Miscellaneous crappy legs

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Dermatitis Neglecta

- First described by Dr Louise Reiche [now in Palmerston North] et al in 1995.

- They noted that the accumulation of sebum, keratin, dirt and debris could be removed by rubbing with an alcohol swab, or soap and water. They considered the condition to be the result of ‘wilful or subconscious self neglect’

- It may be velvety, but typically has large scales like cornflakes, or like fish-scales.
A typical case....

• 84 yr old F, ref. by Wound Care Clinical Nurse Specialist.

• Femoral popliteal bypass Feb. 2009

• Blisters and infection in left leg Aug. 2009 -> antibiotics

• Vascular surgeons requested 2 x weekly light compression bandaging

• Rx: white soft paraffin/liquid paraffin ointment
• Left leg covered in large brownish scales like fish scales, or cornflakes.

• Eczematous changes on the upper third of the leg also

• Lipodermatosclerosis on the lower third of the leg (due to vascular disease)
• Treatment: we removed the scales, lifting them up with a fingernail and peeling them off.

• Then locoid cream to be applied when dressings changed every 2\textsuperscript{nd} day (on account of the underlying eczematous changes)
3 weeks later:
All the reports of dermatitis neglecta have echoed Poskitt’s belief that the disorder arises as a result of failure to wash the affected area, either because it is

– painful
– or the patient is fearful of touching it following injury or disease
– or for whatever reason there is inadequate hygiene
Dermatitis neglecta has been reported in the following circumstances:

• Following a face peel
• At the site of sunburn
• Overlying a pacemaker
• Overlying mastectomy or other surgical scar
• Following multiple fractures and nerve palsy
• Overlying eyelid eczema
• In hyperesthetic skin due to neuralgia
If the problem is due to hygiene failure then how come...

- Poskitt’s case (which followed sunburn) spared the area under the watch (this area was as unwashed as the rest of the forearm – it just hadn’t been sunburned)

- Gomez-Fernando’s case only affected ankles & feet – this patient was said to neglect their hygiene – did they wash down to their knees and then get tired??

- It doesn’t routinely affect those Inuit or desert dwellers who wash infrequently?

- It doesn’t affect lots of us, who don’t briskly and routinely rub our faces with flannels?
• Normally epidermal cells reproduce, mature, lose their nuclei (becoming corneocytes) and these ‘dead’ corneocytes are shed. This process goes on all the time.

• Dead Corneocytes don’t need to be scrubbed to make them fall off.

• We suspect that injuries/or disease processes in the right circumstances cause a localised defect in Corneocyte shedding—hence a build up of those dead cells

• Washing or alcohol swabs or manual debridement removes them—but the reason they are there in the 1st place may NOT be lack of washing.
- Dermatitis neglecta is a problem (usually localised) of corneocyte retention, perhaps triggered by injury or disease.

- Mechanically removing the scales solves the problem until either the problem with corneocyte desquamation solves itself or the disease that provoked it is treated. (eg eczema)

- We think that dermatitis neglecta occurs relatively commonly following actinic damage, or peripheral vascular disease.
This was able to be removed with an alcohol swab
Removed by a vigorous rub with saline-soaked gauze
Before

After. (Manually removed the larger flakes, and rubbed with a saline-soaked gauze for the rest)
• Fungal nail problem longstanding. The adjacent skin build up started after a hip operation – and she couldn’t reach her feet any more.

• It was all removed with a blade and scissors
There is no point putting cream on it  It just sits on the surface
Actinic damage
Bowens disease (SCC in situ)  Porokeratosis
Basal cell carcinomas
SCC

Seborrheic keratosis
Lots of things can present as a boring red splodge

BCC’s

Amelanotic melanoma
Often we use dermoscopy to tell who is which

**BCC**

**Amelanotic melanoma**
Erosive pustular dermatosis

- Most common on the scalp, but also occurs on the leg
- Inflammatory condition of unknown cause
- Can be precipitated or aggravated by trauma eg cryotherapy, efudix, surgery, compression bandaging
Erosive pustular dermatosis
Erosive pustular dermatosis
Erosive pustular dermatosis

Copied from BJD 2002 147 765-769. Brouard et al

- Patients are usually elderly
- Usually badly sun damaged, atrophic skin
- Associated with autoimmune disorders
- Female predominance
Differential diagnoses:

- Bacterial or fungal infection
- pyoderma gangrenosum (superficial form)
- pustular psoriasis
- Pemphigoid
- Pemphigus
- SCC
Investigations:

• Swabs

• Biopsy with DIF (although, if you have ruled out infection, and SCC, the other differentials such as pemphigoid and superficial pyoderma gangrenosum, may all respond to potent topical steroids)
• Usually EPD affects the leg, or the scalp; rarely it may affect other sites; there are two reports of it affecting both the leg and the scalp, and one of a generalised eruption.

