

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

'OLD FAITHFUL' GEYSER OF
NAPA VALLEY, CALIFORNIA

THE ECZEMAS



UPDATED CHAPTER
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From Greek: 'Eczema' to boil out

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

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DIAGNOSIS PREQUEL: A SYSTEMATIC APPROACH TO DIAGNOSING SKIN CONDITIONS

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CHAPTER TWO: PSORIASIS

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CHAPTER ONE: THE ECZEMAS

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Eczema is an all embracing term for a heterogeneous group of related dermatoses. The terms eczema and dermatitis are often confused but are wholly interchangeable. However, occupational “dermatitis” still seems to equate with the spectre of compensation!

It is important to define which type of eczema we are dealing with, as there are several major distinct patterns, although these often coexist/overlap. Treatment plans, though, may be very different. We will deal with the most common categories of eczema, namely atopic, seborrhoeic, discoid, contact, varicose, asteatotic and pompholyx.

ATOPIC ECZEMA



Atopy is a syndrome of allergic hyper-reactivity. The clinical manifestations are asthma, allergic rhinitis and eczema. Atopic eczema is from the Greek a-topos, meaning alien, and eczema, meaning to boil out. It is a very significant problem involving up to 15 – 20% of children by the age of 7 and 2 – 10% of adults.¹ Consequently, the physician is very often not only dealing with the patient but the parents as well! Optimistically, however, the majority of childhood eczema improves or resolves by adulthood with reported clearance rates of 65% by age 7 and 74% by age 16.² There remains, however, an estimated 11,000 adults with significant atopic eczema in the UK.³ It has been recently recognised that a small but significant percentage of between 1-3% of new diagnoses of primary atopic eczema are elderly. Eczema is also responsible for 10 – 20% of referrals to dermatology out-patient departments and comprises 30% of all dermatological consultations. There is evidence to support an increasing prevalence in primary care in the last 30 years with parallel increases in asthma and allergic rhinitis. A family history of atopy is obtainable in 70% of cases.

TWO TYPICAL ATOPIC ECZEMA PATIENTS SHOWING: (A) DRYNESS AND INFLAMMATION AND (B) WITH INFECTION



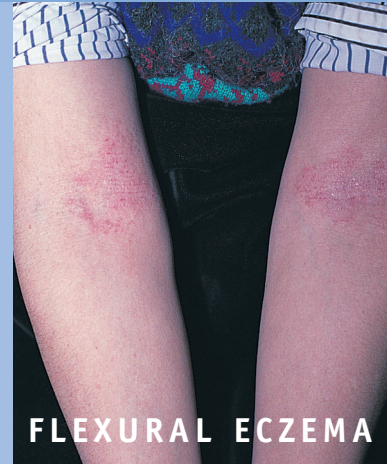
The clinical diagnosis of atopic eczema is not usually difficult to make. For the purists the absolute diagnostic criteria are as follows:

DIAGNOSTIC CRITERIA *

Must have an itchy skin condition plus 3 or more of the following:

- visible flexural eczema
- personal history of flexural dermatitis
- personal history of dry skin in the last 12 months
- personal history of asthma or allergic rhinitis (or history of atopic disease in a first degree relative)
- onset of signs and symptoms under the age of 2 years

* Ref. NICE Guidelines "Atopic Eczema in Children" Dec 2007

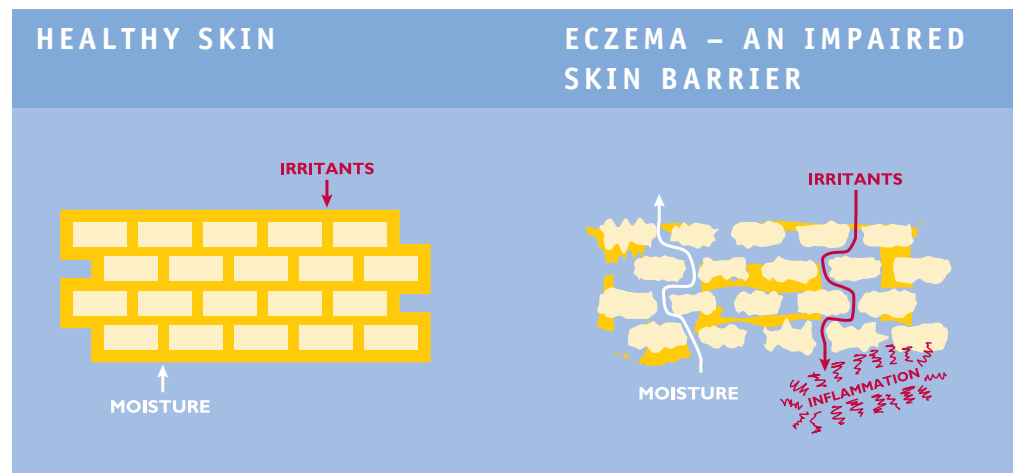


Anecdotally, one tip to avoid misdiagnosis is "If it ain't itchy, it ain't eczema." Be aware also that in dark skinned ethnic groups, eczema can most commonly affect extensor surfaces or present in a discoid or follicular pattern. Post inflammatory pigmentary disturbance is also a much greater issue.

