BMJ Best Practice

Dyshidrotic dermatitis

Straight to the point of care

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Summary

Dyshidrotic dermatitis is a chronic, relapsing-remitting vesicular eruption of the palms and soles; classically pruritic; also known as dyshidrotic eczema dermatitis.

The common exacerbating factor is irritation, as seen in frequent hand washing, hyperhidrosis, and stress. However, the underlying aetiology is unknown.

Diagnosis is based on characteristic history and physical examination.

The foremost objective in treatment is identification and avoidance of exacerbating factors.

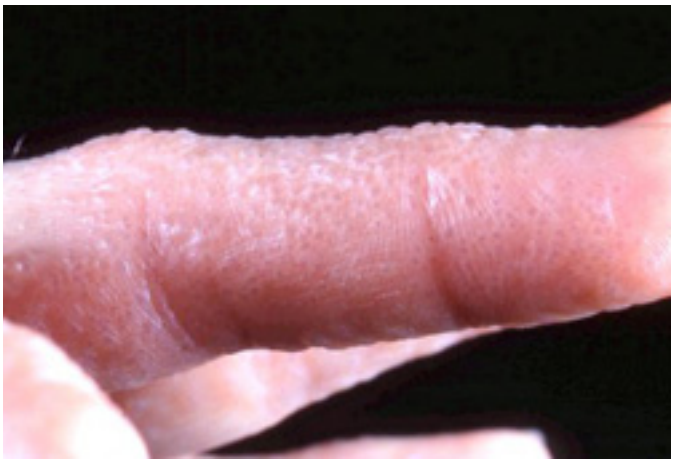
First-line therapy is topical corticosteroids or immunomodulators.

For severe eruptive bullae on the palms and soles, which are consistent with pompholyx, a short-term course of oral corticosteroids may be helpful as a temporising measure.

Other therapeutic options, if symptoms don't respond to initial treatment, include phototherapy, oral immunomodulators, and nickel-directed therapy.

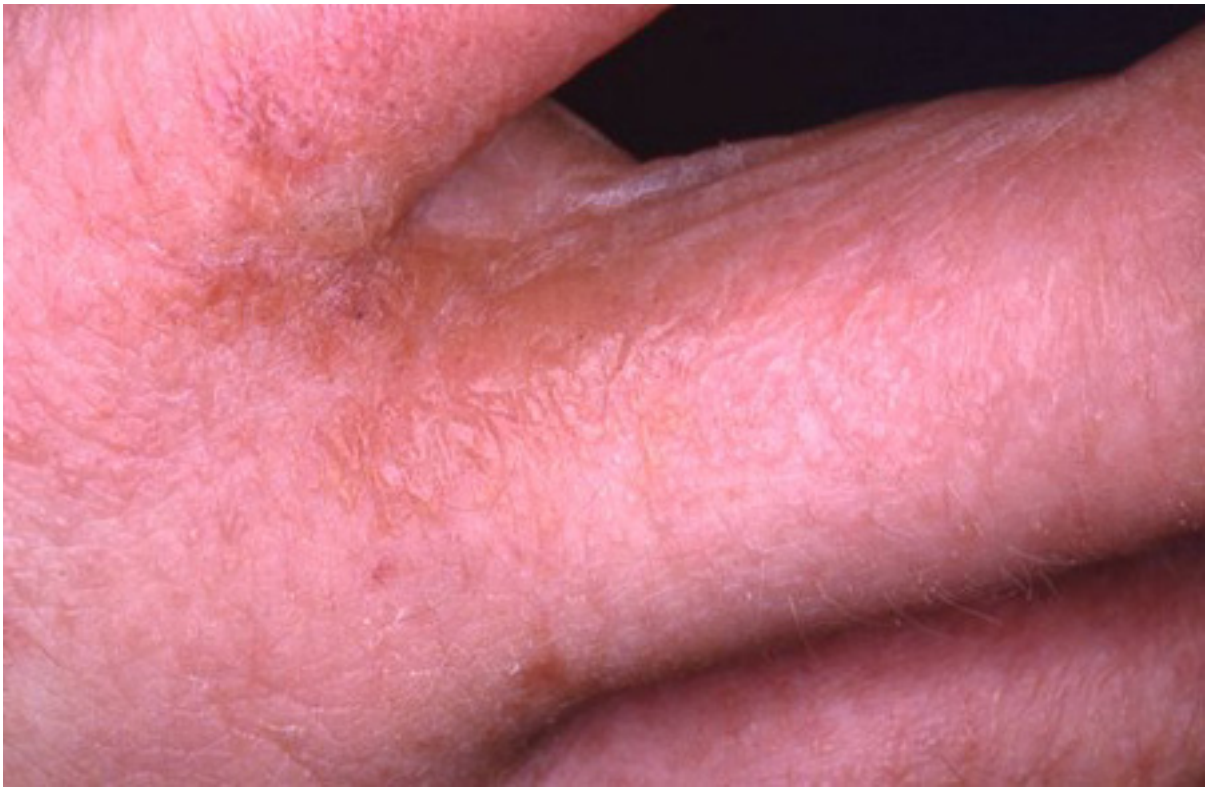
Definition

Dyshidrotic dermatitis is a form of chronic dermatitis affecting the hands and feet. It is characterised by recurrent crops of 1- to 2-mm vesicles on the palms, soles, often appearing on the medial and lateral aspects of the fingers and toes.[1] Crops persist for 2 to 3 weeks and return at variable intervals. Pruritus accompanies the eruptions and may even precede them. Pompholyx is a term often used synonymously with dyshidrotic dermatitis, but some advocate for the term to be reserved for more acute, severe eruptions of large bullae on the hands and feet.[2]



