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REVIEW ARTICLE

Differential diagnosis of contact dermatitis: A practical-approach review by the EADV Task Force on contact dermatitis

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Abstract

The diagnosis of eczema ('dermatitis') is mostly clinical and depends on the clinical history and exploratory objective findings (primary lesions, patterns). Contact dermatitis remains as an important condition in the group of eczematous disorders, with important socioeconomic and occupational relevance. Although irritant and allergic contact dermatitis have a different pathogenesis, both are characterized by a rather typical morphology, are triggered by external factors and tend to occur primarily in the area of contact with the exogenous agent. In addition, allergic and irritant dermatitis may also co-exist. The importance of diagnosing contact dermatitis, especially when allergic in nature, is both due to the possibility of avoiding the trigger, and due to its role in aggravating other skin conditions. Nevertheless, the heterogeneity of clinical presentations in daily practice may pose an important challenge for the suspicion and correct diagnosis of contact dermatitis. Furthermore, other conditions, with different pathogenesis and treatment, may clinically simulate contact dermatitis. The Task Force aims to conduct a review of the unifying clinical features of contact dermatitis and characterize its main clinical phenotypes, and its simulators, in order to contribute to an early suspicion or recognition of contact dermatitis and enable a correct differential diagnosis.

BACKGROUND

Contact dermatitis (CD) is a type of skin inflammatory reaction that occurs secondary to contact with a specific substance. CD is mostly eczematous in nature. The definition of eczema relies on morphologic traits.[1] 'Eczema' may also be referred to as 'eczematous dermatitis' – or simply 'dermatitis', with equivalent denotation in clinical practice.[2] Eczematous dermatitis presents with a remarkable diversity of primary lesions, such as erythema, macules, papules, vesicles and bullae, as well as secondary lesions like scaling, erosions, crusts, lichenification, hyperkeratosis and hypo- and/or hyperpigmentation. Erythema, vesicles, oedema and exudative changes are more typical in acute phases, whereas more papular, fissured, scaly and even hyperkeratotic states are seen in subacute and chronic phases (Figure 1a).[3] Furthermore,

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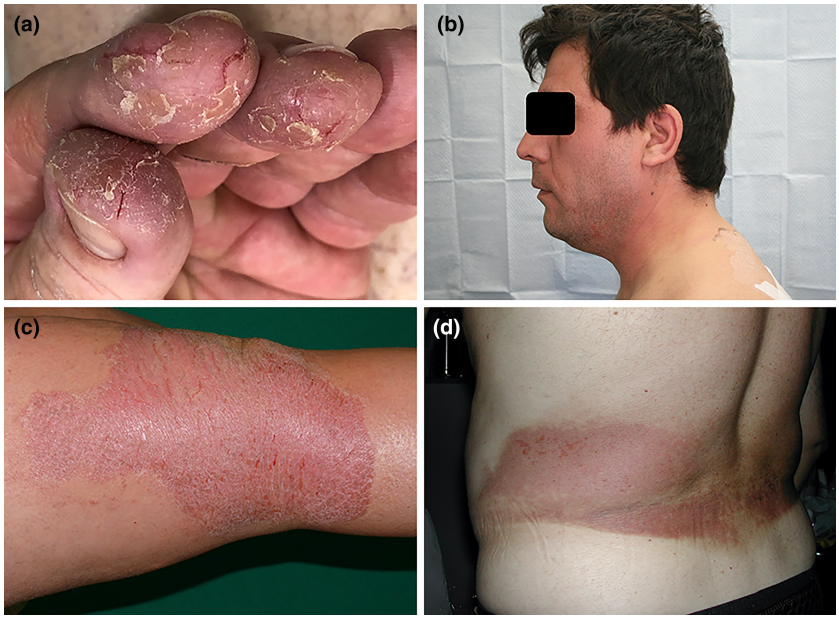

FIGURE 1 The multi-faceted presentations of contact dermatitis. (a) Pulpitis due to allergic contact dermatitis from diallyl disulfide present in garlic. (b) Airborne contact dermatitis due to potassium dichromate in a welder with retroauricular, submental, nasolabial and mild frontal involvement. (c) Photoallergic contact dermatitis from ketoprofen presenting as sharply demarcated eczematous lesions on the leg. (d) Violaceous erythema and areas of hyperpigmentation after healing along with areas of vesicles and erosions on the back of a patient as a manifestation of phototoxic contact dermatitis induced by common rue (*Ruta graveolens*).

overlap between acute and chronic lesions may also occur, for example during flares in chronic dermatitis.[3] It is essential to highlight that history taking and physical examination, supplemented with additional diagnostic tests, and, if applicable, along with studying the effect of avoidance and re-exposition to the suspected culprit(s), are essential for establishing an accurate diagnosis of contact dermatitis (CD) and providing optimal care for the patient.[4]

The aim of this practical review is to provide an up-to-date guidance for dermatologists on how to adequately suspect and diagnose CD.

Classification of eczematous disorders

Eczematous disorders include CD and photo-induced CD, atopic dermatitis, dyshidrotic eczema, asteatotic eczema, seborrheic dermatitis, nummular eczema, stasis dermatitis, eczematous cutaneous adverse drug reactions and protein contact dermatitis (PCD).[2] Identifying the precise disorder(s) may be challenging because of overlapping clinical patterns and the difficulty in histopathological differentiation of eczematous conditions. Thus, diagnosis is mostly based on clinical findings and, if possible, complementary tests.

Clinical forms of contact dermatitis

Contact dermatitis may present with many different and subtle manifestations.[5] Irritant contact dermatitis (ICD), allergic contact dermatitis (ACD), photo-induced CD (including photoallergic CD and phototoxic CD) and PCD emerge as different categories within the spectrum of CD. The differentiation of these categories is facilitated by the presence of differences in terms of triggers and pathogenic mechanisms. Clinical manifestations of CD may vary by factors such as the triggering culprit, route and duration of exposure, skin type and duration of inflammation.[3] Clinical examination *per se* is generally regarded as having limited reliability for the differentiation of these CD subtypes, although some clinical clues may occasionally provide guidance (see below).[3]

Irritant contact dermatitis is believed to be the most common subgroup of CD, accounting for even 80% of all cases.[6] However, taking into account the occurrence of potential pitfalls in the diagnosis of ACD, this percentage could include misdiagnoses of other subgroups of CD. It may be occupational, especially related to wet-work conditions, and several factors may contribute to the susceptibility of ICD (age, sex, atopy, body region). Its clinical features are very polymorphous.[7] The heterogeneity of ICD is related to the complex genetic and environmental mechanisms, but it is always characterized by the direct or indirect skin injury induced by external stimuli, activating the innate immune system.[6] From a clinical perspective, lesions are considered to be well-demarcated and confined to the area of contact with the irritant, even if in selected cases some spreading may occur (e.g., from the hands to the forearms).[8] In ICD of the hands, the interdigital spaces and dorsal side of the hands and fingers are often involved, yet this is not a specific sign.[3] Lesions of ICD heal rather quickly if the triggering agent(s) are removed ('*decrescendo*' phenomenon).[9,10] Chronic ICD may present as dry, erythematous, scaly lesions

with lichenification,[9] a form often entailing a poor prognosis.[11] The predominating subjective symptom of ICD is not pruritus, but burning sensations, pain and skin soreness in acute ICD,[11] while pruritus and eczema may also be present in more chronic ICD.