POST INFLAMMATORY HYPOPIGMENTATION



The aetiology of eczema, however, remains unclear but is generally considered to be a complex interplay between immunological, genetic and environmental factors. However a key factor appears to be a deranged epidermal barrier function due to malfunction of the protein, filaggrin. This protein acts in analogous building terms to the mortar that holds the bricks together; if it is deficient, the result is the equivalent of a dry stone wall with cracks and imperfections allowing the ingress of exogenous agents such as allergens and micro-organisms triggering a pathological immune cascade. This “building bricks” analogy can often be helpful in explanation of the vital role of emollients as supplementary “mortar” gluing the epidermal “bricks” together!



FIRST CONSULTATION

When the patient/family first present I have found, despite many years in practice, it is virtually impossible to cover management principles in less than half an hour, and yet we continue to work in systems both in primary and secondary care that expect us to do so in half the time or less! Nurse support has enormous potential in this area although unfortunately Health Visitors appear to be becoming increasingly peripheral in General Practice.

The key issues at the first consultation are:

1. ESTABLISH there is no ‘cure’ for atopic dermatitis but the outlook should remain upbeat and optimistic. Treatment is readily available and if properly administered can hugely ameliorate the quality of life of patient and parent alike. There are a few broadly predictive factors. The previous family history may give some insight into the suspected severity. Early onset (under 3 months of age) and the presence of ‘reverse pattern’ eczema where the involvement of extensor rather than flexor surfaces predominate, are both said to be associated with a poorer prognosis. The principle of management should be “acceptable” quality of life until remission.

2. ASSESS the present clinical picture. Severity can be very variable between individuals and also within the same individual. How widespread is the eczema? Is there lichenification (thickening of the skin)? Are there exacerbating/complicating exogenous factors? Is there overt bacterial or viral infection? Is there evidence of infestation,

especially pediculosis and scabies which can both lead to secondary eczemas?
Beware also the presentation of ‘unilateral’ eczema, as this may be fungally mediated. Diagnosis can be further complicated by the misapplication of topical steroids altering the morphology, so called ‘tinea incognito’. Assessment of the social and psychological implications must also be made.

A holistic assessment chart is published within the NICE guidelines “Atopic eczema in children” Dec 2007 within the section on Key Priorities for Implementation.

3. CLARIFY previous and present treatments, both conventional and complementary, making very specific enquiries into quantities of topical preparations used. A lack of understanding of emollients by doctors and patients alike often results in their underusage.

4. ACKNOWLEDGE empathetically how difficult it is to have a young child with significant eczema – the distress, the sleep deprivation, the frustration, and the despair! Children with significant atopic dermatitis put more of a strain on family dynamics than most health professionals first realise.

5. ACCEPT the genuine fears and concerns about standard treatments with steroids/ antibiotics, counteract scare mongering and misinformation, and firmly re-establish that these are the fundamental cornerstones of both safe and effective treatment at the present time if used under the proper guidance. Indeed, it is now almost more common to observe under-use than abuse of steroid preparations.

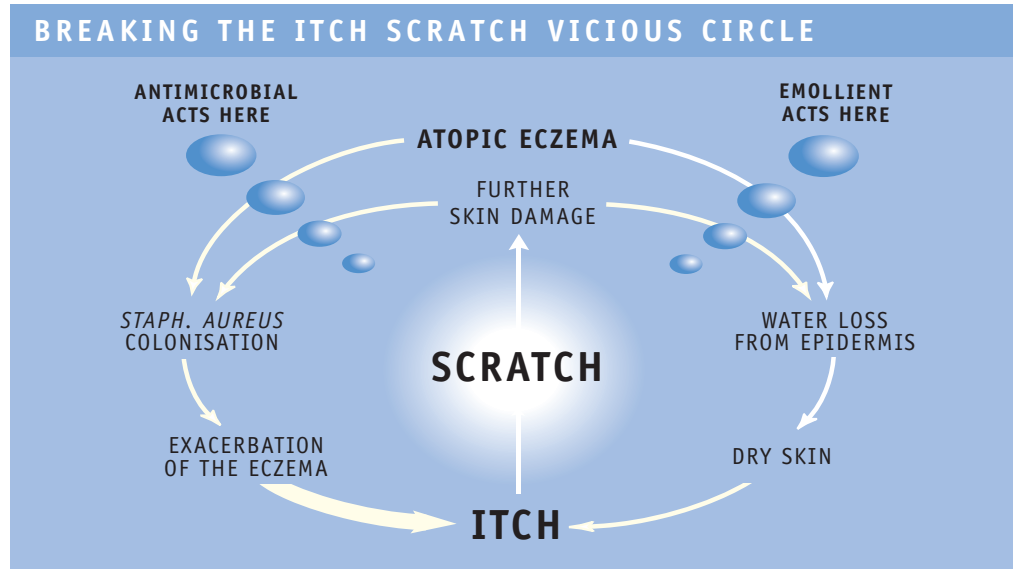
6. ENCOURAGE and facilitate the adoption of self help as a principle of management for any chronic condition by education of the patient/parent. Reinforce this with written material, software programmes, local and national self help groups notably the most excellent National Eczema Society, and facilitating access to nurse-led clinics, if such are readily but all too rarely available. Information should be factual, not controversial and, ideally, available in different languages if applicable. As with, for example, asthma and diabetes mellitus, the patient/parent must have a good understanding of maintaining satisfactory control, but also how to recognise deterioration and apply strategies for intervention and know when to seek more expert help. Introduce the “toolbox” concept in which the patient/parent has a number of treatment options open to them at home and a clear understanding of what to use in what circumstances.