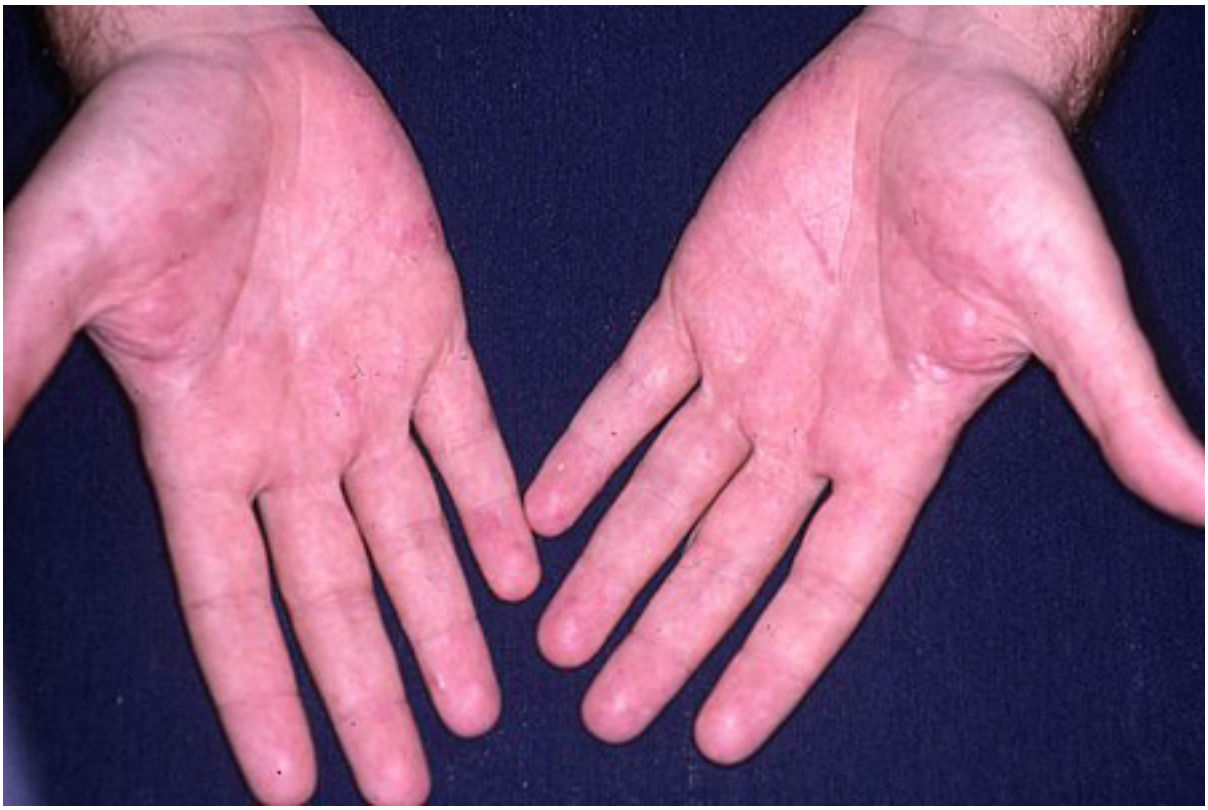
Dyshidrotic eczema

Photograph courtesy of Dr Spencer Holmes; used with permission



Dyshidrotic eczema

Photograph courtesy of Dr Spencer Holmes; used with permission



Dyshidrotic eczema

Photograph courtesy of Dr Spencer Holmes; used with permission

Epidemiology

Epidemiological data on dyshidrotic dermatitis as a single condition (i.e., not under the wider umbrella of hand dermatitis) is lacking.[1] A large Swedish study found the prevalence of dyshidrotic dermatitis in a general population of 107,206 people to be 0.05%.[3] Another study in Portugal reported that, of 714 subjects with hand dermatitis, 20% had dyshidrotic dermatitis.[4] An analysis of insurance claims for dyshidrotic eczema from 27 million people in the US with private healthcare coverage revealed 34,932 patients were diagnosed in 2018 with 214,974 visits in that year. The average age was 37.1 years and most patients (61.0%) were female.[5]

Aetiology

Dyshidrotic dermatitis is of unknown aetiology. A number of risk factors and potential contributing factors have been investigated. There is conflicting evidence about whether atopy confers increased risk.[6] [7] Some investigators have found that contact allergy (especially to metals) is correlated with dyshidrotic dermatitis, while others have not.[8] [9] Nickel-sensitive patients may have flares of dyshidrotic dermatitis following dietary exposure or systemic administration of the metal.[10] [11] The high eccrine duct density on the palms and soles may lead to concentration of metal on these surfaces when sweating leading to flares in nickel-sensitive patients.[12] Hyperhidrosis appears to be an aggravating condition for a subset of people with dyshidrotic dermatitis.[6] [13] Common irritants such as water, detergents, and solvents may also have a role in exacerbating the condition in some patients.[14] [15] One study demonstrated a correlation between dyshidrosis and smoking, oral contraceptives, and acetylsalicylic acid.[7] Emotional stress can also trigger outbreaks of dyshidrotic dermatitis, but the condition is not linked to sex or age.[3] [4] [6] [8] [16]

