The lymphatic system plays an integral role within the body’s circulatory system. When this fails to work effectively, however, morbidity levels rise for the individual, as does the demand placed on healthcare services. Failure of the lymphatic system leaves individuals with long-term chronic conditions that require regular input from a range of health professionals.

Oedema
Oedema can be either acute or chronic and consists mainly of water, but oedema that arises as a result of the failure of the lymph system is also rich in protein (MacDonald and Geyer, 2010). Chronic oedema is an umbrella term that incorporates conditions such as lymphoedema, lymphoovenous oedema and dependency oedema, which appear when the lymphatic system fails. It is important that the presence of lymph stasis is recognised as this indicates the failure of the lymphatic system.

Lymph stasis can be identified following a holistic and comprehensive assessment. When chronic oedema affects the limbs, in particular the legs, a number of problems can arise that challenge the impact on the individual’s daily activities. Once the underlying cause of oedema is established/diagnosed, then the correct management regimen can be formulated.

Chronic oedema is, as the name suggests, a chronic illness, which is multifactorial and often associated with ageing (Bodenheimer et al, 2009). Moffat et al (2003) suggest that the prevalence of chronic oedema is likely to be around 5.4/1,000 in those aged 65 years and above, as opposed to 1.33/1,000 in those younger than 65 (Moffat et al, 2003). The UK Nursing and Midwifery Council (NMC) has observed that two-thirds of acute general NHS hospital beds are occupied by people aged 65 years and over (NMC, 2009). It advocates that the nurse’s role should promote independence and wellbeing as the majority of chronic illnesses that affect the lives of older people can be prevented (NMC, 2009).

The Oxford Institute of Ageing’s working papers (OIA, 2009) state that approximately one in six workers will report a chronic illness at some point. Those who are aged 45 and over are twice as likely to report this, and of those aged 55–64, heart disease and musculoskeletal disorders are most prevalent. In 2009 the Health and Safety Executive (HSE) commissioned a report focusing specifically on the prevalence of lower limb musculoskeletal disorders and found that the studies reflected the nature of the problem in different occupations, for instance, the incidence in military recruits was as high as 79.8%, while nursing was 41.5%. The report concluded that the most commonly identified disorders and injuries were — hip, osteoarthritis (OA), knee, bursitis and meniscal damage, stress fracture, and venous disorders (HSE, 2009).

It is well documented that musculoskeletal disorders can have a profound effect on the circulatory system, particularly the veins (Panny et al, 2009; Widgerow, 2009). In US society, chronic venous disease (CVD) is prevalent in around 20% of the population (Deatrick et al, 2010) and in a large-scale, cross-sectional study conducted in Edinburgh, of those persons aged 55–64 there was a prevalence of 60% (Evans et al,1998). Williams et al (2005) suggest that CVD is a contributing factor to the development of chronic oedema and should be identified by a thorough clinical differential diagnosis.

Most studies generally have a small participant cohort that may not be truly representative of the population, however, a study conducted in the UK in 2003 looked at a reasonable-sized (n=689) population of patients with venous leg ulcers in the community and found that oedema was present in 77% (Adam et al, 2003).

Skin disorders
Skin disorders occur for a number of reasons in individuals with lymphoedema. Bacterial infections are commonly seen but not always recognised as a cause of lymphoedema. Cox (2006) identified that 46% of patients admitted for cellulitis had lymphoedema and that around 30% of these patients with one episode of cellulitis will see a recurrence (Cox, 2006).

Woods (2007) suggested that repeated bacterial infections that involve both the superficial and subcutaneous layers of the skin can cause damage to the lymphatic channels due to constant inflammation.

Justine Whitaker, MSc, RN, is Director & Nurse Consultant, Northern Lymphology Ltd and Senior Lecturer, University of Central Lancashire.
which, in turn, causes scarring/fibrosis. This constant bombardment of infections was initially thought to be the cause of lymphoedema associated with the chronic inflammatory disease, psoriasis, however; Kunstfeld et al (2004) demonstrated that skin lesions due to psoriasis show lymphatic hyperplasia — a known cause of lymphoedema. It is well documented that effective management of lymphoedema can significantly reduce the incidence of cellulitis, therefore, reducing the possible need for hospital admission (Mortimer, 1996; Todd, 1999). Todd (1999), demonstrated a reduction of cellulitis incidence by 66% after six months of lymphoedema treatment.