7. ARRANGE appropriate investigation and follow up.

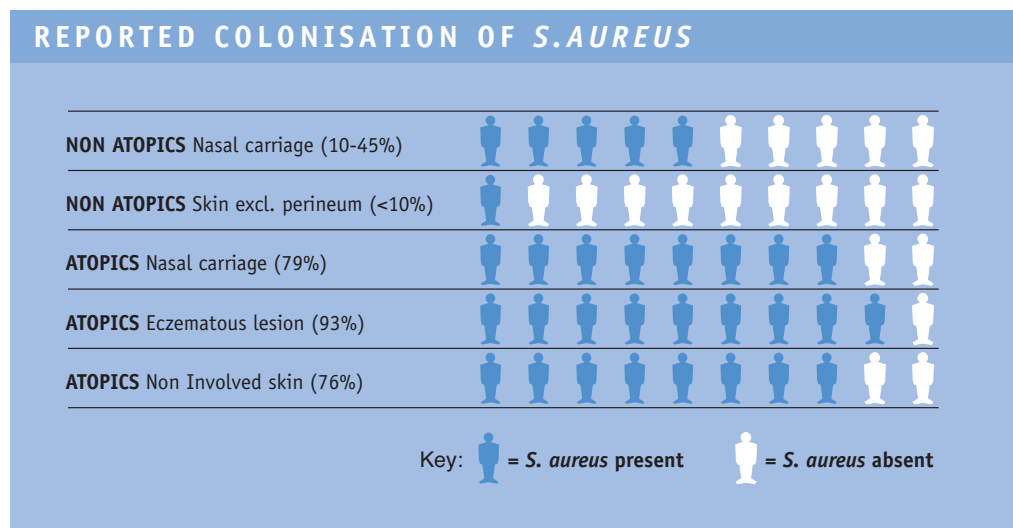
TOP TIP 1: I often find the analogy of dealing with eczema is like dealing with fire when explaining management principles to patients/parents. There is a preventative strategy with Total Emollient Therapy and then the need for intervention when the fire (=itch) is alive. Treatments, mainly topical steroid preparations, are the equivalent of “fire extinguishers”. These can be more or less powerful but you may not necessarily need to use “fire extinguishers” when the “fire” is out! This analogy can provide some protection against the routine and potential overuse of steroids.

MANAGEMENT PRINCIPLES

The skin should form an effective barrier to the environment. This is compromised in atopic eczema due to, what I explain rather simplistically, as a reduction of natural moisturisers, leading to dryness, microfissuring and the ingress of contaminants and allergens, ‘dirt and germs’, resulting in inflammation and infection and the inevitable consequence of the itch/scratch/damage cycle.

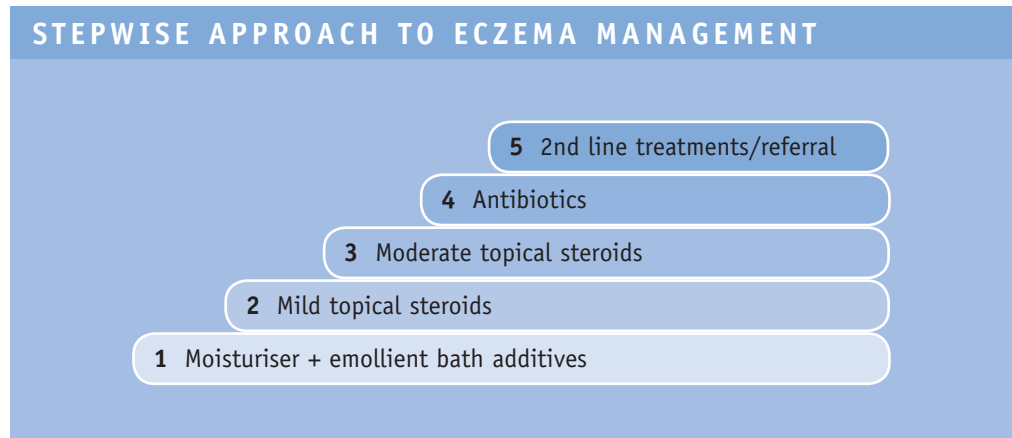


The patient/parent must be able to confidently recognise the stage of the eczema. Is it dryness alone, dryness plus inflammation, dryness, inflammation and infection? (See slides on page 1). It has been recognised as long ago as 1955 that atopic eczema improves with antibiotics even in the absence of overt infection.⁴ Skin bacteria, most particularly *Staphylococcus aureus*, are present in much greater numbers on atopic skin. *Staphylococcus* is **not** a customary member of the cutaneous microflora with the exception of the perineum. The bacteria provoke an immune mediated response due to the production of so called ‘super antigens’. These are proteins that act as potent immunostimulators releasing a cascade of inflammatory mediators into the skin.⁵



A stepped approach to management is published in the NICE guidelines “Atopic eczema in children” Dec 2007.