Pathophysiology

Dyshidrotic dermatitis is not related to an alteration in eccrine ducts as is suggested by its name. On histopathology, it is simply spongiotic dermatitis in the setting of acral skin. It is the thickened stratum corneum that causes the classic 'tapioca' appearance of dyshidrotic dermatitis by its impedance to vesicular rupture.[17] The intra-epidermal portion of eccrine ducts is unaffected by this process.[17]

Case history

Case history #1

A 40-year-old woman presents with intensely pruritic rash of the hands and feet that is relapsing-remitting. She recalls that, 1 or 2 days prior to an eruption, she will notice an itching sensation in her palms and soles. The rash consists of small vesicles on the palms, soles, and lateral fingers. It lasts for a couple of weeks before desquamation, leaving no trace. She has several episodes a year and cannot cite specific triggers, although she does note that washing dishes makes it worse. Her medical history is otherwise unremarkable.

Approach

A detailed history and physical examination is usually sufficient to diagnose dyshidrotic eczema, however, special tests may be warranted to rule out other processes, if clinical presentation is suspicious.

History

The classic history is of pruritic, sometimes painful, vesicles on the soles, palms, medial and lateral aspects of the fingers and toes. These persist for several weeks then desquamate. Eruptions recur chronically at variable intervals.

Physical examination

On physical examination there are 1- to 2-mm vesicles on a non-inflammatory base which are located on the palms, soles, medial and/or lateral aspects of the fingers and toes. Hyperhidrosis is a frequently reported exacerbating factor and/or co-morbidity.[6][15] [19] A subset of patients with psoriasis have symptoms limited to the palms and soles, which may be difficult to distinguish from dyshidrotic dermatitis although, most will also have characteristic salmon-coloured plaques with overlying silvery scales on other sites, particularly the elbows and knees. Similarly, patients with vesiculo-bullous disease (e.g., pemphigus or pemphigoid) may exhibit vesicles or bullae at acral sites but most commonly exhibit the eruption elsewhere on the body.

Histology

Biopsy is not typically required for diagnosis. It is necessary if there is clinical difficulty in distinguishing the condition from psoriasis, palmoplantar pustulosis, or bullous pemphigus. When biopsy is performed, spongiotic dermatitis in acral skin is observed in the case of dyshidrotic dermatitis. Immunofluorescence is negative, ruling out immune-mediated bullous disorders.

Laboratory

Not routinely performed as diagnosis is clinical. However, a potassium hydroxide (KOH) test for bullous tinea, an epidermal scraping with mineral oil to evaluate for scabies infestation, or patch testing to evaluate contact allergy can be performed to rule out differential diagnoses. Possible allergens include personal hygiene products, occupational exposures, and metals.[20]

History and exam

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include exposure to irritants.

skin lesions (common)

- Non-erythematous 1- to 2-mm vesicles located on the palms, soles, medial and/or lateral aspects of the fingers and toes.

pruritus (common)

- Dyshidrotic dermatitis is characteristically highly pruritic. A prodrome of pruritus is common.

Other diagnostic factors

hyperhidrosis (common)

- This is a frequently reported exacerbating factor and/or co-morbidity in patients with dyshidrotic dermatitis.[6] [19]

recurrent eruptions (common)

- May recur over weeks to months.

pain (uncommon)

- Some patients complain of pain more than pruritus.

Risk factors

Strong

exposure to irritants

- Exposure to irritants, such as water, detergents, and solvents, may be an exacerbating factor in some patients with dyshidrotic dermatitis.[15] [18]

Weak

atopy

- Some investigators have found an association between atopy and dyshidrotic dermatitis, while others have reported no correlation.[6] [7]

metal allergy

- There is conflicting evidence of an association between dyshidrotic dermatitis and metal allergy.[8] [9] Exposure to the offending metal in allergic patients may cause a flare and these patients should avoid such contact. Nickel is the most commonly implicated metal, but other metals such as chromium and cobalt are also possible exacerbating factors.

hyperhidrosis

- Hyperhidrosis may be an aggravating factor for a subset of patients with this condition as there are reports of improved dyshidrotic dermatitis after treating hyperhidrosis.[6] [13]

emotional stress

- Emotional stress can trigger outbreaks of dyshidrotic dermatitis.[16]

Investigations

1st test to order

Test	Result
clinical diagnosis • Usually no tests are necessary.	**features of dyshidrotic dermatitis**