Allergic contact dermatitis is also very common in daily practice, and an increase in its prevalence has been evidenced in the last years.[12] Skin involvement in ACD is the result of a specific, immune-mediated delayed reaction against haptens that come in contact with the skin. In its acute form, it has a tendency to develop erythema, papules, vesicles and, occasionally, bullae, whereas more evolved forms usually present with the features of chronic dermatitis.[9] Pruritus is the cardinal symptom of ACD. The most frequently involved body areas are the face, hands and forearms, with legs and trunk often being other frequent sites of involvement.[2] ACD may typically present with extension to areas outside of the area that is in direct contact with the allergen ('spreading reaction; id-reaction'),[3] possibly due to allergen persistence, extensive exposure to the allergen, areas with resident-memory T cells that had previous contact with the allergen, or even due to haematogenous spread. In contrast with ICD, a '*crescendo*' phenomenon may occur in ACD, even after allergen removal.[9] However, these diagnostic clues do not fully reflect the clinical diversity and complexity of ACD. In fact, several publications have already previously attempted to elucidate the clinical patterns/forms of presentation of ACD.[1,5,12]

One consensus classification of ACD includes 13 main categories[5]: direct exposure ACD, exacerbating pre-existing ACD, multifactorial dermatitis including ACD, ACD by proxy, ACD mimicking angioedema, airborne ACD, photo-induced ACD, systemic ACD, PCD, allergic contact stomatitis, erythroderma/exfoliative dermatitis, non-eczematous ACD (e.g., lymphomatoid ACD) and ACD with additional respiratory/mucosal symptoms. Additional minor forms of ACD presentation include, for example, hair loss, increased hair growth, tattoo reactions, granulomatous reactions, among many others. The aim of this article is not to revisit each of these categories, but to outline the clinical features that may be of utmost importance for clinicians.

ACD by proxy or connubial/consort dermatitis is a form of ACD with a particular mechanism of allergen exposure. In this case, the allergen source is another individual and the exposure is by close contact.[13] An illustrative example would be cheek ACD after transfer of a product from the partner's lips. This ectopic mechanism should be considered when no clear rationale for personal contact with a demonstrated allergen is found. The causative agents are commonly topical medications.[13]

Airborne ACD is a diagnostic challenge for many dermatologists. The fact that allergens contact with the skin is under the form of small, air-distributed particles, vapours and gases, may easily lead to confusion with photo-induced CD. Airborne ACD typically involves the face, neck and/or the neckline without any spared areas (Figure 1b), in contrast to phototoxic and/or photoallergic CD.[9] Clinical clues to look for are involvement behind the ears, under the chin, under the nose and on the upper eyelids, which are the so-called 'shadow zones', typically unaffected in photo-induced CD.[9] Nevertheless, both airborne and photo-induced CD may co-exist, as has been reported for photo-aggravated, airborne ACD from isothiazolinone-containing paints and detergents.[14] Moreover, as the eczematous lesions in airborne ACD are often quite ill-defined, the diagnosis may be challenging.[9]

Photo-induced CD includes two separate conditions. **Photoallergic CD** results from skin inflammation caused by a photosensitive allergen combined with sunlight or artificial light.[15] It is characterized by relatively ill-defined, eczematous lesions in light-exposed areas (Figure 1c), such as face, ears, V-area of the neckline and below the cuffs of the two upper limbs, whereas 'shadow zones' (see above) remain spared.[9] **Phototoxic reactions** involve rather non-specific immune mechanisms and present mostly as an exaggerated sharp-delimitated sunburn that tends to precede subsequent hyperpigmentation (Figure 1d). Overlap features of both conditions may present in a patient.[16]

Systemic CD includes a spectrum of clinical presentations that occur after systemic exposure to a contact allergen. Several different allergens may lead to such pictures of ACD, such as drugs, plant products and metals.[17] Clinical pictures are characterized by widespread/generalized eczema, often involving body folds, or by acute vesicular palmoplantar dermatitis (resembling dyshidrotic eczema or 'pompholyx'). Systemic ACD may have some additional, relatively specific skin manifestations, such as the recurrence of dermatitis at the site of previous contact after systemic exposure to the allergen, also observed in experimental oral challenge trials.[18] Systemic ACD to drugs is a type of delayed IVa (T-helper 1 inflammation) or IVc (cytotoxic T lymphocytes) hypersensitivity reaction, caused by topical or systemic drugs.[17,19] It presents with widespread eczematous eruptions, worsening/reactivation of eczema, with symmetrical drug-related intertriginous and flexural exanthema (SDRIFE), or with erythroderma. SDRIFE is characterized by a sharply defined symmetrical erythema (red to purple-red) of the buttocks and inner thighs, in the latter with V-shaped distribution, with involvement of at least one other flexural location and without systemic symptoms and signs (Figure 2a,b).[20,21]

Protein contact dermatitis belongs to the group of immediate hypersensitivity skin reactions and follows repeated episodes of contact urticaria, although the latter is not always evident for the patient.[22] Pruritus, erythema, wheals or angioedema occurring in a matter of minutes after skin contact with a culprit agent are characteristic in the acute phase, while the subacute phase is followed by vesicles and other eczematous features.[23] After the acute phase, chronic hand dermatitis (erythema, lichenification, fissures and sometimes residual scales) may be seen.[23] PCD shows a predilection for the hands (especially the fingertips), sometimes extends to the wrists and arms and is typically, yet not exclusively, occupational in origin. Rarely, the face and other locations may be involved.[24]

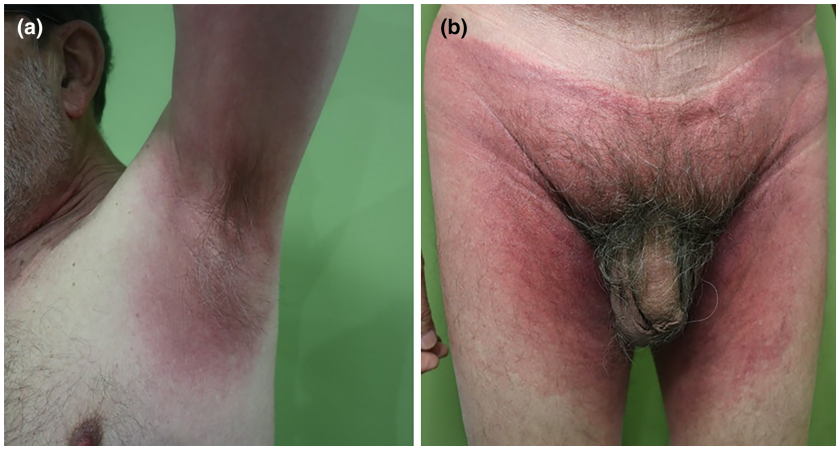

FIGURE 2 Symmetrical Drug-related Intertriginous and Flexural Erythema (SDRIFE) secondary to azithromycin. (a) Symmetrical erythema of the axilla, here only showing the left axilla. (b) Symmetrical V-shaped erythema in the inguinal area.

Non-eczematous and other minor clinical forms of presentation of ACD are addressed in the section of 'Uncommon clinical findings of CD'. Even though these different clinical forms of CD share many common findings, their varied clinical presentations often lead to diagnostic delay and/or unnecessary complementary tests.