I try to illustrate the principles of eczema management to patients with the use of a simple stepwise diagram as follows:



The patient should understand that they are **always** on Step 1, practising regular moisturisation and soap avoidance/soap substitutes (=Total Emollient Therapy). If there is deterioration, they should ascend the steps fairly rapidly every few days until the eczema comes under control again. Often there is resistance to moving beyond Step 2 but a short period using a more potent steroid plus or minus an antibiotic will often rapidly produce such an improvement that the patient can return to moisturisers alone and paradoxically reduce their overall steroid use in the longer term. There are a number of “at risk” sites for topical steroid use, peri-orbital and flexural areas in adults and additionally facial skin in babies and infants. For children from 2 years upwards, the relatively new class of topical calcineurin inhibitors, tacrolimus and pimecrolimus can be introduced.

Their origins were derived from Japanese mountain soil fungus! There is increasing longitudinal data underlining the safety of these preparations although warning must be given regarding the initial but usually transient burning sensation that these products can produce. Tacrolimus is available in an ointment in 2 strengths 0.03 and 0.1%; it is a more potent preparation than pimecrolimus which is available in a cream formulation. Twice weekly maintenance regimes are becoming fashionable (and licensed).

Depending on the motivation and insight of the patient/parent, it is perfectly reasonable for the doctor to provide the medications for self management up to and sometimes including Step 4. I would equate the use of the most potent steroids with 2nd line treatment. This ‘broad brush’ management plan is not always applicable, but it is a useful care model for the majority in my experience. We must give clear and precise guidelines about the use of topical treatments. Terms such as liberally, frequently and, most particularly, **sparingly**, should have no place in our discussions.

EMOLLIENT WEEKLY USAGE GUIDELINES*

CREAM/OINTMENT

LOTION

15-30g

100ml

FACE

25-50g

200ml

BOTH HANDS

50-100g

200ml

SCALP

100-200g

200ml

BOTH ARMS OR BOTH LEGS

400g

500ml

TRUNK

15-25g

100ml

GROINS/GENITALIA

* Ref. BNF online September 2017

Based on adult twice daily application for 1 week. NB These recommendations do not apply to topical corticosteroids.



Too often, these terms reflect our ignorance; too often topical treatments are provided in ‘homeopathic’ quantities. For whole body application, adults require approximately 500 ml and children 250 ml weekly. For adults ‘whole body’ cover requires 30g of ointment or 20g of cream for a single application. For topical steroid application, the concept of the ‘finger tip’ unit is now widely understood and utilised.

There are various protocols which have their disciples such as the “weekender” regime where a more potent topical steroid might be used for 2 days in each week in order to try and gain a balance between efficacy and safety.

We do not expect our patients to troop back to the surgery every week or so for maintenance drugs for blood pressure or diabetes, and nor should they have to for their topical medications. Suboptimal prescribing leads to embarrassment and disillusionment, resulting in suboptimal clinical outcomes. For those patients who pay for their scripts, the benefit of prepaid prescriptions should be calculated.

INFECTION AND ATOPIC ECZEMA



The most common cause for sudden deterioration in atopic eczema is infection, although this may often not be overt. An extended course of anti-staphylococcal antibiotics is always a useful strategy to consider. Most community acquired *Staphylococcus aureus* remains consistently sensitive to flucloxacillin. Skin and nose swabs are not routinely necessary to check on sensitivities. Flucloxacillin, unfortunately, is an unpopular paediatric antibiotic due to its bitter taste. It is also much more expensive in its liquid form, cost effective alternatives are erythromycin and cefradine.

Topical antibiotics/antiseptics in combination with topical steroids e.g. Fucidin H and Fucibet, have a place in the treatment of localised infection, although topical fusidic acid is relatively expensive. In addition, there have been some recent reports of locally increasing staphylococcal resistance to fusidic acid. The extent and significance of this remains as yet unclear. Despite wider usage, resistance to topical mupirocin remains relatively low.

I illustrate to the patient the rationale of using antibiotics/antimicrobials with the anecdote of 'weeds in the garden'; like bacteria on atopic skin, weeds can thrive under certain environmental conditions. Although you can never rid your garden of weeds, a good dose of weed killer can work wonders in the short to medium term!

This would be a logical role for the use of antimicrobial emollients although as yet hard evidence is not available as how best to use these products.

DISCUSSION POINTS

There are a number of miscellaneous discussion points relating to the management of eczema.

1) USE OF ANTIHISTAMINES – histamine is not the dominant mediator of itch in eczema⁶ but sedative antihistamines remain useful for short term use in children whose sleep patterns are significantly disturbed. Habituation however, develops quickly and these drugs should not be used for more than a few days at a time.

2) DIETARY MANIPULATION – atopic individuals are at higher risk of food allergy/intolerance. This is an issue that is invariably raised but is of exaggerated value in the absence of a firm history of food reactions, especially immediate hypersensitivity reactions. Dietary manipulation is most helpful in the under 1 age group and it is much less likely to help in children of 4 years or over. There is no reliable method, however, of selecting those children statistically most likely to benefit. The foods most commonly indicated in food allergy are dairy produce, beef, eggs, chicken, additives, fish, wheat and nuts. Empirical exclusion diets have demonstrated 30 – 40% improvement over 3 – 4 weeks with strict adherence.⁷ Dietary manipulation, however, should be carried out under expert supervision and the concept of challenge/rechallenge understood by the patient/parent. In young children, input from a dietician is required. We must always remain true to the principle that treatment should never be worse than the condition! There is an increasingly accepted theory, the “dual allergen hypothesis” that atopic eczema may not be caused by food allergy but that such allergies develop as a consequence of sensitisation through the skin itself and indeed the development of tolerance subsequently is through oral exposure mediated through the gut.



