic effect), new topically applied immunomodulatory agents such as imiquimod, used in the treatment of actinic keratoses, may induce reaction patterns that are sometimes reminiscent of herpes infections (Figure 6).

Simulators of contact dermatitis

Unlike the above-mentioned differential diagnoses, the following section on simulators of contact dermatitis will include only those clinical entities that meet the aforementioned two essential prerequisites for a suspected diagnosis of “contact dermatitis”: (1) clinically, there is an eczematous appearance, (2) based on the distribution pattern (apparent or actual), symptoms are triggered by contact; thus, reactions are usually localized.

Relatively common, the induction of otherwise barely symptomatic or asymptomatic psoriasis by mechanical (or chemical) triggers (Koebner phenomenon) is



Figure 5 Acute irritant contact dermatitis of the hands caused by a polyurethane (PU) foam cleanser. In case of occupational skin damage, incorrect usage of potentially toxic products frequently leads to unintended exposure. Patients often reveal this fact only in the context of a formal doctor-patient discussion or once toxic skin damage has become obvious.



Figure 6 The patient was referred to our department for treatment of suspected hemorrhagic herpes zoster of the trigeminal nerve (with involvement of the nasociliary nerve). The correct diagnosis: local (zosteriform) skin reaction due to topically applied imiquimod for the treatment of actinic keratoses. The inflammation may serve as an indicator for therapeutic efficacy.



Figure 7 Mechanically induced psoriatic lesion (Koeber phenomenon, note: no reaction underneath the ring), initially misdiagnosed as allergic contact dermatitis to gold. At the time of diagnosis, the patient showed no other psoriatic lesions.



Figure 8 Initially diagnosed as irritant contact dermatitis, this is another example of Koeber phenomenon due to mechanical manipulation in a patient with known psoriasis.

such an example. Here, both allergic (Figure 7) as well as irritant (Figure 8) contact dermatitis may be mimicked, because an isolated or individual psoriasis lesion may frequently simultaneously exhibit erythema, desquamation, and sometimes even pustules, typical morphological features of eczema as well. Epidemiologically less significant but no less burdensome, other disorders with a completely different



Figure 9 A patient with Norwegian scabies (crusted scabies). The intensely pruritic skin lesions were misdiagnosed as chronic irritant hand dermatitis (chain smoker, immunosuppressed). In addition, there were crusted lesions on the extensor surfaces of the elbows and knees as well as the ankles. Highly contagious due to massive infestation with mites.

Epidemiologically less significant disorders may also very well mimic contact dermatitis. Examples include Norwegian scabies, dermatitis artefacta, or mechanically triggered Hailey-Hailey disease.

pathogenesis (but with eczema-like lesions at superficially plausible contact sites) may also very well mimic contact dermatitis. Examples include Norwegian scabies (with predominant involvement of the hands) (Figure 9), dermatitis artefacta in the context of delusions of parasitosis (Figure 10), or mechanically triggered Hailey-Hailey disease (Figure 11).

In recent years, there has been an increase in cutaneous adverse drug reactions that, depending on their severity, may mimic contact dermatitis. Drugs most frequently involved include the new oncologic agents that target specific molecular structures, thus hampering cell growth/differentiation or exerting immunological effects (targeted therapeutic agents).

Kinase inhibitors in particular (epidermal growth factor receptor inhibitors [EGFRI] such as cetuximab, erlotinib, gefitinib, lapatinib; multikinase inhibitors [MKI] such as sorafenib, sunitinib, pazopanib; BRAF inhibitors [BRAFI] such as vemurafenib; MEK inhibitors such as selumetinib) may mimic contact dermatitis. Mechanisms include the induction of generalized or local seborrhea with subsequent dry eczematization as well as the triggering of partially eczematous hand-foot syndrome (palmoplantar erythrodysesthesia), which may exclusively occur on palms and soles [22–24]. Figures 12–14 depict eczematous adverse drug reactions to mono- and multikinase inhibitors frequently encountered and easily confused with contact dermatitis. In this context, we would like to refer the reader to targeted therapies beyond the field of oncology, especially new agents for the treatment of hepatitis C. For example, the serine protease inhibitor telaprevir is associated with a substantial number of cutaneous adverse reactions. Although the majority of these reactions are relatively harmless, differential diagnostic considerations may lead to confusion with eczematous disorders (Figure 15).



Figure 10 Retired female farmer with chronic palmar dermatitis, unsuccessfully treated with topical corticosteroids by her family physician. The patient herself suggested “small animals” as cause. Typical clinical presentation of dermatitis artefacta, with skin lesions brought on by continuous self-inflicted manipulation. Patients typically present skin samples and “insects/parasites” (various plastic bags). Psychiatric therapy is indicated.



Figure 11 The patient was referred with a suspected intolerance reaction to adhesive tape (stoma pouch). Correct diagnosis: Hailey-Hailey disease, induced by friction.