Other tests to consider

Test	Result
skin biopsy • This test is rarely ordered, as dyshidrotic dermatitis is a clinical diagnosis. It is useful when other diagnoses, such as bullous disorders, palmoplantar pustulosis, or psoriasis, are being considered.	**histology reveals spongiotic dermatitis of acral skin; immunofluorescence is negative**
potassium hydroxide (KOH) • KOH may be used to rule out bullous tinea, especially when clinical onychomycosis is present.	**negative**
patch testing • To evaluate contact allergy. • Possible allergens include personal hygiene products, occupational exposures, and metals.[20]	**numerous allergens are possible, including allergy to the corticosteroid used to treat the patient**
skin scraping • An epidermal scraping with mineral oil to evaluate for scabies infestation. • Consider this test if excoriated papules are present elsewhere on the body (other than the hands and feet).	**negative**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Tinea manuum/pedis	• Tinea may rarely present with vesicles. • Patients with tinea often have prominent scale and may also have maceration, especially between the toes, and thickened toenails (onychomycosis).	• Potassium hydroxide (KOH) prep will be positive, with fungal hyphae appreciated, in tinea. • Consider sending a skin scraping for fungal culture if further confirmation is desired.
Palmoplantar psoriasis	• Pustular palmoplantar psoriasis is characterised by pustules on the palms and soles, as opposed to the vesicles of dyshidrotic dermatitis. • Classic lesions of psoriasis (thick, scaling plaques on the elbows and knees) may be present.	• Skin biopsy of dyshidrotic dermatitis will reveal spongiotic dermatitis. • Psoriasis will have characteristic findings such as psoriasiform hyperplasia and Munro micro-abscesses.
Allergic contact dermatitis	• History may reveal exacerbating factors in contact allergy. In addition, the lesions are more inflammatory than those of dyshidrotic dermatitis. • Allergy to topical corticosteroids can be a particularly vexing problem because the dual competing effects of applying the medication in this situation are decreasing inflammation and propagating the allergic response.	• Patch testing confirms suspected contact allergy.
Scabies	• Erythematous papules, especially in the finger webs on the hand. • Excoriated papules are likely to be found elsewhere in scabies infestation, especially the genital region and umbilicus. • Burrows may be seen. • Close contacts of the patient will often have a similar eruption.	• A visualisation of skin scrapings from affected areas using mineral oil, glass slides and light microscopy revealing presence of a mite, scybala, or eggs would distinguish scabies from dyshidrotic dermatitis.
Pemphigus	• Pemphigus involves skin other than acral sites and is often characterised by	• Skin biopsy will have positive immunofluorescence and

Condition	Differentiating signs / symptoms	Differentiating tests
	flaccid or ruptured bullae as opposed to tense bullae.	characteristic findings on histopathology.
Bullous pemphigoid	• Bullous pemphigoid involves skin other than acral sites and may also present with urticarial lesions.	• Skin biopsy will have positive immunofluorescence and characteristic findings on histopathology.
Irritant contact dermatitis	• Irritant contact dermatitis often presents with burning and pain sensation over itch. Primary lesions can be hyperlinear and scaly. Hands, and particularly the dorsal surface due to reduced skin thickness are more often affected than the feet.	• A detailed history of possible exposures to irritants will help assess this as a possible differential.

Criteria

Dyshidrotic eczema Area and Severity Index (DASI)[21]

The DASI is a severity index scale based on a score of the following factors: number of vesicles per square centimetre; erythema; desquamation; itch; and extension of the affected area. The grades are as follows: mild (0 to 15), moderate (16 to 30), and severe (31 to 60). It is utilised more as a research tool than in clinical practice.

Approach

Dyshidrotic dermatitis characteristically follows a relapsing-remitting course and so will most often resolve without any treatment, however, patients will appreciate therapies to lessen the duration and prevent recurrence of uncomfortable eruptions.

Lifestyle measures

Advise patients to avoid identified triggers or exacerbating factors. Provide all patients with strategies to maintain effective skin barrier mechanisms such as frequent use of emollients, avoidance of irritants, and use of protective gloves or footwear.[22] Advise against prolonged wet work and harsh cleansers, and suggest immediately moisturising with a heavy emollient, preferably a white soft paraffin-based product, after each exposure to water.[22] In addition to these measures, some patients require further adjunctive therapies:

Pruritus

- Symptomatic control of pruritus may be achieved with oral antihistamines.

Hyperhidrosis

- Hyperhidrosis is an exacerbating factor for some patients with dyshidrotic dermatitis.
- In this subset of patients, modalities that interrupt eccrine sweat function, such as topical aluminium chloride, iontophoresis, topical glycopyrronium, and botulinum toxin type A, may be helpful.[23] [24] [25]
- These treatments are relatively safe, although an adverse effect of botulinum toxin type A is temporary hand weakness.
- Botulinum toxin type A is preferred over iontophoresis.

Nickel allergies

- Several studies suggest a systemic role for nickel-related exacerbation of dyshidrotic dermatitis in nickel-allergic patients.[26] [27]
- After nickel-sensitive patients are identified through patch testing or oral challenge, they should stringently avoid contact with all objects containing nickel. A dimethylglyoxime kit is used to test frequently encountered objects for nickel content.
- If after these precautions the patient is still experiencing flares, some authors recommend attempting to decrease systemic nickel exposure.[27] [28] A diet low in nickel is useful in some motivated patients with stubborn dyshidrotic dermatitis and a nickel allergy proven via patch-test or oral challenge.[27] [28]
- Other methods employed for decreasing the systemic effects of nickel are treatment with oral agents (such as vitamin C, iron, disulfiram and sodium cromoglicate) and hyposensitisation.[29] [30] [31] A metabolite of disulfiram binds divalent metals, including nickel, while sodium cromoglicate inhibits mast cell degranulation. Iron deficiency anaemia is understood to enhance nickel absorption (secondary to upregulation of divalent metal transporter 1 [DMT1] on enterocyte luminal surfaces) and is treated with iron supplementation. Vitamin C acts to suppress nickel absorption.[32]
- However, nickel-directed therapies, as a whole lack large, randomised, controlled studies.