3) CHINESE HERBAL MEDICINE – this is well documented to have had a beneficial effect in some patients with atopic eczema. The present standpoint, however, is risk outweighs benefit. Chinese herbal medicine practitioners are unregulated, as are the preparations themselves. Steroid derivatives have not infrequently been isolated. Hepatic toxicity has been reported and implicated in a few fatalities. The medication is universally unpalatable if ingested. Avoidance must presently be advocated!

4) OTHER COMPLEMENTARY THERAPIES – without appearing cynical, homeopathy, acupuncture, herbalism, reflexology etc, have **in my personal experience** been very disappointing. Most improvement seen is temporary and is probably explained by the natural refractory pattern of the condition. There is interesting work going on with behavioural modification but tangible results are not yet to hand. This concept of teaching people not to scratch when itching is a difficult one for me, especially in the context of 2 quotations that have been attributed to King James VI and Oscar Wilde, respectively.

‘The itch is such an uncommon pleasure, it should be denied to the common man!’

‘The only way to get rid of temptation is to give in to it!’

5) WET WRAP BANDAGING – this is a technique going through a renaissance – a sort of ‘imposed’ behaviour modification! It can be very useful in selected cases especially if limb eczema predominates, although whole body wrapping can also be carried out. It requires a high level of commitment from the parents and the expertise is all too often not readily available.

More recently a range of “dry wrap” garments have also become available. Recently the results of the “CLOTHES Trial” were published and these unequivocally showed that there was no benefit from the use of silk garments in the management of atopic eczema despite a current cost of over £2 million to the NHS.⁸

6) FEEDING – there is no current evidence to support any advantage of breast feeding over bottle feeding in terms of severity of atopic eczema and that also the timing of weaning makes no difference either. As regards milk substitutes, there has been benefit demonstrated for high risk infants with the introduction of milk hydrolysate formulae⁹ but no such benefit demonstrated for soya milk. However, research has also demonstrated that if hydrolysates are substituted for milk feeds, that the extent of hydrolysis of the preparations makes no difference, although fully hydrolysed products are significantly more expensive.

7) ALLERGEN AVOIDANCE – aeroallergens, such as house dust mite and animal dander may aggravate eczema. Reports vary as to the efficacy of counter measures. Studies differ on the efficacy of stringent anti-dust controls. In one such study, these showed a very limited response while another reported significant clinical benefit! This may be a worthwhile option if a parent is highly motivated and even obsessive, and the child is willing to have their teddy bear committed to the freezer to inactivate house dust mite!

The role of food allergy is greatly overestimated. Careful history taking should elucidate any immediate Type 1 food reactions; otherwise it should only be considered further in children with severe unresponsive eczema especially if there are co-existent gut motility problems or failure to thrive. Advise against allergy testing as a routine especially being wary of commercial services.

8) TACHYPHYLAXIS – patients often observe “my skin seems to have got used to my steroid cream.” This can be partially explained by the concept of tachyphylaxis; the clinical observation that a topical steroid is more active at the beginning of treatment than later on. To achieve the same effect, the patient may need to apply the steroid after ever shortening intervals. Alternatively, switching to another steroid preparation of **similar potency** for a short period can be a useful manoeuvre, resisting the temptation to use even more potent topical preparations. This, however, is not evidence based!

INVESTIGATION IN ATOPIC ECZEMA

SKIN/ALLERGY TESTING – Such testing is available in a number of formats – patch, prick and RAST/other blood assays. They are frequently requested inappropriately by patients and doctors alike due to the lack of understanding of the classification of the various immune reactions. It is beyond the scope of this article to discuss immune mediated reactions in detail. Suffice to say that the more commonly requested patch testing is most applicable to patients with a history suggestive of allergic or occupational dermatitis. Such tests can, however, be selectively justified for atopics if there is a sudden unexplained deterioration, where the history indicates the possibility of aggravating exogenous factors. Prick testing clarifies immediate hypersensitivity or urticarial reactions (Type 1). There are many false positives and negatives. Similarly, IgE/RAST testing measures specific IgE antibodies in the peripheral blood. False positives are common and 75% of positive RAST tests are not always clinically significant. There is no benefit from such tests being used as routine.

OTHER INVESTIGATION – Growth chart monitoring can be useful in children severely affected by eczema, as growth retardation is a risk. This is multifactorial and not primarily due to the risk of steroid absorption.