DIFFERENTIAL DIAGNOSES OF CONTACT DERMATITIS

To correctly diagnose CD, it is essential to understand its clinical presentations and to ensure an adequate diagnostic approach, which includes the differential diagnosis of a wide range of conditions. In accordance to guidelines, this process requires a comprehensive assessment of past medical history, questioning on the rash development, occupational and private exposures and, if applicable, the evaluation of the interrelationship.[25] Further anamnesis may be required depending on the clinical suspicion. This section presents the main differential diagnoses of CD and highlights clinical features to take into account for a complete anamnesis and exploration during the differential diagnosis process for eczematous and non-eczematous disorders.

Inflammatory conditions

Several entities may resemble the clinical presentation of CD due to the eczematous lesions and/or the pattern(s) of distribution. In addition, clinical situations such as the lack of response to conventional therapies or unexplained flares could raise the possibility of additional (superimposed) CD.

Other eczematous disorders

The diagnosis of **atopic dermatitis** is based on a series of clinical criteria.[26] The diagnosis is made in the setting of eczematous, relapsing lesions with a typical morphology that correspond to the atopic dermatitis age-specific pattern (cheeks, forehead and extensor involvement in infantile forms, and later flexural, head and neck and hand lesions in adolescents and adults), together with the hallmark symptom of pruritus. Other important features are an early age of onset and personal/family history of atopy. In addition, increased serum IgE, the presence of minor atopy-associated cutaneous features (e.g., keratosis pilaris, hyperlinear palms and generalized xerosis) and susceptibility to cutaneous infections (e.g., Staphylococcus aureus and Herpes simplex) may also guide our clinical suspicion. With respect to atopic dermatitis, ACD should be considered as either an alternative diagnosis and/or an exacerbator.[26] ACD in patients with atopic dermatitis should be taken into consideration in cases when history and/or physical examination are suggestive of ACD, such as flares, or an inappropriate response to topical treatment (e.g., topical corticosteroids) or emollients, or when specific patterns are observed that reflect exposure to a causative agent. In this regard, ACD could be suggested by marked facial or eyelid involvement, increased severity in neck folds and vesicles on dorsal hands and fingertips.[27] Moreover, systemic ACD may occasionally result in a phenotype mimicking atopic dermatitis.[28] Patch testing is recommended in these situations, or in the setting of known atopic dermatitis in the absence of improvement to standard treatments.[27]

Dyshidrotic eczema is defined as chronic, intermittent, highly pruritic episodes of vesicles with faint erythema that involve hands (palms, lateral aspects of fingers) and feet (soles). Less frequently, bullae may be seen.[29] Scaling occurs when vesicles dry, but an erythematous background of lesions occurs less frequently. It usually runs a seasonal course and is occasionally related to different triggers such as hyperhidrosis, contact irritants and even metal hypersensitivity.[30] The possible relationship with metal contact allergy and the common clinical patterns of dyshidrosis and acute ACD may hamper the correct diagnosis.[30] Moreover, acute palmoplantar vesicular eruptions may also be a clinical presentation

of systemic ACD that could mimic dyshidrotic eczema (see above).

Asteatotic eczema, or **craquelé dermatitis**, is the result of extremely dry skin after excessive epidermal water loss. Dry, cracked skin that can evolve into fissures and erythematous patches with the typical, almost diagnostic, polygonal or curvilineal pattern in fissured skin is seen.[31]

The diagnosis of **seborrheic dermatitis** remains a clinical one, presenting with the characteristic symmetrical erythema and yellowish-to-white 'greasy' scaling that may occur in nasolabial and retroauricular folds, eyebrows, scalp, and in interscapular and pre-sternal areas. Concomitance of ACD and seborrheic dermatitis may occur.[32] Seborrheic dermatitis refractory to therapy, or worsening upon topical products, should be regarded as clues for secondary ACD. ACD may also mimic seborrheic dermatitis (e.g., from cosmetics and nail lacquers).[33]

Nummular eczema is defined by the sharply defined, coin-shaped, eczematous plaques, usually symmetrically distributed, with an anatomical preference for the lower and upper limbs.[34] Contact allergy may be associated with nummular eczema in up to 25% of patients, particularly from nickel and preservatives.[35,36] ACD with a nummular pattern has also been described in relation to persulfates, and contact allergens present in antiseptics.[37,38]

Stasis dermatitis is the consequence of chronic venous insufficiency. Erythematous and eczematous patches and plaques restricted to the lower legs, without clear-cut borders, and classically involving the medial malleoli, are the most common findings.[39] Pruritus, scaling and lichenification may occur. Brown hyperpigmentation is a consequence of the dermal deposition of hemosiderin. Patients with stasis dermatitis, especially those with leg ulcers, may readily develop ACD to the personal care products, topical medications and wound dressings.[40,41]

Delayed-type hypersensitivity drug reactions include a maculopapular eruptions, fixed drug eruption, acute generalized (localized) exanthematous pustulosis (AGEP/ALEP), drug reaction with eosinophilia and systemic symptoms (DRESS), systemic CD, erythema multiforme and Stevens–Johnson/toxic epidermis necrolysis (SJS/TEN) syndrome.[42,43] It is important, when evaluating potential drug eruptions, to take into account that CD may occasionally simulate such drug eruptions (e.g., erythema multiforme-like CD[44–47] or ACD mimicking AGEP[48,49]), and that, occasionally, these drug reactions may also mimic CD.[50,51] Notably, eczematous cutaneous adverse drug reactions, or eczematous drug eruptions, are general terms used to define rashes with eczematous features that occur following the administration of systemic medications.[52] These reactions may develop in the context of systemic CD (see above).[52] The diagnosis may be difficult as the exact clinical presentation may vary for each drug, but most develop within weeks to months after the onset of the treatment.[52]

Table 1 outlines the main eczematous disorders included in the differential diagnosis of CD with anatomical and clinical clues.

Non-eczematous disorders

The differentiation of **psoriasis** (and its variants) from CD may pose several challenges in clinical practice, with previous literature citing the intricated clinical and pathological differences and similarities.[53,54] It is important to stress the difficulty in distinguishing chronic plantar/palmar dermatitis from plantar/ palmar psoriasis. Furthermore, the term 'eczema in psoriatico' is frequently used to define the co-occurrence of psoriasis and eczematous disorders, such as CD.[55] Although it has been suggested that patients with psoriasis could be less prone to ACD due to differences in the inflammatory milieu,[56] clinicians need to be aware that ACD can certainly complicate psoriasis, with ACD sometimes presenting psoriatic features, namely through the Köebner phenomenon.[57]

Lichen planus and its variants share common clinical and sometimes histopathological lichenoid features. Dermoscopy, with the rather typical Wickham striae, may contribute to the distinction.[58,59] However, CD may also mimic lichen planus ('lichenoid CD').[60] In regard to cutaneous lesions, the eruption initially involves contact sites, followed by a characteristic spreading reaction, yet with less characteristic polygonal lilac papules, and often without mucosal lesions.[61] Moreover, the onset of lichenoid CD is rather acute with sometimes frank eczematous lesions being noticeable in the contact site.[61] Nevertheless, the clinical, histopathologic and dermatoscopic resemblance may be high, and distinction can remain difficult.[62] In addition, lichenoid contact allergy has been particularly described in the oral mucosa, mostly due to dental amalgam.[63] Interestingly, positive patch test reactions may initially be eczematous and later on become lichenoid.[61]

Lichen simplex chronicus (LSC) is a form of localized itch with secondary thick lichenified plaques in the areas of scratching.[64] It is symmetrically found in easily accessible body regions, such as the ankles, shins, dorsal hands, upper back, neck and anogenital regions. Occasionally, it has been suggested that ACD from hair dye may precede in some cases the development of LSC, and it can be anticipated that long-lasting ACD may, in some cases, easily transform into LSC.[65]

Regarding **acne vulgaris**, **perioral dermatitis** and **rosacea-like dermatitis**, one should remember that almost all the subgroups of CD may have perioral and face involvement, and that some cases may effectively mimic flares of the aforementioned conditions.[66,67] Recalcitrant rosacea, including its oedematous complication, referred to as 'Morbihan disease', or perioral dermatitis should raise the possibility of superimposed CD and should lead to the evaluation of face cosmetic and topical products. If cheilitis and/or perioral PCD are present, the use of musical instruments, toothpastes and even food allergens should also be evaluated. Moreover, some reports have highlighted that ACD from fragrances and formaldehyde releasers may resemble or aggravate rosacea, whereas others have stressed that acne vulgaris/rosacea with superimposed ACD can mainly present as a flare-up of the original dermatosis.[68]

TABLE 1 Diagnostic clues of eczematous conditions included in the differential diagnosis of contact dermatitis.