Patients unresponsive to lifestyle measures

If the condition persists after conservative lifestyle measures are taken, topical corticosteroids are frequently used.[15] [33] [34] A potent preparation may be needed for the first couple of weeks to gain control of the eruption. This is then tapered to a less potent formulation for the remainder of the treatment course.[35]

Topical immunomodulators, such as tacrolimus and pimecrolimus, are helpful corticosteroid-sparing agents for long-term maintenance and when there are concerns about skin atrophy with topical corticosteroids.[15] [36] [37] [38] However, topical immunomodulators do not penetrate thick skin well, so if these agents are used on thickly keratinised skin, a keratolytic (such as a cream containing urea) can be coadministered to improve absorption.[36] [37]

Lifestyle measures may need to be continued in selected patients, despite escalation of therapy.

Patients unresponsive to topical therapies

A short course of oral prednisolone is sometimes helpful if patients' symptoms are severely flared and cannot be controlled with skin-directed methods.[2] Long-term use of oral corticosteroids is discouraged because of the well-known range of adverse effects. Topical corticosteroids and lifestyle measures are continued if necessary.

Recalcitrant disease

Patients at this level of severity should seek consultation with a dermatology speciality clinic.

Phototherapy or immunosuppressants may be considered. Topical psoralen (e.g., methoxsalen) plus ultraviolet A (PUVA) and narrowband UVB are effective measures.[15] [38][39] [40] [41] Oral PUVA is a second-line option as, while effective, it can carry a risk of skin cancer with repeated exposure.

Systemic immunosuppressants are used with some success in patients with dyshidrotic dermatitis that is unresponsive to conventional therapy, and for recalcitrant disease unresponsive to phototherapy.[42] [43] [44] [45] However, evidence of efficacy for dyshidrotic dermatitis is weak.[38]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing (summary)		
all patients		
	1st	lifestyle measures
	adjunct	topical corticosteroid or immunomodulator
	adjunct	oral corticosteroid
	adjunct	antihistamine
	adjunct	nickel-directed therapy
	adjunct	therapy for hyperhidrosis
with recalcitrant disease	adjunct	hand/foot phototherapy
	adjunct	systemic immunosuppressant therapy

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing

all patients

1st **lifestyle measures**

» Avoidance of triggers or exacerbating factors identified for a specific patient.

» Advise frequent and liberal use of emollients on the affected areas, especially after any contact with water. Use white soft paraffin-based formulations, or a cream formulation if the patient does not tolerate this.

» Advise patients against prolonged wet work and suggest wearing protective gloves if necessary. Recommend use of mild cleansers only and avoidance of skin products with fragrances or dyes.

adjunct **topical corticosteroid or immunomodulator**

Treatment recommended for SOME patients in selected patient group

Primary options

» clobetasol topical: (0.05%) apply sparingly to affected area(s) once daily at bedtime for up to 2 weeks, then switch to lower-potency corticosteroid

OR

» fluocinonide topical: (0.05%) apply sparingly to affected area(s) twice daily for up to 2 weeks, then switch to lower-potency corticosteroid

OR

» mometasone topical: (0.1%) apply sparingly to affected area(s) once daily until resolution of symptoms

OR

» triamcinolone topical: (0.025% or 0.1%) apply sparingly to affected area(s) twice daily until resolution of symptoms

Secondary options

Ongoing

» tacrolimus topical: (0.1%) apply to affected area(s) twice daily

OR

» pimecrolimus topical: (1%) apply to affected area(s) twice daily

» Used in patients who are unresponsive to lifestyle measures alone.[15] [33] [34]

» Potent preparations (e.g., clobetasol, fluocinonide) are used for up to two weeks a month before switching to an intermediate-potency corticosteroid (e.g., mometasone, triamcinolone) to avoid adverse effects such as skin atrophy and telangiectasia. Maintenance therapy consists of a medium-potency topical corticosteroid.[35]

» Ointment-based formulations are generally considered superior to creams or lotions, as they are more effective and contain fewer preservatives and additives.[22] Occlusion with plastic wrap or hydrocolloid dressing enhances efficacy, however, it also increases the risk of adverse effects, especially with high-potency corticosteroids.[34]

» Topical immunomodulators such as tacrolimus and pimecrolimus are considered for chronic management and can be used for maintenance therapy (as corticosteroid-sparing agents) when there are concerns about skin atrophy after control is gained with topical corticosteroids.[15] [36] [37] [38]

» These agents are less effective on thickly keratinised skin such as on the soles. A keratolytic (such as a urea cream) to improve absorption may be used.[36] [37]

adjunct **oral corticosteroid**

Treatment recommended for SOME patients in selected patient group

Primary options

» prednisolone: 60 mg orally once daily for 3-5 days

» Reserved for short courses in severe circumstances (e.g., pompholyx). The addition of an oral corticosteroid can lead to dramatic improvement in cases unresponsive to lifestyle measures alone or topical therapy.

MANAGEMENT

Ongoing

» A short course of prednisolone is advised in these circumstances; dose tapering is not required for this treatment duration.[2] Longer treatment courses of oral corticosteroids are strongly discouraged due to their well-known adverse effects, which include hyperglycaemia, hypertension, osteoporosis, and cataracts.

adjunct **antihistamine**

Treatment recommended for SOME patients in selected patient group

Primary options

» diphenhydramine: 25-50 mg orally every 4-6 hours when required, maximum 300 mg/day

OR

» hydroxyzine: 25 mg orally every 6-8 hours when required

» Pruritus control may be achieved with oral antihistamines (e.g., diphenhydramine, hydroxyzine).