USEFUL HINTS AND TIPS



Some management principles have already been discussed. Here are some additional hints which I hope will prove helpful:

- a) The best emollient is the cheapest that the patient finds effective and is willing to use. Cosmetic acceptability is a major determinant of use. Emollients ‘waterproof’ the skin and may also have some anti-inflammatory and thus steroid sparing effects.¹⁰ There are also now a choice of emollients with antimicrobial agents specifically directed at reducing staphylococcal colonisation. These have proven a very useful adjunct to our therapeutic armoury and can be used both as bath additives and soap substitutes or applied directly to the skin. Various emollients **claim** additional advantages. Those containing urea may be useful on very thickened and scaly skin although irritant to broken skin, while lauromacrogols may have additional anti-pruritic properties. There is no good co-operative clinical evidence, however, to support these claims. In a dermatological utopia, emollients should be applied a minimum of 3 – 4 times a day and these should be applied **after** the application of topical steroids.
- b) Practise strict soap avoidance – soaps dry on the skin to leave irritant alkaline residues and strip the skin of its natural oils, which both exacerbate eczema. Exceptions to the former are neutral pH soaps, such as Neutrogena and Dove.
- c) Topical steroids should not be prescribed without emollients! All steroid cream preparations are potentially dehydrating.
- d) Be familiar with 1 or 2 topical steroid preparations in each potency class.

TOP TIP 2: The recent “BEEP” study demonstrated an advantage to using regular emollients immediately from birth in babies with a high risk profile for developing significant atopic eczema i.e. before the disease is manifest. This would appear to be a simple, safe and cheap intervention to consider where there is a strong family history.¹¹ However, care needs to be exercised with the use of highly occlusive ointment based preparations in very young infants which can lead to interference with the sweat gland apparatus and potential overheating.

PERI-ORAL DERMATITIS



e) Be familiar with the common local cutaneous side effects of excess steroid usage i.e. striae, purpura/telangiectasia, atrophy, rosacea and peri-oral dermatitis. Long term application of even mild topical steroids to the eyelids have also resulted in the development of glaucoma. The newer, once daily, steroid preparations (mometasone and fluticasone) claim lower risk of cutaneous side effects. Compliance is easier although such products are relatively more expensive. Topical steroid absorption is greatest on the face due to the relative thinness of the skin and the flexures due to opposing surfaces and increased local humidity.

f) Ointments have advantages over creams – they are inherently more moisturising because of the ointment vehicle and don't contain preservatives resulting in a lower incidence of contact sensitivity. Lotions are preferable for hair bearing areas. Alcohol based preparations can sting significantly if applied to areas of cracked or broken skin.

g) Confusion often arises relating to the optimal frequency of bathing. The time honoured advice was to reduce. Regular daily bathing induces more benefits than risks by removing dirt, bacteria and environmental allergens. The regular use of emollient bath additives is also advocated. A range of combined emollient/antiseptic bath additives have an added anti-staphylococcal effect e.g. Dermol 600 Bath Emollient, Oilatum Plus. Studies have shown that such treatments can reduce *Staph. aureus* numbers 6,000 fold!¹²

h) Eczema is **not** a contraindication to immunisation.

OTHER ECZEMAS

Classification of endogenous and exogenous eczemas can be simplistic. There are, however, a number of significant eczematous processes which we will now consider separately.

SEBORRHOEIC ECZEMA – this is a common condition often presenting in children and young adults. It is important to differentiate it from atopic eczema as causation and treatment are different. The pathogenesis is not yet fully understood but an overgrowth of normally commensal *Pityrosporum* yeasts is considered a major aetiological factor. It often presents in childhood as a widespread facial and truncal eczema between the ages of 1 and 6 months in contrast to atopic eczema which often develops a little later. Scalp involvement frequently co-exists – ‘cradle cap’ in babes, seborrhoeic capitus (dandruff) in adults. The rash of seborrhoeic eczema is usually more clearly delineated and less symptomatic than atopic eczema. The child is usually not distressed and flexural involvement is also common. The differential diagnosis includes candidal infection and infantile psoriasis. Treatment revolves around the use of mild steroid anti-yeast combinations such as Daktacort or anti-fungal treatments such as topical ketoconazole. Seborrhoeic eczema often affects young adults with a scaly, mildly itchy facial eruption involving the scalp, eyebrows, nasolabial folds and post auricular creases with co-existent flexural and presternal involvement. Blepharitis is a common complication. It can be difficult to differentiate from other facial dermatoses e.g. rosacea. The presence of pustulation, telangiectasia and the absence of scaling in an older age group would point towards the latter diagnosis. Very acute onset widespread seborrhoeic dermatitis may indicate immunosuppression, particularly HIV.



SEBORRHOEIC ECZEMA



DISCOID ECZEMA – also known as nummular eczema after the Greek ‘nummus’ meaning coin. It most commonly presents in middle to late middle years, predominantly in men. There is often a past history of atopy. As with many eczemas, pathogenesis remains unclear. Involvement of the limbs predominate. Single lesions need to be differentiated from superficial basal cell carcinoma, granuloma annulare, fungal infections and Bowen’s disease. Intense itch is often present and requires potent topical steroids to control it.



POMPHOLYX – a distinctive pattern of eczema selectively affecting the thicker skin of the palms of the hands (cheiro-pompholyx) and/or the soles of the feet (podo-pompholyx). There is often no background of atopy. This can occur at any age but most especially young adults; there is no sexual predominance. There are characteristically small vesicles affecting the lateral aspects of the fingers ‘like sago grains.’ Pustules may develop and secondary infection is often a feature. The condition is notoriously itchy and can be quite disabling. Bullae can occur. A history of intercurrent stress is often obtainable. Exogenous agents are not often implicated. Treatment embraces the use of rest, potassium permanganate soaks, emollients, potent/very potent topical steroids, and short courses of antibiotics or even systemic steroids. Further investigation is usually unhelpful and the differential diagnosis would include palmoplantar psoriasis and localised bullous conditions. There is also the phenomenon of the fungal ‘id’ reaction, where coexisting tinea infection can trigger pompholyx at a distant site. Pompholyx is often a recurrent condition.