Condition	Predilection sites	Clinical (anamnesis and exploratory) clues
Allergic CD	It may affect any area (face and neck, scalp, hands/feet, trunk, arms/legs, nails, body folds, genital and/or be generalized)	Pruritic dermatitis, with partial or no response to corticosteroids, with recurrent flares. Ill-defined lesions, except for the volar palmar and wrists borders,[25] with spread beyond areas of direct exposure.[9] '*In crescendo*' phenomenon[9]
Irritant CD	Mostly hands, but occasionally in arms, folds, genitals	Well-demarcated, rash in the area of contact with irritants.[6] Worsening in cold season[6] and '*decrescendo*' phenomenon.[9] Burning or stinging sensations may be present
Photo-induced CD	Photo-exposed areas, mostly face and neck, scalp or generalized in exposed trunk and extremities	Identification of light as a trigger and evaluation of the use of topical or systemic drugs with phototoxic and/or photosensitizing properties. Photoallergic CD is ill-defined in contrast to phototoxic CD and may spread to covered body parts[25]
Systemic CD	Folds, hands/feet, generalized	Investigation of the use of topical and/or systemic drugs as well as contact with other products such as metals, plants and drugs.[17] When affecting the palmoplantar regions, a vesicular presentation may predominate the clinical picture
Protein CD	Mostly in hands	Atopic skin diathesis. Local immediate symptoms upon contact (mostly itch and erythema or contact urticaria) in initial stages and vesicles later[23]
Atopic dermatitis	Face and neck and/or hands/feet and/or arms/legs (flexures) and/or be generalized	Personal and familial history on atopy with early age of onset (though it can develop at any age). Specific age distribution patterns associating dry skin and intense itch
Dyshidrotic eczema	Hands and feet	Existence of hyperhidrosis, seasonal flares or contact with irritants
Asteatotic eczema	Arms and legs or generalized	Investigation of deficient use of moisturizers. Polygonal or curvilineal pattern in fissured skin
Seborrheic dermatitis	Face and neck, scalp, genital or be generalized (trunk)	Investigation of triggers (e.g., stress, alcohol and neurological diseases). Seborrheic areas with less pruritus. Stinging/burning may be associated
Nummular eczema	Arms and legs or be generalized	Coin-shaped, eczematous, symmetrical plaques
Stasis dermatitis	Legs	Well-demarcated macular patches and plaques in a background of chronic venous insufficiency of the legs and local symptoms of venous insufficiency
Delayed-type hypersensitivity drug eruption	Face and neck (DRESS, AGEP, SJS/TEN, FDE), hands and feet (erythema multiforme), genital (FDE), generalized (most types, including maculopapular eruptions)	Detailed history and timing of systemic medication Clinically very heterogeneous

Abbreviations: AGEP, acute generalized exanthematous pustulosis; CD, contact dermatitis; DRESS, drug reaction with eosinophilia and systemic symptoms; FDE, fixed drug eruption; SJS/TEN, Stevens–Johnson syndrome/Toxic epidermal necrolysis.

Polymorphic light eruption is characterized by a days' duration rash of usually smooth-topped erythematous papules that tend to form plaques, even if the rash may be heterogeneous.[69] The diagnosis is clinical, and many clues differentiate it from CD, such as the sun-related trigger, and especially the shorter duration and typically relapsing nature of the rash. Nevertheless, photo-induced CD—alone or superimposed (e.g., from sunscreens), remains an important consideration, especially when the eruption is acute, without any previous history, and/or when relapses do not have the same characteristic morphology.

Within the spectrum of auto-immune diseases, **lupus erythematosus** and **dermatomyositis** have to be considered in the differential diagnosis of CD. In regard to lupus, acute malar rash and some forms of subacute and discoid lupus erythematosus are part of the differential diagnosis of CD. CD has very occasionally complicated cutaneous lupus erythematosus.[70] Moreover, as in psoriasis and rosacea, a secondary ACD may also trigger a relapse of an underlying cutaneous lupus erythematosus, and, similar to lichen planus, patch tests may then also transform from an eczematous patch into a lesion with histopathological

characteristics of lupus ('contact lupus').[71] Lymphomatoid ACD may also completely mimic cutaneous lupus, or related disorders (e.g., Jessner lymphocytic infiltration of the skin/Jessner-Kanof's disease, which, similar to CD, may histopathologically show the presence of eosinophils). Since cutaneous lesions of dermatomyositis affect the scalp, face, eyelids and hands (mechanic's hands), and associate pruritus, the differential diagnosis between CD and dermatomyositis based on clinical signs and symptoms is often challenging. Furthermore, ACD can also be superimposed and even delay the diagnosis of this particular auto-immune disease.[72] In addition, both lupus erythematosus and dermatomyositis, when cutaneous lesions are photo-aggravated, need to be considered as differential diagnoses and even mimickers of photo-induced CD.[73]

Infectious diseases

Skin infections may also resemble CD. However, an accurate examination and complementary tests will normally lead to the elucidation of the diagnosis. In contrast to inflammatory conditions, overlap or association with CD is uncommon.

Tinea infections, (Figure 3a,b) including **Tinea incognito**, have been described as mimickers of ACD, particularly in children.[74] To rule out the possibility of tinea, KOH test and/or culture of scrapings need to be performed.

Scabies can present with very unspecific signs (e.g., eczematous dermatitis, urticarial eruptions, intense pruritus and excoriations), yet may also have some specific dermatoscopic hallmarks (e.g., white lines and delta wing sign), and suggestive clinical signs (e.g., linear skin tunnels in the palmar creases and wrists and scabietic nodules in the genital and umbilical region). Importantly, superimposed CD to some topical treatments of scabies has been described.[75] Therefore, lack of improvement despite correct treatment will require to rule out not only the persistence/re-infection but also irritancy/contact allergy to the ingredients of topical treatments.

Non-bullous and bullous **impetigo** may resemble, or complicate CD,[76] causing impetiginized dermatitis. Bacterial cultures can be used for diagnostic confirmation. In addition, severe cases of ACD, with severely exudating skin lesions, may mimic impetigo.[66]

Erythrasma is a bacterial infection characterized by dark erythematous-brownish patches and fine scaling that primarily affects interdigital spaces and intertriginous areas. It may be confused with (A)CD (e.g., from fragrances and textiles). Wood's lamp examination may show a characteristic 'coral red' fluorescence.