adjunct **nickel-directed therapy**

Treatment recommended for SOME patients in selected patient group

Primary options

» disulfiram: consult specialist for guidance on dose

OR

» sodium cromoglicate: 200-400 mg orally four times daily

» A diet low in nickel is helpful to some motivated patients with stubborn dyshidrotic dermatitis and a nickel allergy, proven by patch-test or oral challenge.[27] [28]

» A low-nickel diet includes avoidance of all canned foods and foods prepared with nickel-containing utensils and pots, as well as avoidance of the following foods: asparagus, beans, broccoli, carrots, corn, lettuce, mushrooms, onions, pears, peas, rhubarb, spinach, tomatoes, kale, alfalfa sprouts, leeks, lentils, pineapple, raspberries, prunes, dates, figs, herring, shellfish, tea, soy protein powder, chocolate, cocoa, baking powder, marzipan, nuts, sunflower and sesame seeds, licorice, vitamins containing nickel, whole wheat

Ongoing

flour, bran, buckwheat, millet, multi-grain bread, oatmeal, unpolished rice, and rye bran.

» Other methods employed for decreasing the systemic effects of nickel are treatment with oral agents (such as vitamin C, iron, disulfiram and sodium cromoglicate) and hyposensitisation.[29] [30] [31]

» However, nickel-directed therapies, as a whole, lack large, randomised, controlled studies.

adjunct **therapy for hyperhidrosis**

Treatment recommended for SOME patients in selected patient group

Primary options

» aluminium chloride topical: (20% solution) apply to the affected area(s) once daily at bedtime for 2-3 days or until anhidrosis, followed by once or twice weekly at bedtime when required

OR

» glycopyrronium topical: (2.4% pad) apply one pad to the affected area(s) once daily

OR

» botulinum toxin type A: consult specialist for guidance on dose

» Hyperhidrosis is an exacerbating factor for some patients with dyshidrotic dermatitis.

» In this subset of patients, modalities that interrupt eccrine sweat function, such as topical aluminium chloride, iontophoresis, topical glycopyrronium, and botulinum toxin type A, may be helpful.[23] [24] [25]

» Botulinum toxin type A treatment is associated with pain at the time of injections, and the adverse effect of temporary hand weakness. Careful dilution and injection techniques are necessary to prevent complications.[25]

» Botulinum toxin type A is preferred over iontophoresis.

with recalcitrant disease **adjunct** **hand/foot phototherapy**

Treatment recommended for SOME patients in selected patient group

MANAGEMENT

Ongoing

» Phototherapy can be a useful adjunctive treatment in patients who are partially responding to conventional therapy.

» Topical psoralen (e.g., methoxsalen) plus ultraviolet A (PUVA) is an effective treatment.[15] [38][39] [40] [41] Numerous regimens exist, but an 8-week regimen is often used. Topical PUVA has local adverse effects of blistering and hyperpigmentation, but the systemic adverse effects of nausea or vomiting and persistent whole-body photosensitivity seen with oral PUVA treatment are rare.[46]

» Oral PUVA is a second-line option; however, it carries a risk of ocular damage and skin cancer.[47]UV-A-1 does not require the use of psoralens (which increase the risk of carcinogenesis), but it is not widely available.[48].

» The major risk of PUVA is carcinogenesis, which is dose-dependent. Narrowband UVB is another effective option.[15] [38] In the active management phase, patients are treated three times a week. Patients are given an 8-week trial of active management ,after which treatments can be tapered down in the maintenance phase.

adjunct **systemic immunosuppressant therapy**

Treatment recommended for SOME patients in selected patient group

Primary options

» methotrexate: 12.5 to 20 mg orally once weekly, taken on the same day of each week

--AND--

» folic acid: 1 mg orally once daily

OR

» azathioprine: 100-150 mg/day orally initially; reduce to 50-100 mg/day for maintenance dose

OR

» ciclosporin: 2.5 mg/kg/day orally given in 2 divided doses

OR

» mycophenolate mofetil: 1.5 g orally twice daily initially and taper dose gradually according to response

Ongoing

» Systemic immunosuppressants are used with some success in patients who are unresponsive both to conventional therapy and to phototherapy.[42] [43] [44] [45] However, evidence of efficacy for dyshidrotic dermatitis is weak.[38]

» These agents are only employed in treatment-resistant disease as their adverse effects can be serious. These include: hepatotoxicity, renal toxicity, pneumonitis, pancytopenia, increased risk of malignancy or infection, teratogenicity, hypertension, hyperlipidaemia, and oligospermia.

» Folic acid supplementation reduces the likelihood of haematological adverse effects of methotrexate.

» Patients at this level of severity should seek consultation with a dermatology speciality clinic.

Emerging

Excimer laser

The 308-nm excimer laser also affects modulation of inflammatory cells and plays a role in the treatment of hand dermatitis.[49] However, the increased cost and time associated with excimer laser means that treatment is typically logistically difficult to prescribe when compared to phototherapy with a hand/foot narrowband ultraviolet (UVB) box.