VENOUS ECZEMA – this results from longstanding venous hypertension leading to microcirculatory damage and the typical features of lipodermatosclerosis and haemosiderin leakage and deposition in the skin. There may be frank ulceration. Venous eczema can be complicated by superimposed irritant eczema from exudative ulcers and also a very high propensity of this area to develop contact sensitivity to a wide range of dressings and topical medicaments. Treatment is to the underlying cause and it is important to remember the truism that ‘a venous ulcer is only a manifestation of the underlying disease process.’ Protection, support and the use of emollients and mild to moderate topical steroids form the major principles of management. Patch testing is often indicated.

VENOUS ECZEMA



ASTEATOTIC ECZEMA (eczema craquelé) presents as a ‘crazy paving’ pattern in the anterior distal lower limbs of predominantly elderly patients. Over washing and overheating are the major aetiological factors but if extensive or severe, consider intercurrent systemic problems such as hypothyroidism or malnutrition. Treatment is with frequent application of emollients plus or minus topical corticosteroids and avoidance of provoking factors.

ALLERGIC/IRRITANT DERMATITIS/ECZEMA – these conditions most commonly affect the hands and cannot be easily differentiated; indeed, they may often co-exist. There is an equal incidence in men and women. Hand eczema affects 2% of the population at some time. With irritant eczema, 85% of patients have a history of atopy. In contrast, atopy does not increase the risk of allergic dermatitis which is much the commoner of the two varieties. Irritant eczema is caused by direct physical or chemical injury from acids, alkalis, antiseptics or detergents. It can be acute, caused by a single contact or chronic from cumulative exposure. Common irritants are dust, detergents, cleaners, lubricants/industrial oils, preservatives and synthetic fibres. The finger webs

and the dorsum of the hands are most commonly involved. Vesicular reactions are less common. In contrast, allergic dermatitis is usually a Type IV, cell mediated, delayed hypersensitivity reaction. This is relatively rare but is recognised to be increasing in the paediatric population. Vesicular reactions are much more common. Common allergens are rubber, nickel, cement dust, plants, topical antibiotics and cosmetics. Between 10 – 20% of all women have a contact allergy to nickel on testing. This is also present as a contaminant in cheaper gold preparations. It is frequently believed that there is a high contact sensitivity to lanolin. Lanolin is a complex substance derived from sheep wool; it has had unjustifiably bad press. Medical grades of lanolin have a very low sensitising potential.



The family practitioner should be able to recognise common patterns of allergic contact dermatitis; these can be very specific e.g. periumbilical dermatitis due to nickel jean studs. Contact allergy is usually a life long sensitivity once established, although it may take several years to develop. The most likely sites involved are the hands, face, legs and feet; secondary spread is common.

Patients with chronic hand eczema can be very challenging. The condition can result in long term disability. Historically there has been a poor response to available treatments even with more potent “second line” treatments such as PUVA and oral immunosuppression. A retinoid, alitretinoin, has been licensed and received NICE approval specifically for this group of patients allowing more hope for the future.

There is usually a timescale between the contact and the reaction developing of 1 – 3 days. History taking is of paramount importance in elucidating the influence of likely irritants/allergens. Clinical patterns are often indistinct. Patch testing is important to help reach a precise diagnosis. Irritant eczema is often a diagnosis by exclusion. There are no reliable figures available for the incidence of occupational dermatitis due to problems of under-reporting and the acceptance by workers that this is a bona fide occupational hazard. Pre-emptive careers advice to children with significant atopy is useful to avoid exposure to occupations with a high risk of irritant eczema, especially engineering, nursing, building and, most particularly, hairdressing. Barrier creams are ineffectual. Effective measures revolve around education, protection, avoidance, and interval rehydration and remoisturisation. The prognosis for occupational dermatitis, once established, is poor even after a change of occupation. It is unclear why this should be so. In the UK alone, it is estimated that 4 million workdays are lost to dermatological problems, especially occupational dermatitis.