Other entities such as **viral exanthems**, including **pityriasis rosea**, **herpetic lesions** and **candidiasis** are regularly included in the differential diagnosis of CD, especially in early stages. Cutaneous candidiasis affects mostly body folds (inguinal, inframammary, anogenital) with one characteristic finding, the presence of peripheral papules and pustules, accompanying erythema and maceration. However, differentiation from 'pustular' ACD (e.g., from fragrances) may need to be considered.[77] Herpes simplex is common in clinical practice and could be a misdiagnosis with intense, vesicular ACD, and vice versa, although the associated pain should direct the clinician to the right diagnosis.[78]

Other diseases

This section includes a myriad of conditions that may occasionally be part of the differential diagnosis of CD.

Genetic conditions, such as **Hailey–Hailey disease** (autosomal dominant due to mutations in ATP2C1 mutations) and **Darier's disease** (autosomal dominant disease due to ATP2A2 mutations), may clinically resemble CD and even co-exist.[79] **Grover's disease**, although not considered a genodermatosis, is another dermatosis that might mimic CD. All three conditions are histopathologically characterized by acantholysis, which is useful to differentiate those from spongiotic conditions. Other genetic syndromes such as **peeling skin syndrome type B, Severe dermatitis—multiple Allergies—Metabolic wasting (SAM) syndrome, Netherton syndrome** and **Omenn syndrome** present with recalcitrant atopic dermatitis-like manifestations, and with additional cutaneous and non-cutaneous features suggesting their diagnosis.[80] The underlying skin barrier alteration could predispose these patients to secondary CD, even if evidence is lacking to date.

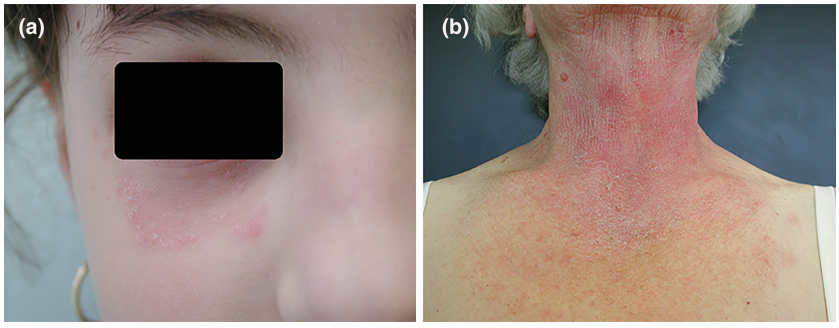

FIGURE 3 Tinea faciei in a child and Tinea incognita. (a) Erythematous annular and macular patch with a scaly, spreading border that also contains papules in a child with Tinea faciei. (b) Photo-distributed rash with eczematous appearance in a woman who had already been treated with topical corticosteroids. A detailed anamnesis and a closer look, altogether with microbiological studies, led to the diagnosis of Tinea incognito.

Premalignant and malignant disorders may also mimic CD. Importantly, any 'atypical' eczema which fails to respond to topical corticosteroids may represent a premalignant or malignant disorder, namely **cutaneous T-cell lymphoma, parapsoriasis *en plaque*, mammary and extramammary Paget's disease, and in situ squamous cell carcinoma,** including **Bowen's disease** and **erythroplasia of Queyrat**.[1] Therefore, in such cases, a skin biopsy is mandatory to establish the exact nature of the lesion.[81] Mycosis fungoides, with its multiple clinical variants, and pathogenesis linked to 'persistent allergen stimulation', has been associated with an increased prevalence of delayed hypersensitivity.[82] In addition, the clinical/histopathological differentiation between mycosis fungoides and lymphomatoid ACD may pose a significant challenge,[83] since both may be indistinguishable and lymphomatoid ACD is mainly T cell-based, and only rarely B-cell based.[84]

Other conditions that have seldom been included in the differential diagnosis of CD are **bullous pemphigoid, porphyria cutanea tarda** and psychodermatologic diseases such as **delusions** and **dermatitis artefacta**.

Table 2 outlines the main non-eczematous disorders included in the differential diagnosis of CD with anatomical and clinical clues.

Differential diagnosis for special locations (face and neck, hands and feet and intertriginous areas) with diagnostic clues

Some anatomic areas entail difficulty during the differential diagnosis process due to specific clinical features of conditions in these areas. Table 3 highlights some clinical clues to consider in clinical practice. Figures 4 and 5 show different conditions on the face and hands/feet, respectively.

UNCOMMON CLINICAL FINDINGS OF CONTACT DERMATITIS

It is difficult to summarize rather uncommon forms of CD, since our knowledge mainly stems from case reports or small case series. Uncommon clinical forms do not correspond to primary clear-cut lesions of eczema, yet a diagnosis is achieved through complementary tests (patch testing, in particular, but also others, such as skin biopsy) and correlation with exposure. A myriad of skin manifestations has been reported including: **(de)pigmented dermatitis**,[98–102] **purpuric dermatitis**,[103–105] **skin and oral lichenoid dermatitis**,[61,62] **erythema multiforme-like dermatitis**,[44–47] **lymphomatoid dermatitis**,[106–109] **granulomatous dermatitis and cheilitis**,[110–112] **pustular dermatitis**,[113–117] **neutrophilic and eosinophilic dermatitis**,[118] **bullous dermatitis**[119–121] and **sclerodermoid dermatitis**.[122] In addition, in rare cases, ACD may not present as dermatitis, but rather with nail or hair alterations (hair loss or increased hair growth).[123,124] Furthermore, delayed contact allergy may also present with extracutaneous manifestations with or without associated skin findings, as in the case of allergy to prosthetic or external materials[125] (e.g, pseudotumors[126] and implant failure[127,128]). A special clinical manifestation of CD includes 'angioedema-like' contact dermatitis, which is sometimes referred to as oedematous CD, or 'bullous' CD. Although this is often misinterpreted as classic angioedema, the observed oedema (often facial) is itchy, lasts longer than 3 days and often resolves leaving a residually scaly dermatitis or dry skin, all three being features not consistent with a classic angioedema. Different allergens have been associated with this particular clinical presentation, including paraphenylenediamine (PPD) and methylisothiazolinone.[129,130]

CONTACT DERMATITIS: THE GREAT MIMICKER AND ITS SIMULATORS

This section includes a schematic presentation of (i) CD presenting with a morphology and/or distribution suggesting another skin condition, and (ii) CD simulators, that is, conditions with CD-like features. The first situation is the result of the variable clinical manifestations of CD and stresses the diagnostic difficulty in clinical practice. In contrast, CD simulators are conditions that may exhibit eczematous features and/or present a pattern or distribution that suggests CD.[1] For further examples, see Table 4.

COMPLEMENTARY TESTS

The diagnostic approach will also entail complementary tests, which will mostly be, for CD, patch and photopatch testing and, if needed, histopathological exam.

Patch and photopatch testing

Patch and photopatch testing are essential procedures in daily routine to investigate allergic and photoallergic CD. Patch testing is considered the gold standard to diagnose contact allergy resulting from type IV hypersensitivity.[172] It should be performed in all patients with clinical manifestations of contact allergy, including its unusual forms.[172] In addition, some clinical situations in which patch testing is proposed are previously well-controlled chronic dermatoses that lose response to treatment, unexplained flares, dermatitis that manifests in an atypical distribution and new-onset dermatitis that responds to therapy but returns rapidly after treatment discontinuation.[173] Counselling patients regarding the allergen(s) involved, after interpretation of the clinical relevance of positive results, is essential to allow allergen avoidance. Furthermore, follow-up consultation can help to ensure the understanding and

TABLE 2 Diagnostic clues of non-eczematous conditions included in the differential diagnosis of contact dermatitis.

