In order to understand lymphoedema and chronic oedema skin breakdown and thus consider the most appropriate treatment options and strategies for patient education, the normal function and structure of the skin must be understood.

This abridged version of the chapter of the same name, from the International Lymphoedema Framework document, Best Practice for the Management of Lymphoedema (2nd ed.), outlines the skin conditions commonly seen in lymphoedema and chronic oedema and offers management strategies for these.

Key words:
Skin care in lymphoedema and chronic oedema
Function of the skin
Specific interventions

Renewal of the stratum corneum is a constant process, regulated by the action of proteases. If the process is affected by an imbalance of proteases and protease inhibitors, the stratum corneum may thin and crack, potentially allowing the entry of irritants and allergens and making skin barrier function less effective. In addition, decreased levels of NMF, particularly urea, and the breakdown of the lipid lamellae, cause skin to dry as in effect the ‘mortar’ holding the corneocyte ‘bricks’ together, crumbles, leading to greater TEWL.

Over-hydration of the skin (occlusion, prolonged hydration) will lead to water loss by enlarging and connecting the aqueous lacunae between the lipid layers, making the barrier ‘leaky’.

The effect of lymphoedema on the skin
Chronic disturbance of lymph flow results in chronic inflammation in the swollen body parts with enhanced activity and proliferation of cells contained in the epidermis, underlying dermis (including vessels) and fat tissue. The clinical signs resulting from these alterations are:

- thickening of the skin and of all underlying tissues (fat, connective tissue, fascia)
- hyperkeratosis
- papillomatosis
- hyperpigmentation
- fibrosis with loss of skin suppleness
- deepening of the skin folds

Papillomatosis: Papillomatosis produces firm raised projections on the skin due to dilation of lymphatic vessels and fibrosis, and may be accompanied by hyperkeratosis (Figure 2). Sometimes dilated lymph vessels may form cystic ‘vesicles’ leading to lymphorrhoea and fistulisation following rupture.

Skin is actively maintained in homeostasis by a dynamic repair response; after a disturbance, epidermal hyper-
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chronic oedema through the combined effects of hyperaemia, gravity, loss of lympho-venous pump and immobility.

Dry skin: Dry skin develops when alterations of the stratum corneum barrier lead to a loss of water, lipids or the natural moisturising factor (NMF) of the epidermis. The wicking effect of bandages may aggravate this, making the skin less pliable and elastic and prone to cracks and fissures. Dry skin may vary from slightly dry or flaky to rough and scaly. Barrier perturbation, mechanical factors (scratching the itchy skin), and application of irritant substances further delay recovery and lead to release of pro-inflammatory cytokines. In more advanced stages the skin may become dull red, oozing, crusty, excoriated and presenting nummular lesions of astuteotic eczema or irritant dermatitis.

Hyperkeratosis: Hyperkeratosis is caused by over-proliferation of the keratin layer and produces scaly brown or grey patches. It relates to mechanical trauma, for example, repeated low grade friction and repetitive mechanical trauma in suboptimal footwear (open heels, 'slippers') and under compression bandages at pressure sites. It must be distinguished from acanthosis nigricans in endocrinopathies like morbid obesity and the metabolic syndrome.

Maceration: In deep skin folds, occluded skin sites, and around areas with lymph leakage, the skin frequently becomes wet and macerated, losing its defence against infection, and allowing easy penetration of applied substances/allergens. An over-hydrated epidermis is more susceptible to blistering and breakdown.

Folliculitis: Is the inflammation of the hair follicles causing a red rash with pimplles or pusules, and is most commonly seen on hairy and/or occluded areas (head, trunk, buttocks, limbs). It may precede cellulitis/erysipelas. In some cases (i.e. irritant folliculitis), it caused by friction (compression treatment), or the application of occlusive substances such as petrolatum topical preparations.

Contact dermatitis: When applying topical medication, skin care products, or through occupational exposure, people suffering from chronic oedema (especially in venous disease) and lymphoedema are at risk for the development of allergic or cumulative irritant contact dermatitis. Signs may include itchy or painful fissures, desiccation, erythema and even vesicles, but predominantly lichenification and hyperkeratosis. Contact dermatitis (Figure 5) is the result of an allergic or irritant reaction. It usually starts at the site of contact with the causative material, but may spread. The skin becomes red, itchy and scaly, and may weep or crust.

Venous eczema: Also known as varicose eczema or stasis dermatitis, usually occurs on the lower legs, particularly around the ankles, and is associated with varicose veins (Figure 6). The skin becomes pigmented, inflamed, scaly and itchy.
Ulceration: Ulceration is unusual in primary lymphoedema patients; in most cases it is to be attributed to trauma or comorbidities/underlying diseases. It is important to establish the underlying cause because it determines treatment and whether compression is appropriate.

Lymphangiosarcoma: In the most severe cases of lymphoedema, lymphangiosarcoma, a rare form of lymphatic cancer (Stewart-Treves syndrome) can develop. It mainly occurs in patients who have been treated for breast cancer with mastectomy and/or radiotherapy. The sarcoma first appears as a reddish or purplish discoloration or as a bruised area that does not change colour. It progresses to an ulcer with crusting, and eventually to extensive necrosis of the skin and subcutaneous tissue. It can metastasise widely.

Skin care
Maintenance of skin integrity and careful management of skin problems in patients with lymphoedema are important to minimise the risk of infection.