Alitretinoin

The retinoid alitretinoin has been extensively studied in hand dermatitis with some efficacy.[38] At present only limited data exists for use in dyshidrotic dermatitis, and more data is necessary before use is recommended.[50]

Dupilumab

Dupilumab, a monoclonal antibody against interleukin-4 receptor alpha, was established as a treatment for atopic dermatitis via two randomised, placebo-controlled phase 3 trials (SOLO1 and SOLO2) and is approved for this indication.[51] [52] Its use for chronic hand eczema, including vesicular subtypes, has been studied in phase 2b and observational trials where subjects achieved clinical and symptomatic improvement.[53] [54] There have also been published case reports and case series of successful use of dupilumab in dyshidrotic eczema.[55] [56] [57]

Janus kinase (JAK) inhibitors

The small molecule JAK inhibitors encompass a class of newer topical and oral medicines directed towards the JAK/STAT signalling pathway which are implicated in several inflammatory diseases including dermatitis.[52] Topical JAK inhibitors such as ruxolitinib, tofacitinib, and delgocitinib have demonstrated benefit in atopic dermatitis in phase 2 and 3 trials.[58] [59] Topical ruxolitinib is approved for atopic dermatitis and was studied for hand eczema.[60] The oral JAK inhibitors abrocitinib, upadacitinib, and baricitinib have randomised, controlled, clinical trial evidence for efficacy and treatment of atopic dermatitis with upadacitinib currently being studied for hand eczema.[61] [62] [63] [64] [65] [66] [67] [68] [69] There is a case report of treatment success with the upadacitinib for dyshidrotic eczema.[70]

Crisaborole

Crisaborole is a topical phosphodiesterase-4 inhibitor that is approved for atopic dermatitis, based on two large double blinded vehicle-controlled studies.[71] [72] Although it has not been studied specifically in dyshidrotic eczema, it has shown some efficacy in hand dermatitis in a single institution case series.[73]

Primary prevention

Patients with metal allergy should avoid contact with the implicated metal. Avoiding wet work and harsh detergents and soaps should help prevention.

Patient discussions

All patients with dyshidrotic dermatitis should practise excellent hand care by avoiding irritants, such as harsh soaps and prolonged water exposure, as well as frequently moisturising with a white soft paraffin-based (preferable) or cream emollient. Patients who identify exacerbating factors such as allergens should avoid contact with those factors. Gloves and protective footwear should be considered and worn to protect the skin barrier.[1]

FOLLOW UP

Monitoring

Monitoring

There is no specific monitoring required in dyshidrotic dermatitis other than drug monitoring in the management of the condition. The long-term goal is fastidious hand care and avoidance of triggers to reduce the need for medications, if possible.

Complications

Complications	Timeframe	Likelihood
secondary infection	**variable**	**low**
May occur from breaks in the skin caused by pruritus. Pruritus can be controlled with symptomatic treatment as well as by using more aggressive treatment options for dyshidrotic dermatitis to decrease disease intensity.		

Prognosis

Dyshidrotic dermatitis commonly follows a chronic relapsing-remitting clinical course. Most often, remission occurs spontaneously or is achieved with treatment, but recurrence is the rule.[1] Patients are best served by identification of any and all exacerbating factors, to avoid recurrences.

Diagnostic guidelines

Europe

Guidelines for diagnosis, prevention and treatment of hand eczema (https://pubmed.ncbi.nlm.nih.gov/34971008)

Published by: European Society of Contact Dermatitis **Last published:** 2022

Treatment guidelines

Europe

Guidelines for diagnosis, prevention and treatment of hand eczema (https://pubmed.ncbi.nlm.nih.gov/34971008)