Condition	Predilection sites	Clinical (anamnesis and exploratory) clues
Lichen planus	Hands and wrists, feet and ankles, scalp, nails, generalized, mucosal (oral and genital)	Investigate past or associated disease (e.g., hepatitis C), systemic drugs (e.g., ACEIs, NSAIDs and thiazides) and use of dental amalgam. In nails: nail plate thinning, trachyonychia, dorsal nail pterygium. In mucosa: erosions, Wickham striae
LSC	Easily accessible locations	Investigate scratching habit and associated lesions
Psoriasis	Any area, including scalp and nails, may be generalized	Investigate family history, arthritis, obesity, triggers (infections, stress, tobacco, alcohol, medications). At exploration: pinpoint bleeding (Auspitz sign), presence of Koebner phenomenon, characteristic dermatoscopic features (regular dotted vessels in a red background with silvery scales)[85]
Lupus erythematosus	Face (acute or discoid), scalp (discoid), trunk (subacute), arms/legs (profundus) or generalized	Evaluate photosensitivity, associated systemic symptoms (mostly in acute and subacute forms) and baseline diseases. If associated systemic lupus erythematosus, consider systemic clinical hallmarks.[86] Wide spectrum of skin lesions depending on lupus subtype (acute, subacute or chronic)[87]
Dermatomyositis	Face, scalp, arms and legs, nails	Photosensitivity and presence of systemic symptoms or blood test hallmarks, even though amyopathic forms exist.[88] Skin findings: Gottron papules, Gottron sign, Holster sign, mechanic hands, heliotrope rash, hallux patch
PLE	Photo-exposed areas	Photosensitivity; usually spares the face and presents with recurrent episodes
Acne—Rosacea	Face	Use of occlusive topical products in the face and triggers (e.g., UV-exposure, alcohol, hot beverages and spicy foods)
Tinea	Face, scalp, hands/feet, arms/legs, generalized	Assess contact with affected individuals and/or animals; check the feet Dermoscopy may be helpful in the differential diagnosis (e.g., tinea capitis).[89]
Scabies	Hands and feet or generalized	Assess contact with affected individuals. Intense nocturnal itch. It spares the face (except children, immunocompromised). Typical hallmarks (e.g., delta wing sign)
Impetigo	Mostly face	Investigation of contact with affected individuals. Non-bullous: vesicular phase leading to honey-coloured crusts. Bullous: flaccid bullae with subsequent erosions
Erythrasma	Body folds	Evaluate obesity and hyperhidrosis. Clinical diagnosis favoured by Wood's lamp
Candidiasis	Body folds and genitals	Evaluate obesity, diabetes and/or hyperhidrosis. Erythema, maceration, peripheral 'milky-white' pustules and papules
Viral exanthems	Mostly generalized	Heterogenous clinical presentation with systemic prodromes
Herpes simplex	Localized lesions	Painful recurrent vesicular lesions in the same region
Hailey-Hailey disease	Body folds	Presence of hyperhidrosis or friction in the area
PCT	Mostly in hands and forearms	Evaluate alcohol use, oestrogen use, viral infections, iron overload and smoking Skin blisters, vesicles, bullae and increased fragility of the skin on sun-exposed body areas with secondary hyper- and hypo pigmented scars and milia.[90]
Bullous pemphigoid	Hands, forearms or generalized	Evaluate medication and neurological baseline diseases. Initially urticarial and papular lesions with pruritus, which are followed by vesicles and bullae on normal or erythematous skin. Usually spares the head region.
CTCL	Localized or generalized	Heterogenous presentation[91]
Mammary and extramammary Paget's disease	Body folds, nipple and areola, and/or genital	Evaluate baseline diseases and presence of local symptoms that could guide towards an underlying malignancy.
Bowen disease	Mostly extremities	Evaluate sun-exposure habits. Red, scaly patches/plaques with variable associated features (erosion, fissures, pigmented, nodular)[92]
Queyrat erythroplasia	Genital	Evaluate the presence of local symptoms Well-demarcated, velvety, shiny, bright red, plaque-like appearance[92]

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; CTCL, cutaneous T-cell lymphoma; LSC, lichen simplex chronicus; NSAID, nonsteroidal anti-inflammatory drug; PCT, porphyria cutanea tarda; PLE, polymorphic light eruption.

proper avoidance of identified allergens and causes of CD, which can result in reinforcing self-management skills of the patients. Patch testing improves the quality of life in patients with ACD.[174] Photopatch testing is indicated in case a photoallergic CD is suspected, since additional ultraviolet exposure is needed to induce the reaction. Its value as diagnostic tool has been recognized in the study of any dermatitis in photo-exposed areas, or for evaluating

TABLE 3 Clinical clues in the differential diagnosis of body regions with specific features (face and neck, hand and feet and body folds).

Condition	Location	Specific clues
Allergic CD	Face and neck	**Rinse-off pattern** (preauricular and mandibular areas)[12] **Geographic pattern** (area of application of a topical product)[12] **Bilateral patchy pattern** on the face (due to cosmetics)[12] **Airborne pattern** (upper eyelids, nasolabial fold, retroauricular and submental areas. The bridge and tip of the nose may be unaffected)[12] **Drip pattern** (lower eyelids and cheeks, due to topical eyedrops)[12] **Contour pattern** (bilateral periocular, mostly due to cosmetics)[12]
	Hands and/or feet	**Pincer grasp pattern** (due to allergic CD from acrylates, diallyl disulfide and food or irritant CD due to friction). See Figure 1a. **Periungual pattern** (allergic CD from nail cosmetics)[93] **Shoe pattern** (dorsum of feet sparing interdigital webs due to (photo)allergic CD to topical medications)[12] **Plantar pattern** (the arches and interdigital of skin may be spared)[12]
Irritant CD	Hands and/or feet	**Apron pattern** (interdigital spaces and extending to palmar and dorsal surfaces) **Ring pattern** (under the ring area).[12] Rule out metal allergy if unfavourable course **Glove pattern** (dorsum of the hand, wrists and fingers eczema without interdigital involvement).[12] It can be seen in allergic CD
	Body folds	Examine contact with body fluids and use of diapers/daily pads
Atopic dermatitis	Face and neck	**Cheek dermatitis** in children **Retroauricular** dermatitis with **fissuring**[94] (also seen in irritant CD)
	Hands and/or feet	Dorsal hand with involvement of fingers and wrists. Less palmar involvement.[95]
Seborrheic dermatitis	Body folds	Ill-defined salmon-coloured thin patches
Photoallergic CD	Face and neck	On the bridge of the nose, forehead and cheeks. Upper eyelids, retroauricular and submental areas are spared.[12]
Psoriasis	Hands and/or feet	**Palmar grasp pattern**. Concomitant involvement of the dorsum of the hands and wrists may point to allergic CD.[12]
	Body folds	**Inverse psoriasis**: symmetrical well-defined shiny red patches **Fissures/erosions in folds** may be indicative of psoriasis,[96] especially in the intergluteal crease.
Lichen planus	Hands and/or feet	Typical clinical features, particularly on wrists and ankles, but also with a hyperkeratotic papular pattern, erythematous scaly pattern or even verrucous lichen planus.[1,97]
Lupus erythematosus	Face and neck	**Butterfly shaped** malar rash (acute), **annular/psoriasiform** lesions on the neck (subacute) and/or discoid **atrophic** lesions in head and neck and scalp (chronic).[87]
Dermatomyositis	Face and neck	Scalp seborrheic-like rash, heliotrope rash, facial erythema, shawl sign
Dyshidrotic eczema	Hands and/or feet	Vesicles on lateral and dorsal sides on fingers. **Palmar grasp pattern** may be associated.[12]
Erythrasma	Body folds	Brown or dark red, asymptomatic, persistent patches
Hailey-Hailey disease	Body folds	Erythematous plaque containing painful vesicles that rupture and form erosions, typically in the border of the lesions
Mammary and extramammary Paget's disease	Body folds, nipple and areola	Well-demarcated plaques with erosive, ulcerated, scaly or eczematous pattern

Abbreviation: CD, contact dermatitis.

photosensitivity due to systemic drugs.[175] Although elaborate information about these techniques is beyond the scope of this article, detailed recommendations are found in previous reference works.[172,175]

Histopathological findings of contact dermatitis and its differential diagnoses

This section details the main histopathological hallmarks of CD and the most representative conditions included in its differential diagnosis.