The general principles of skin care include:
- washing daily, using pH neutral soap, natural soap or a soap substitute, drying thoroughly
- if skin folds are present, ensuring that they are clean and dry, monitoring the affected and unaffected skin for cuts, abrasions or insect bites
- applying emollients
- avoiding scented products
- using vegetable-based products rather than those containing petrolatum or mineral oils in tropical climates

The aim is to preserve skin barrier function through washing and the use of emollients. Ordinary true soaps have an alkaline pH of 9-10, so should be avoided because they dry the skin; natural or pH neutral soap can be used. Synthetic detergents (‘soap-free soap’), have a pH of 5.5-7 to minimise skin barrier disruption. Body wash emulsion systems combine a syndet with moisturisers or emollients. Lipid-free cleansers may contain glycerin and other emollients, while cleansing creams contain waxes and mineral oil. Moisturisers should be used after cleansing the skin in order to replace the lipid film barrier that has been disrupted by washing.

Emollients re-establish the skin’s protective lipid layer, preventing further water loss and protecting the skin from bacteria and irritants. In general, ointments, which contain little or no water, are better skin hydrators than creams, which are better than lotions. The best method of emollient application is unknown. Some practitioners recommend applying them using strokes in the direction of hair growth to prevent blockage of hair follicles and folliculitis. Others recommend applying emollients by stroking towards the trunk to encourage lymph drainage.

Skin care regimens
Skin conditions that can occur in patients with lymphoedema require careful management. They may occur simultaneously and require combinations of regimens. The general principles of skin care apply to all conditions and where an intervention has proved unsuccessful, the patient should be referred as appropriate.

**Intact skin/dry skin:** Apply emollients twice daily to aid rehydration. If heels are deeply cracked, emollients and hydrocolloid dressings may help.

**Hyperkeratosis:** Frictional and mechanical causes such as footwear, bandages, rubbing and scratching need to be addressed. Emollients with low water content are recommended. Lymphoedema compression bandaging (LCB) reduces the underlying lymphoedema and improves skin condition.

**Papillomatosis:** May be reversible with adequate compression, although if it does not improve after one month, refer to a lymphoedema service.

**Contact dermatitis:** Avoid causative irritants and allergens and restore epidermal barrier function. Acute episodes of contact allergic dermatitis are treated with a potent topical corticosteroid in ointment form, and should be reviewed after seven days. Treatment should continue for three to four weeks, during which time the strength of the steroid and amount applied are gradually reduced.

**Venous eczema:** Adequate compression treatment is expected to reverse the secondary skin changes seen in venous insufficiency including venous eczema. Treatment is with topical corticosteroids in ointment form for seven days, followed by a moderate corticosteroid.

**Lymphangiectasia:** Treatment is compression with lymphoedema compression bandaging (LCB). If there is no response to initial compression or the lymphangiectasia are very large, contain chyle or cause lymphorrhoea, the patient should be referred immediately to a lymphoedema practitioner.

**Lymphorrhoea:** The patient may require medical review to determine the underlying cause. The surrounding skin should be protected with emollient and non-adherent absorbent dressings should be applied to the weeping skin. Lymphoedema compression bandaging will reduce the underlying lymphoedema, but needs to be changed frequently to avoid maceration of the skin. Frequency will be determined by strikethrough and the rate of swelling reduction. In the palliative situation, light bandaging may be more appropriate. If there is no improvement after six weeks of treatment, the patient should be referred to the lymphoedema service.

**Folliculitis:** Swabs should be taken for culture if there is any exudate or an open wound. An antiseptic wash/lotion should be used after washing and emollient applied.

**Cellulitis/erysipelas:** Patients with lymphoedema are at increased risk of acute cellulitis/erysipelas, an infection of the skin and subcutaneous tissues. The cause of most episodes is believed to be Group A haemolytic streptococci.

Symptoms are variable. Episodes may come on over minutes, grumble over several weeks or be preceded by systemic upset. Symptoms include pain, swelling, warmth, redness, lymphangitis, lymphadenitis and sometimes blistering of the affected part (Figure 7). More severe cases have a greater degree of systemic upset, for example, chills, rigor, high
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fever, headache and vomiting. In rare cases, these symptoms may be indicative of necrotising fasciitis.

The focus of the infection may be tinea pedis, venous eczema, ulceration, ingrowing toe nails, scratches from plants or pets, or insect bites. Box 1 outlines the principles involved in the management of acute cellulitis/erysipelas at home or in hospital.

It is essential that patients with cellulitis/erysipelas, who are managed at home, are monitored closely, ideally by the general practitioner. Prompt treatment is essential to prevent further damage that can predispose to recurrent attacks.

Criteria for hospital admission
The patient should be admitted to hospital if they show:

- signs of septicemia (hypotension, tachycardia, severe pyrexia, confusion or vomiting)
- continuing or deteriorating systemic signs, with or without deteriorating local signs, after 48 hours of oral antibiotics
- unresolved or deteriorating local signs, with or without systemic signs, despite trials of first and second line oral antibiotics

Conclusion
Lymphoedema and chronic oedema can have a major impact on skin function. While some of the conditions are relatively easy to manage, others, such as cellulitis, can present an emergency situation.

Prevention of course, is key, and community practitioners are ideally placed to work with patients and their family to implement prevention and early management strategies.

References

Reflection Points
1. How relevant is skin care of lymphoedema patients to your practice?
2. After reading this article, would you explore or change your practice? In what ways would you do this?
3. How can simple skin care alleviate or prevent some of the lymphoedema-associated conditions discussed in the article?