• The authors showed that IL8 (a strong neutrophic chemoattractant) was elevated in serum and over expressed in epidermal keratinocytes.

• Compared with pyoderma gangrenosum where IL8 is produced in large amounts by lesional dermal fibroblasts.
• Treatment is usually a potent topical steroid eg dermol.

• Sometimes systemic steroids, dapsone, isotretinoin, mesalazine, zinc, (all of which reduce IL8 expression)
Erosive pustular dermatosis of the leg before dermol cream

A few weeks later
Stasis dermatitis

- Venous hypertension in superficial veins is transmitted to dermal microcirculation, resulting in increased permeability of dermal capillaries

- Macromolecules such as fibrinogen are able to leak into the skin, polymerise into fibrin to form a fibrin cuff around the capillaries

- Leukocytes get trapped and release mediators that attract more leukocytes, add to the fibrosis, and create inflammation
• Unabated venous hypertension results in pigmentation, eczema, dermal and subcutaneous fibrosis (lipodermatosclerosis) and ulceration

• Anything that worsens peripheral edema (CHF, obesity, amlodipine...) will aggravate the situation

• Cellulitis can compound the picture
Other kinds of dermatitis (eczema)

Severe discoid eczema
Suspect contact allergy with sudden onset dermatitis, that is localised, severe, or unresponsive to treatment.

Contact allergic dermatitis
Asteatotic eczema

Papulovesicular eczema
Lichenified eczema

Lichen simplex chronicus
Other kinds of leg mischief

Necrobiosis lipoidica

Erythema nodosum
Other kinds of leg mischief

Vasculitis

Sneddon Wilkinson Disease
Other kinds of leg mischief

Mild pyoderma gangrenosum

• The differential here is erosive pustular dermatosis, autoimmune blistering diseases, infections, even pustular psoriasis.
Pyoderma gangrenosum

Lab Invest 2000 80 595-604

- IL8 production increased by lesional fibroblasts.

- Oka et al showed that if you construct an adenovirus which has DNA which encodes for a major increase in IL8 expression in infected fibroblasts, and then inject it into mice, you can fairly quickly produce lesions which are clinically and histologically pyoderma gangrenosum.
Pyoderma gangrenosum

• Painful ulcers
• May be shallow or deep
• Often with blue-ish undermined edge
• Associated with inflammatory bowel disease, cancer
• May be precipitated by trauma
Other kinds of leg mischief

Bullous amyloidosis

Sweets syndrome
Other kinds of leg mischief

• Pemphigoid

• A very common condition in the elderly, but it can occur at any age

• While blisters are usually generalised, the may be localised. Pre tibial pemphigoid is a recognised variant.

• pemphigoid may be triggered by trauma, scabies, phototherapy
• Generally pemphigoid produces large tense bullae
Sometimes one just gets erosions

Which may resemble other conditions like pemphigus foliaceous
• In the elderly, if the presentation is typical, one can usually make a clinical diagnosis.

• Otherwise, histology with immunofluorescence is required.

• Treatment is with potent topical steroids, or prednisone, or other immunosuppressing drugs, or dapsone. Typically one continues treatment for years.

• Dressings are NOT a treatment – they simply make it more comfortable, and soak up the juice.
Elephantiasis verrucosa nostra

Occurs in some patients with lymphedema

Secondary lymphedema may be due to:

- Neoplasms
- Trauma
- Radiation treatment
- Congestive heart failure
- Hypothyroidism
- Chronic venous stasis
- Obesity
• Treat the underlying condition

• Reduce edema through compression etc

• Treat infection

• Topical or systemic retinoids may improve skin appearance (in my experience the results are disappointing)

• Blisters may arise as a result of the edema
Treatment basics

• Identify what the problem is.....

• Is this ulceration due to vascular insufficiency, pyoderma gangrenosum, erosive pustular dermatosis, pemphigoid?
• Its not uncommon to find combinations of things

• This is an obese patient with lymphedema, a history of multiple episodes of cellulitis in the legs, multiple co-morbidities, dermatitis neglecta, mild elephantiasis nostras verrucosa, actinic damage including SCC
• This person has stasis dermatitis, erosive pustular dermatosis, contact dermatitis

• Allergic contact dermatitis is fairly common in crappy legs, where there is impaired barrier function, and exposure to numerous creams and dressings over a long period
• Sorting out what you are dealing with might only require clinical examination, or it might require swabs, biopsy, assessment of the vascular status, allergy testing
• Try and treat the underlying problem, where possible.

• If there is venous insufficiency, deal with the problem (with compression, surgery, aescin, rutin etc)

• If contact allergy is possible, try and identify the allergen(s) so it(they) can be avoided
• About 50-70% of those with chronic ulcers will have at least one positive patch test

• Common allergens include wool alcohol, preservatives, colophony, antibiotics, antiseptics, and steroids.