Published by: European Society of Contact Dermatitis **Last published:** 2022

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Images

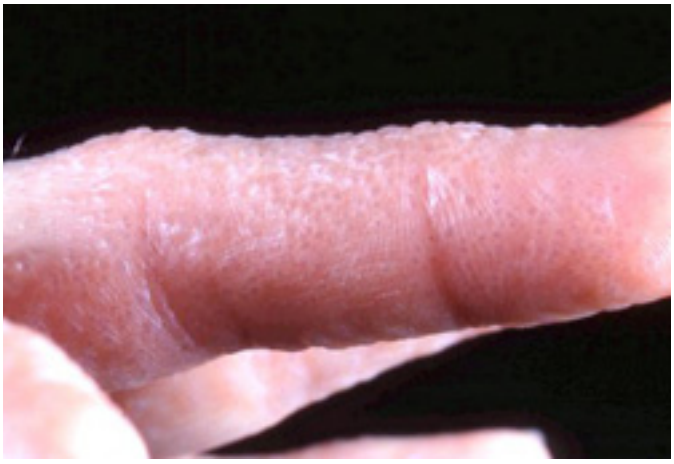

Figure 1: Dyshidrotic eczema

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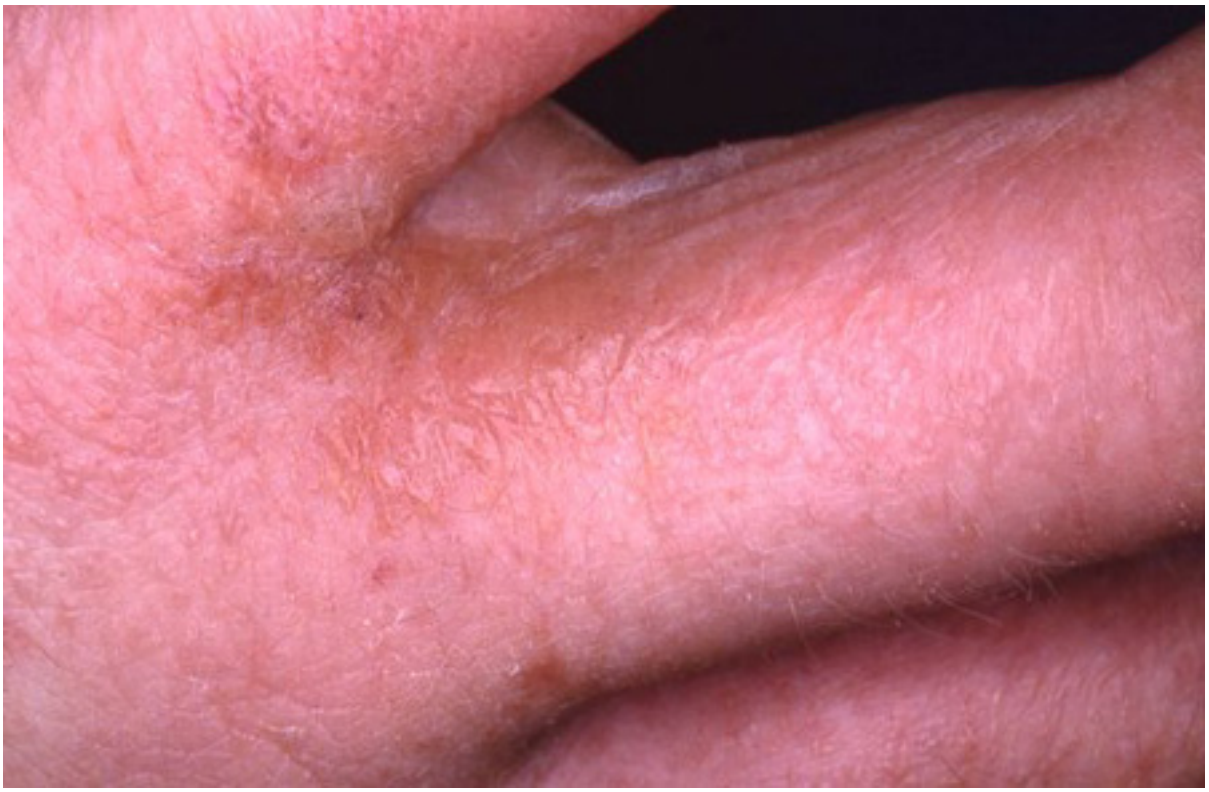

Figure 2: Dyshidrotic eczema

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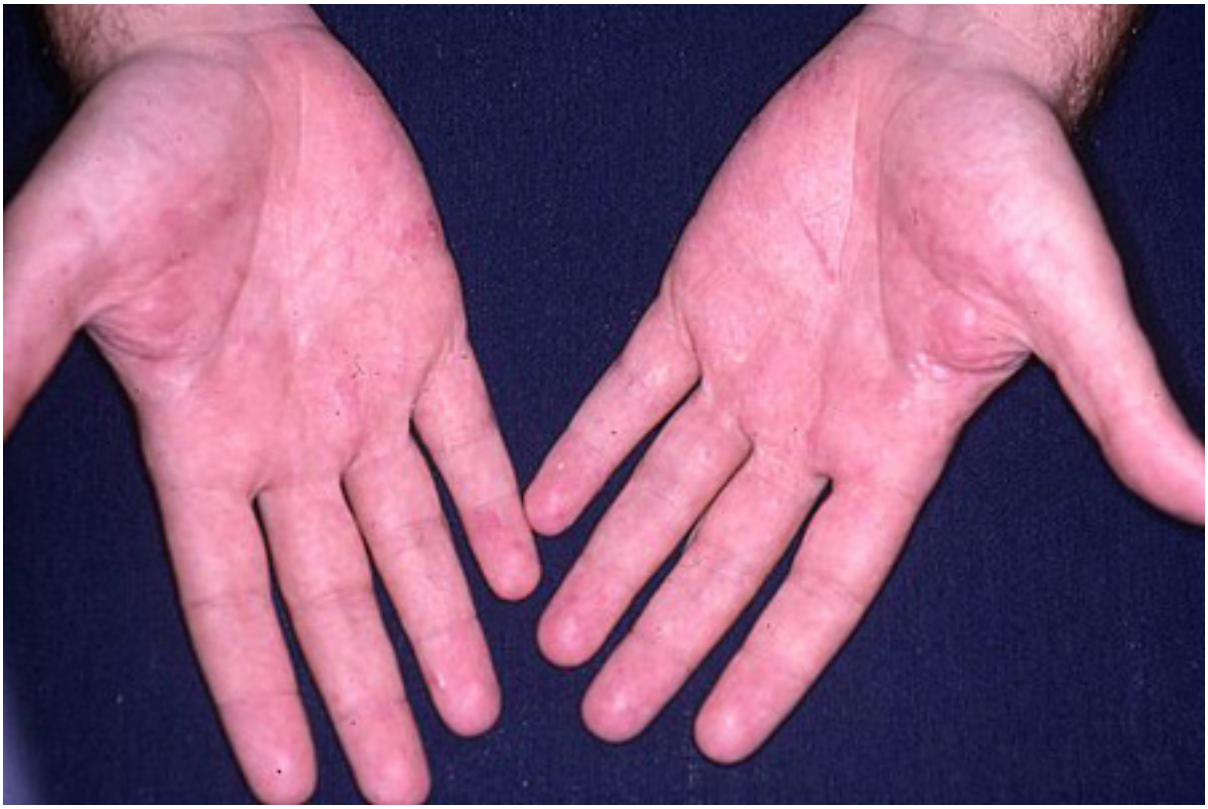

Figure 3: Dyshidrotic eczema

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Please note that recommended formulations and doses may differ between drug databases drug names and brands, drug formularies, or locations. A local drug formulary should always be consulted for full prescribing information.

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BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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