PATCH TEST – BRITISH SOCIETY FOR CUTANEOUS ALLERGY RECOMMENDED BASELINE SERIES¹³

TEST SUBSTANCE	WHERE IT IS COMMONLY FOUND
Potassium dichromate	Cement, leather, wood preservatives, mascara, eye shadow
Neomycin sulfate	Antibiotic creams and powders
Thiuram mix	Rubber, fungicides, soaps, animal repellents
p-Phenylenediamine	Hair dyes
Cobalt chloride	Jewellery, dental plates, prostheses, inks and paints
Caine mix III	Local anaesthetic, antipruritic creams, haemorrhoid creams
Formaldehyde	Preservatives, detergents, shampoos, textiles
Colophony	Adhesive plasters, mascara, eye shadow, varnishes, rosin, solder fluxes
Quinoline mix	Clioquinol and chlorquinaldol in local anti-infective agents
<i>Myroxylon pereirae</i> (Balsam of Peru)	Flavourings and fragrances
N-Isopropyl-N-phenyl-4-phenylenediamine	Black rubber
Lanolin alcohol	Lanolin creams, lotions and cosmetics
Mercapto mix	Rubber
Epoxy resin	Adhesive, surface coating, electron microscopy, paints and inks
Parabens mix	Preservative in foods, cosmetics and pharmaceutical products
4- <i>tert</i> -Butylphenol formaldehyde resin	PTBP Resin, adhesive for shoes and watch straps, inks, papers and film developers
Fragrance mix I	Fragrances
Quaternium 15 (Dowicil 200)	Preservative in cosmetics and toiletries
Nickel sulfate	Scissors, door handles, keys, coins, zips, buckles, 'gold' lingerie fasteners, jean studs, watch straps & buckles etc.

PATCH TEST – BRITISH SOCIETY FOR CUTANEOUS ALLERGY RECOMMENDED BASELINE SERIES CONT.¹³

TEST SUBSTANCE	WHERE IT IS COMMONLY FOUND
Cl+Me-isothiazolinone	In Kathon CG. Preservative in cosmetics, shampoos, detergents and cooling fluids
Mercaptobenzothiazole	Rubber in shoes, gloves, tyres, condoms, swimwear, oils and antifreeze
Amerchol L101	Lanolin alcohols in cosmetics, topical products
Sesquiterpene lactone mix	Compositae e.g. chrysanthemums
p-Chloro-m-cresol	Fungicide in creams, topical antiseptics etc.
2-Bromo-2-nitropropane-1,3-diol (Bronopol)	Preservative in cooling fluids, cosmetic creams, shampoos, hair dressings etc.
Cetearyl alcohol	Emollient in cosmetics and pharmaceutical preparations
Sodium fusidate	Antibiotic
Tixocortol-21-pivalate	Marker of corticosteroid allergy
Budesonide	Marker of corticosteroid allergy
Imidazolidinyl urea (Germal 115)	Preservative in creams, lotions etc. Formaldehyde releaser
Diazolidinyl urea (Germal 11)	As above
Methyldibromoglutaronitrile	In Euxyl K 400 and Tektamer 38. Preservative in cosmetics, detergents etc.
Ethylenediamine dihydrochloride	Stabiliser, epoxy curing agent
4-Chloro-3,5-xyleneol (PCMX)	Preservative in creams, topical antiseptics, pharmaceutical products, soaps etc.
Carba mix	Rubber products
Fragrance mix II	Fragrances
Disperse Blue mix 106/124	Dyes added to textiles and fabrics
Hydroxyisohexyl cyclohexene carboaldehyde (Lyral)	Fragrance in perfumes, toiletries, cosmetics and household products
Compositae mix (Chemotechnique)	Compositae e.g. chrysanthemums
Methylisothiazolinone	Preservative in cosmetics, toiletries etc.
Sodium metabisulphite	An antioxidant found in topical preparations, cosmetics and toiletries

TOP TIP 3: Patch testing has high pick up rates with the clinical presentations of both pruritus anae and pruritus vulvae and should be considered when such patients are not responding to standard managements.

CRITERIA FOR REFERRAL

When do we need to refer to a specialist? There are a multitude of reasons for secondary referral, not least patient pressure and unrealistic expectations. The main reasons, however, I would consider are as follows:

- 1) Diagnostic doubt.
- 2) Refractory eczema; especially resistant hand eczema, despite intensive emollient regimes, topical steroids and antibiotics.
- 3) When patch testing is truly indicated.
- 4) Consideration of 2nd line treatments e.g. systemic drugs, very potent topical steroids or phototherapy.
- 5) Consideration of dietary manipulation in infants with severe refractory eczema.

TEACHING POINTS

- 1) Beware unilateral eczema. Eczema is characteristically a symmetrical disease – you may be dealing with fungal infection. If in doubt, **scrape!**
- 2) Always prescribe enough topical treatment for your patient.
- 3) The most common cause of an exacerbation of atopic eczema is infection.
- 4) 2 – 5% of patients are hypersensitive to some topical steroid preparations.

**USEFUL CONTACT**

National Eczema Society
11 Murray Street, London, NW1 9RE
<http://www.eczema.org>

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Contra-indications, warnings, side effects etc: Do not use if sensitive to any of the ingredients. Keep away from the eyes, inside the nostrils and mouth. Temporary tingling, itching or stinging may

occur with emollients when applied to damaged skin. Such symptoms usually subside after a few days of treatment, however, if they are troublesome or persist, stop using and seek medical advice. Rarely skin irritation (mild rashes) or allergic skin reactions can occur on extremely sensitive skin, these tend to occur during or soon after the first few uses and if this occurs stop treatment. As safety trials have not been conducted during pregnancy and breast-feeding, seek medical advice before using this product.

Care should be taken as emollients which soak into clothing, pyjamas, bedlinen etc. can increase the flammability of these items. Patients should avoid these materials coming into contact with naked flames or lit cigarettes etc. As a precaution, dressings and clothing, etc., should be changed frequently and laundered thoroughly.

Ingredients: Carbomer, glycerol, isopropyl myristate, liquid paraffin, nicotinamide, phenoxyethanol, sorbitan laurate, trolamine, purified water.

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