• The composition of modern dressings such as hydrocolloids, is frequently not given on the packaging
• Several of the hydrocolloid dressings use modified colophony as the tackifying agent.

• Contact dermatitis has been reported with Nuderm, Duoderm extra thin, Comfeel, Combiderm, Tegaderm, Granuflex.

• While some patients react to colophony (rosin) on the patch test, most don’t. Some will react to glyceryl rosinate.

• Contact allergy to sorbitan sesquioleate (a surfactant/emulsifier) in Adaptic also reported.

• Hydrogels such as Intrasite contain carboxymethyl cellulose polymer plus propylene glycol (which is a potential allergen).
Which steroid?

- For **rapid response** in dermatitis, or for **erosive pustular dermatosis**, use a potent one like *dermol* cream or *beta (betnovate)* cream, for 1 or 2 weeks.

- For **long term use**, choose a mild one like *hydrocortisone* – you can use that indefinitely. Sometimes, something a little stronger like *aristocort* or *locoid* is necessary – try not to use those for longer than 6 weeks.

Avoid greasy things on the legs, as they cause folliculitis.
• If the skin is thickened by the dermatitis, use a potent steroid.

• If it's severe, use a potent steroid

• If it’s a steroid-responsive condition, and it’s also infected, use antibiotics + steroids.
Which moisturiser?

The only subsidised ones are:

- Aqueous cream
- Cetomacrogol cream
- Cetomacrogol cream with glycerol
- Emulsifying ointment
- HealthE fatty cream
- Urea 10% cream

- Urea cream can sting
- Aqueous cream has been shown to increase trans-epidermal water loss (i.e., aggravate dryness) – presumably because of the sodium lauryl sulphate content
- No one has tested TEWL with the others
• Aqueous cream contains WSP, emulsifying wax, liquid paraffin, phenoxyethanol and water

• Emulsifying wax is cetostearyl alcohol plus sodium lauryl sulphate as a surfactant

• Emulsifying ointment contains WSP, emulsifying wax and liquid paraffin

• i.e. aqueous cream is just emulsifying ointment plus water and a preservative. So, if aqueous cream is a problem, one assumes emulsifying ointment might be too.
• Other commercial moisturisers such as the Aveeno range (with colloidal oatmeal), QV, Neutrogena, or Cetaphil moisturisers are fine
Don’t think moisturiser will solve this problem. If there are plates of scale, like fish-scales or cornflakes, that can be easily peeled off – then remove them manually.

Treat any underlying skin problem.
• After flicking off these scales, the ongoing management is a gentle rub with a soft flannel.

• The problem is failure to shed dead skin cells and moisturiser doesn’t make you shed skin.
• If skin has been VERY inflamed – whether through rash, or sunburn, then as it heals it will peel. Moisturiser doesn’t solve that either.
• The best moisturiser is the one the patient likes.

• There are few comparative studies of moisturisers, and no good data to recommend one over another.
Barrier function

• Put simply the skin’s barrier is designed to keep the outside out, and the inside in

• The dead cells (corneocytes) on the surface of the skin form the barrier layer – the stratum corneum

• Think of the stratum corneum as being a stack of corneocyte pancakes.

• The pancakes are all buttered (ie there are lipids between each pancake layer) and the top pancake is spread with sebum
• The lipids (the butter) are made up with ceramides (50%), fatty acids, and cholesterol.

• After that it all gets bloody complicated!

• Filaggrin, serine protease and protease inhibitors are all controlled by genes

• But other factors influence these things eg proteases influence filaggrin and ceramide processing. Too much protease activity results in reduced processing and reduced barrier function.

• Increasing the pH (ie reduced acidity) increases protease activity
• Filaggrin degradation products increase acidity (and therefore reduce protease activity)

• Filaggrin degradation products are also hygroscopic ie they attract water and hydrate the barrier layer

• Filaggrin is also involved in the structural integrity of the corneocytes

• It is also involved in the production of chromophores which result in sun protection

• Sun damage and maybe chronologic age have effects on skin acidity, ceramide content, and filaggrin.

• Bacteria secrete proteases and interfere with lipid production

• Soaps reduce acidity and remove surface lipids

• Psychologic stress results in the production of glucocorticoids that reduce lipids
• And that’s the abridged version!

• Cetaphil Restoraderm contains filaggrin degradation products as well as ceramides

• It is certainly of value in some atopics. Its role in elderly sun damaged eczema-prone or itchy skin is less clear. It is relatively expensive.
What to cleanse with?

• Aqueous cream and emulsifying ointment are OK as wash-off products

• Cosmetically superior products:
  ▪ Aveeno body wash 354ml $22,
  ▪ QV wash 250 ml $14,
  ▪ Cetaphil gentle cleanser 250ml $17