The histopathology term 'spongiotic dermatoses' encompasses eczematous disorders, highlighting the predominant epidermal changes in the form of spongiosis, which is the intercellular oedema between keratinocytes.[176,177] Spongiosis may associate with dermal and epidermal T-cell infiltration.[176,177] Spongiosis, however, does not occur only in eczematous disorders, but can be present in many other inflammatory and infectious disorders (e.g., viral exanthems, psoriasis and lichenoid reactions).[177]

The differentiation of eczematous disorders by means of other histopathological characteristics is very difficult. In addition, depending on the features of the reaction, the

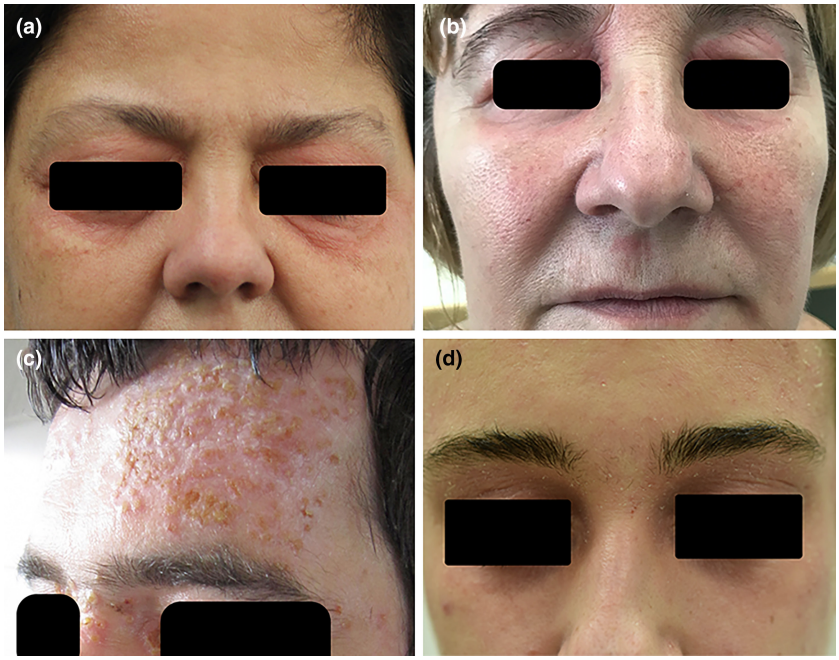

FIGURE 4 Dermatoses involving the face. (a) Contour pattern in allergic contact dermatitis from cosmetics (b) Dermatomyositis with subtle cheek erythema and heliotrope periocular oedema and erythema (c) Facial eczema herpeticum (d) Severe facial eczema in an atopic patient, which should raise suspicion about associated allergic contact dermatitis.

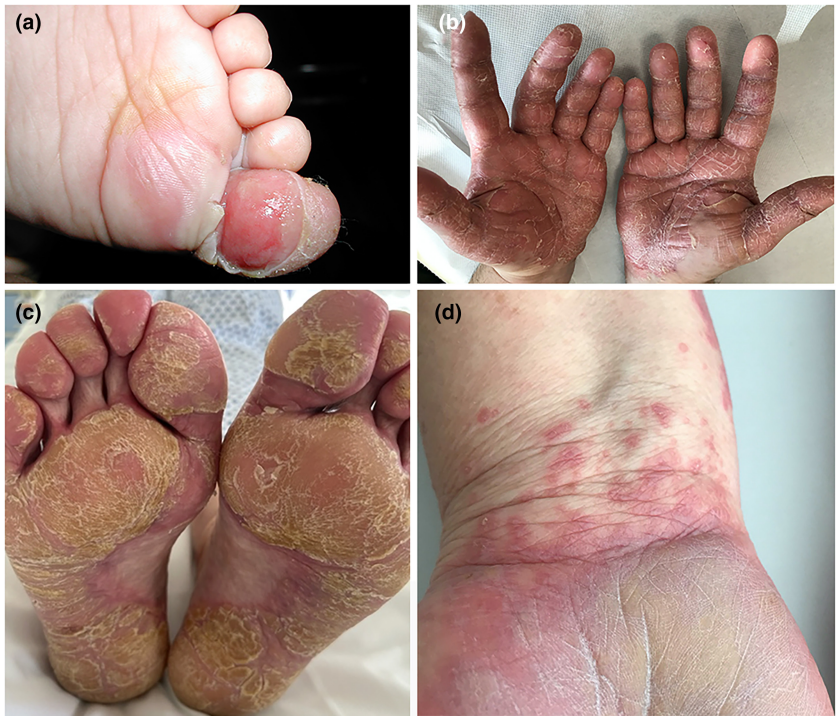

FIGURE 5 Dermatoses involving hands or feet. (a) Erosive phase of a bullous fixed drug eruption. The differential diagnosis of this lesion could include shoe allergic contact dermatitis; however the evaluation with anamnesis of drug intake and previous episodes led to the right diagnosis. (b) Palmar psoriasis with hyperkeratosis, desquamation and induration. (c) Acquired palmoplantar keratoderma which was diagnosed as mycosis fungoides after histopathological exam. (d) Lichen planus involvement of the proximal palmar aspect of the hand with the rather typical lilac papules on the wrist.

histopathologic hallmarks are variable. Acute dermatitis is characterized by severe spongiosis with intraepidermal vesicles,[178] and even eosinophilic spongiosis, particularly in ACD.[177] In subacute dermatitis, the spongiosis becomes less intense, even very mild in chronic dermatitis. Furthermore, chronic stages will show more acanthosis,

TABLE 4 Contact dermatitis: the great mimicker and its simulators.