However, even tried and tested therapeutic principles and drugs such as calcium channel antagonists or antiepileptic agents (Figure 16), which have been around for decades, have been increasingly associated with eczematous drug reactions in recent years. At least in elderly individuals, such reactions have become epidemiologically relevant [25, 26].



Figure 12 Pronounced palmar dryness and desquamation in a patient on erlotinib (kinase inhibitor). The clinical picture may be misinterpreted as irritant contact dermatitis. The manufacturer has issued brochures to inform patients as well as oncologists about possible preventative measures.



Figure 13 Suspected contact allergic reaction to rubber material (oxygen cannula). Apart from eczema near the nostrils, there is also eczematous blepharitis. Typical adverse reaction to EGFR inhibitors (here: erlotinib for the treatment of nonsmall cell lung cancer).

In this context, a special characteristic of the chemotherapeutic antimetabolite capecitabine should be pointed out (Figure 17). On the hands, the adverse reaction – in the form of inflammation and subsequent desquamation – may



Figure 14 Left side (a): contact dermatitis to toothpaste; hardly distinguishable from lip-licking dermatitis in atopic individuals. Right side (b): eczematous cheilitis caused by the kinase inhibitor lapatinib.

Simulators of contact dermatitis mimic the morphological appearance “eczema” and the pathomechanism “contact”. Triggers include pharmaceutical agents in particular.

reach such an extent that the palm lines and (fine) finger lines are lost. As a consequence, traveling to countries that require fingerprints prior to entry (such as the US) may no longer be possible. Given that leaving these countries may also be impossible (or difficult), relevant travel warnings have been issued [27, 28].



Figure 15 The patient was referred with suspected allergic contact dermatitis caused by her bra. The reaction was eventually attributed to the patient's triple drug regimen for hepatitis C (pegylated interferon, ribavirin, telaprevir). Such reactions are seen in up to 33 % of patients on PEG-interferon and ribavirin, yet markedly more frequently in combination with the serine protease inhibitor telaprevir.



Figure 16 Clinical findings of a hyperkeratotic and dyshidrotic eczematous reaction, in this case representing an adverse drug reaction to the antiepileptic agent carbamazepine.

Diagnosis, treatment, and conclusion

As frequently pointed out in the literature (and also often seen in clinical practice), essential differential diagnoses should only be considered after exclusion of relevant



Figure 17 Severe palmar desquamation mimicking irritant contact dermatitis. This was a case of severe hand-foot syndrome induced by the chemotherapeutic agent capecitabine, resulting in the loss of fingerprints.

If the patient already has an allergy ID card or requires a new one based on renewed testing, entering all positive allergic test reactions does not exempt the physician from questioning the respective relevance with regard to the present disease.

sensitizations. In no way, however, does this mean that a sensitization potentially found in a random screening patch test may automatically be determined to be the definite cause. If the patient already has an allergy ID card or requires a new one based on renewed testing, entering all positive allergic test reactions does not exempt the physician from questioning the respective relevance with regard to the present disease, which is also clearly stated in the literature. There always has to be a critical review of the relevance with respect to the disease at hand – even and especially in case of pronounced patch test reactions [29]. This requires an overall assessment of the clinical picture over the course of the disease, a review of the actual allergen exposure of the affected region, as well as an assessment of the therapeutic response. Only then can a sensitization found by patch testing be evaluated for its relevance regarding the symptoms at hand.

The treatment of simulators and above-mentioned differential diagnoses obviously includes procedures established and drugs approved for these entities. Indeed, there may be a certain degree of therapeutic overlap (for example, use of topical corticosteroids in eczematous drug reactions or milder variants of psoriasis; use of retinoids in chronic hand eczema as well as in psoriasis). However, there are also significant differences (antimycotics in dermatomycoses; local and systemic antiscabies agents in Norwegian scabies; CO₂ laser in Hailey-Hailey disease).

With respect to the therapeutic options for patients with eczema such as (topical) corticosteroids, topical immunomodulators, retinoids, UV therapy, and measures to ensure allergen avoidance (job change, protective clothing), we refer the reader to the relevant literature [1, 29]

Note: Allergic contact dermatitis (for example, due to ointment bases, cosmetics, or similar substances) frequently develops on top of atopic dermatitis. While there is indeed short-term relief following allergen avoidance and topical corticosteroid therapy (most forms of eczema and inflammatory eczematous



Figure 18 Interpreted as extensive, blistering, irritant contact dermatitis, the actual diagnosis was pompholyx (dyshidrosis) in a patient with atopy, complicated by severe stress and nicotine abuse.