	CD as a mimicker	CD simulators
Inflammatory	• Other eczematous disorders (atopic dermatitis, seborrheic dermatitis)[28,131,132] • Drug reactions (e.g., acute generalized exanthematous pustulosis and anti-IL-17 reactions)[48,49,133,134] • Psoriasis[135–137] • Lichen planus[138] • Lichen nitidus[139] • Prurigo nodularis[140] • Folliculitis decalvans[141] • Pemphigus vulgaris[142] • Aphthous stomatitis[143] • Angioedema and contact urticaria[144–147] • Lupus erythematosus*[73] • Dermatomyositis*[73] • Polymorphic light eruption**[140] • Actinic prurigo**[148]	• Other eczematous disorders (atopic dermatitis, dyshidrotic eczema, asteatotic eczema, seborrheic dermatitis, nummular eczema)[1,2,7] • Drug reactions (e.g., erythema multiforme and fixed drug eruption)[50,51] • Psoriasis[1] • Lichen planus[162] • Hailey-Hailey disease[1] • Bullous pemphigoid[163,164] • Rosacea[165] • Sweet syndrome[166] • Prurigo pigmentosa[167] • Lupus erythematosus*[73] • Dermatomyositis*[73,168] • Polymorphic light eruption**[73] • Solar urticaria**[73] • Actinic prurigo**[73] • Hydroa vacciniforme**[73] • Cutaneous porphyrias**[73,169] • Dermatitis herpetiformis
Neoplastic	• Mammary and extramammary Paget's disease[149] • Cutaneous T-cell lymphoma (mycosis fungoides)[150–153] • Basal cell carcinoma[154]	• Cutaneous T-cell lymphoma (mycosis fungoides) • Parapsoriasis en *plaque* • Bowen's disease • Mammary and extramammary Paget's disease • Erythroplasia of Queyrat
Infectious	• Herpes simplex[155,156] • Herpes zoster[157] • Impetigo[158]	• Herpes zoster • Impetigo • Scabies[170] • Tinea[74] • Erythrasma • Cellulitis[171]
Others	• Child abuse[159] • Burn[160] • Ischaemia[161]	• Dermatitis artefacta

Note: (*): contact dermatitis and/or photocontact dermatitis; (**): photocontact dermatitis.
Abbreviation: CD, contact dermatitis.

parakeratosis and hyperkeratosis.[179] Spongiotic dermatoses may encompass combined histopathological changes of acute, subacute and chronic dermatitis. Other non-specific findings are perivascular lymphocytes and eosinophils and neutrophils in the stratum corneum (e.g., if secondarily impetiginized).[179] Some particular features may be more characteristic of some types of eczema, which are outlined in Table 5.

Examples of frequently used additional diagnostic tools

The repeated-open-application-test (ROAT) aims to clarify the relevance of a selected doubtful or positive patch test to a product—or its ingredients. This technique can occasionally be more sensitive than patch testing, especially when it concerns ACD from low-concentrated ingredients in the tested product (patch test negative, ROAT positive). The test involves applying the product, or ingredient, which is suspected as a cause of ACD, two times a day on the volar side of the forearm for at least 10 days. Cosmetics and topical drugs are typically tested with this method.[172,188]

The semi-open test has been suggested as a method to study products that may contain irritants (e.g., shampoos, detergents and paints). Normally, it is used with patient-supplied products. Before testing an aqueous product, its pH needs to be verified. Following full evaporation of the product (and observation of any immediate reactions), the completely dried site is covered with tape and readings are performed as with regular patch tests.[189]

Open tests have been suggested as the first step for testing poorly defined products, such as patients' own materials (if they effectively come in contact with the skin), and for products suspected to cause immediate contact reactions, before proceeding to prick testing.

Prick tests, including prick-by-prick tests (e.g., food), are useful diagnostic tools for the diagnosis of contact urticaria and PCD, as well as identifying specific IgE against suspected allergens.

TABLE 5 Histopathological clues of contact dermatitis and its main differentials.

Conditions	Histopathology
Eczematous disorders	
Allergic CD	• Spongiosis is more prominent in the lower epidermis[176,177] • Spongiotic vesicles present at different epidermal levels (distinctive from nummular dermatitis)[177] • Intraepidermal Langerhans cells persist longer and are more distributed[176]
Irritant CD	• Changes vary depending on the causative agent and concentration of the product.[180] • Ballooning of keratinocytes in the upper and mid epidermis[177] • Variable epidermal necrosis and associated neutrophils[177] • Psoriasiform hyperplasia in chronic phases[177]
Atopic dermatitis	• Dermal eosinophils frequently seen[177] • Intraepidermal collections of Langerhans cells are rare[181]
Seborrheic dermatitis	• In acute stages, overlying scale crust containing neutrophils[177] • In subacute stages, less spongiosis and more psoriasiform hyperplasia and acanthosis
Photo-induced CD	• Photoallergic CD is similar to allergic CD • Phototoxic dermatitis: abundant apoptotic keratinocytes, analogous to a sunburn[182]
Dyshidrotic eczema	• Intraepidermal vesicle formation • Vesicles displace acrosyringia at the periphery of the bleb[177]
Stasis dermatitis	• Variable epidermal features: spongiosis, acanthosis, parakeratosis • Hallmark dermal changes: extravasated erythrocytes, hemosiderin-laden macrophages and proliferation of dilated small blood vessels in the papillary dermis[183]
Psoriasis[184]	• Regular acanthosis, with elongated rete ridges • Thinning of the suprapapillary layer of the epidermis • Areas of parakeratosis with collections of neutrophils (Munro microabscesses) • Collections of neutrophils in the spinosum plate (spongiform pustules of Kogoj) • Alternating hypo/hypergranulosis
Lichen planus[184]	• Compact hyperkeratosis without parakeratosis • Hypergranulosis and irregular acanthosis • Basal layer liquefaction • Lichenoid lymphocytes in the papillary dermis • Direct immunofluorescence: immunoglobulins, complement and fibrin stain some keratinocytes in the deeper epidermis and outermost dermis
Drug reactions	• Very variable features • Dermal eosinophils and eosinophilic exocytosis may be noted[184] • Spongiotic features. In morbilliform eruptions, spongiosis and exocytosis may be limited to basal portions of the epidermis.[185] • Papillary dermal oedema • Apoptotic keratinocytes
Lupus erythematosus[184]	• Variable epidermal findings (from atrophy to hyperplasia), depending on the cutaneous form of lupus. • Basal layer liquefaction (interface dermatitis); thickening of the basal membrane • Perivascular and peri-adnexal lymphocytes, sometimes acquiring a lichenoid pattern • Increased mucin deposition • Usually no neutrophils or eosinophils (except in neutrophilic or bullous lupus; eosinophils may be present in lupus tumidus)
Dermatomyositis	• Similar to cutaneous lupus erythematosus, but without vascular ectasia, fibrin and C5b-9 deposition in dermal vasculature and dermo-epidermal junction.[186,187]
Mycosis fungoides	• Epidermotropism with Pautrier microabscesses • Most common phenotype: CD2+, CD3+, βF1+, CD4+, CD8-, CD45RO with loss of mature T-lymphocytes markers (CD7, CD2 o CD5)[91]

Abbreviation: CD, contact dermatitis, except in the immunophenotype of mycosis fungoides.

CONCLUSION

Contact dermatitis is a multi-faceted and very frequently encountered disease in our clinical practice. Because CD mimics other skin conditions, and as other skin diseases may also simulate CD, a good understanding of diagnostic clues pointing towards CD, and its differential diagnoses, is crucial. To this end, we have here presented a practical review, including visual materials, to facilitate its diagnosis. As discussed, the evaluation of several clinical parameters, as well as ruling out other skin conditions, and performing complementary tests are required. Pragmatically, including CD in the differential diagnosis of many inflammatory and non-inflammatory skin conditions may lead to earlier diagnosis, especially of atypical cases, and might even reduce the need to perform further,

sometimes invasive, complementary tests. Similarly, if an external factor is found, and a relevant (photo-)contact allergy is present, counselling and eviction of the demonstrated (photo)allergens can be pursued, with a change in the natural course of the disease and even the avoidance of systemic treatments to control the condition.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The patients or the patient's parents/guardians in this manuscript have given written informed consent to the publication of their case details.

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