Given that most inflammatory eczematous disease entities – at least temporarily – respond to immunosuppressive agents such as corticosteroids in particular, such agents cannot be considered useful differential therapeutic tools that allow for a (post hoc) diagnosis based on prior therapeutic success.

dermatoses respond well to external corticosteroids), such short-term therapeutic success will likely not substantially change the need for long-term care, given the chronically recurrent nature of atopic dermatitis (Figure 18). Given that most inflammatory eczematous disease entities – at least temporarily – respond to immunosuppressive agents such as corticosteroids in particular, such agents cannot be considered useful differential therapeutic tools that allow for a (post hoc) diagnosis based on prior therapeutic success.

Even though the various noncontact-related eczema entities (such as atopic dermatitis, seborrheic dermatitis, stasis dermatitis, and others) likely constitute the epidemiologically most important differential diagnoses for contact dermatitis, the other medical conditions and simulators mentioned herein are by no means to be neglected in differential diagnostic considerations. Only on the basis of a correct diagnosis can adequate therapy and promising prophylaxis be initiated. Such actions ultimately provide the patient with relief of and freedom from symptoms.

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Fragen zur Zertifizierung durch die DDA

1. Wie hoch wird die Prävalenz für Ekzemerkrankungen in Deutschland angegeben?

- a) unter 1 %
- b) 1–5 %
- c) 15–20 %
- d) über 80 %
- e) 100 %

2. Welche Aussage zum Ekzem beziehungsweise Kontaktekzem trifft zu?

- a) Die Diagnose und Definition ist primär morphologisch.
- b) Die Diagnose kann nur histopathologisch gestellt werden.
- c) Die Diagnose basiert auf erhöhten IgE-Werten.
- d) Die Diagnose ist eine Ausschlussdiagnose.
- e) Die Diagnose basiert auf immunhistochemischen Befunden.

3. Welche Effloreszenz ist **nicht** typisch für ein Kontaktekzem?

- a) Papel
- b) Pustel
- c) Comedo
- d) Vesikel
- e) Kruste

4. Welche der folgenden Diagnosen wird als Differenzialdiagnose zu einem Kontaktekzem angesehen?

- a) malignes Melanom
- b) Morbus Bowen
- c) Ecthyma
- d) Cornu cutaneum
- e) Mastozytose

5. Ein Patient hat seit wenigen Tagen eine Kontaktekzem-verdächtige Läsion am Handrücken. Welche der folgenden Diagnosen ist damit differenzialdiagnostisch unwahrscheinlich?

- a) Neurodermitis
- b) Psoriasis vulgaris
- c) Epidermomykose

- d) Lichen ruber verrucosus
- e) Polymorphe Lichtdermatose

6. Welche der folgenden Erscheinungsbilder gehört **nicht** zu den nicht-ekzematösen Manifestationen kontaktallergischer Reaktionen?

- a) Erythema-multiforme-artige Reaktion
- b) pigmentierte Purpura
- c) Lichen-planus-artige Reaktion
- d) granulomatöse Reaktion
- e) Pathergie-Phänomen

7. Welche Aussage zum Epikutantest trifft zu?

- a) Ein positives Resultat muss als relevant angesehen werden.
- b) Ein positives Resultat ist nur relevant, wenn eine berufsspezifische Serie getestet wurde.
- c) Ein negatives Ergebnis schließt ein allergisches Kontaktekzem sicher aus.
- d) Ein Epikutantest ist bei der Abklärung eines Kontaktekzems nicht indiziert.
- e) Ein positives Resultat muss auf seine Relevanz geprüft werden.

8. Welche Medikamente können typischerweise Arzneireaktionen verursachen, die wie Kontaktekzeme erscheinen können?

- a) Reninantagonisten
- b) Gyrasehemmer
- c) Kinasehemmer
- d) Volumenersatzmittel
- e) Impfstoffe

9. Welches Medikament kann so starke ekzematöse Lokalreaktionen auslösen, dass möglicherweise die Hand- und Fingerlinien verschwinden?

- a) Cefazolin
- b) Ceftriaxon
- c) Cetuximab

- d) Capecitabin
- e) Colecalciferol

10. Welche Aussage ist richtig?

- a) Kontaktekzeme können aerogen ausgelöst werden.
- b) Kontaktekzeme entstehen auf vorgeschädigter Haut.
- c) Kontaktekzeme sind üblicherweise in trockenen Hautregionen zu finden.
- d) Kontaktekzeme können nicht an Haarfollikel-reichen Hautstellen auftreten.
- e) Kontaktekzeme sind nicht mit Juckreiz assoziiert.

Liebe Leserinnen und Leser,
der Einsendeschluss an die DDA für diese Ausgabe ist der 21. Dezember 2015. Die richtige Lösung zum Thema „Allergologie – Aktuelles zur Prävention und Diagnostik von Soforttyp-Allergien und zur Therapie der allergischen Rhinokonjunktivitis“ in Heft 8 (August 2015) ist: 1c, 2a, 3d, 4d, 5b, 6d, 7b, 8b, 9d, 10b

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