Dermatitis
Skin inflammation, commonly known as ‘dermatitis’, is a broad term under which many other specific conditions fall. One such condition is eczema. The term ‘eczema’ comes from the Greek for ‘boiling’ in reference to the small blisters that are often seen at the earlier and more acute stages of the disease, but less so as the condition becomes more chronic (Weller et al, 2008). There are many types of eczema, such as ‘gravitational’, which describes venous stasis and is also associated with lymphoedema.

The many types of eczema all share the activation of keratinocytes. Keratinocytes are the most common type of skin cell and are responsible for producing keratin, which is the protein found in hair and nails. A proliferation of keratin is known as ‘hyperkeratosis’, a skin problem that falls within stages two and three of The International Society of Lymphology (ISL) classification of lymphoedema (Best Practice Document, 2006). Within eczema, keratinocytes metabolise quickly and this is associated with the increased production of cytokines. In increased numbers, cytokines cause oedema, blistering and weeping, as well as itching (Weller et al, 2004). All of these symptoms are commonly associated with lymphoedema and CVD.

Ulceration
CVD is also commonly associated with ulceration, particularly of the lower part of the limb, and it is essential to recognise that CVD is a crucial component that will hinder healing and the prevention of infection. Browse et al (2003) state that lymphoedema does not cause venous ulceration. However, according to recent findings, MacDonald and Geyer (2010) suggest that oxygen free radicals, fibrin cuffing and white cell sequestration, contribute to the repair of tissue in a chronically inflamed environment, thus increasing protein permeability. It is this process that in turn damages the lymphatic system. The oedema, being protein rich, develops into the principal pathology contributing to the development of ulceration.

When lymph stasis is present alongside a wound, then the underlying pathophysiology and an understanding of the relationship between the two must be established. MacDonald and Geyer (2010) state: ‘Chronic venous insufficiency leads to venous hypertension, which results in a high filtration pressure causing increased fluid to appear in the tissues, for example, increased lymphatic water load. When the lymphatic transport capacity is exceeded by the water load, a state of low protein oedema occurs after this dynamic failure. Constant lymphatic hypertension causes infiltration of lymph into the perilymphatic tissue, resulting in fibrosclerosis and lymphangitis’ (Figure 1).

The lymphatic system has a series of chambers called lymphangions, which contract and relax, thus moving lymph to the next chamber and up back towards the thoracic duct. The contractions move in a peristaltic manner; which is important to consider when formulating the most appropriate treatment regimen, as this peristalsis works by emptying and refilling the chambers, just like the venous system. The lymphangions require pressure changes to do this when they are not working effectively due to an underlying pathology. Developing an understanding of this mechanism will contribute to the healing of the chronic wound.

Periwound skin
The preparation of the wound bed and the type of medium used to assist
healing are equally important, forming part of the holistic approach to wound rehabilitation. The area surrounding the wound is often referred to as the periwound skin and it can be vulnerable, despite often appearing quite healthy. The All Party Parliamentary Group on Skin, after carrying out research into skin diseases in elderly people in 2000, estimated that 70% had skin problems that significantly impact on all aspects of daily living. Lawton and Langøen (2009) identified several risk factors that contribute to the vulnerability of periwound skin, venous ulcers being one of them. This is due to the potential for maceration around the wound due to excess exudate.

A wound which has surrounding oedema from lymph stasis is likely to have a greater amount of exudate than one which has no oedema. Lawton and Langøen (2009) also state that macerated skin is more permeable to micro-organisms and prone to damage from friction and irritants than intact skin.

**Overcoming hyperkeratosis**

Hyperkeratosis is the build-up of keratin on the skin’s surface, causing the skin to appear dry and scaly. In between these scales, cracks appear and the mortar which binds the skin cells in normal conditions breaks down allowing foreign bodies to enter, thus causing cellulitis. Removing the dry scaly skin allows emollients to penetrate, rebuilding the mortar and rehydrating the skin. Understanding the underlying cause of the lymph stasis aids the clinician in formulating a comprehensive treatment plan. Recognising what may have originally caused the oedema is paramount to preventing an individual from deteriorating and potentially becoming hospitalised. Good self-management and long-term support are crucial to ongoing care of this condition.

Thorough skin hygiene with lymph stasis is paramount. Washing and showering the limb each time the bandage is changed should be equal in priority to getting the right type and level of compression on the limb. When the correct compression is applied, then the limb will reduce rapidly and effectively. It is this reduction in volume that accentuates the production of dead dry scaly skin, making the problem initially look worse. Ensuring the removal of this debris on the skin rapidly and effectively is important to the skin condition, especially as further maceration.

**Figure 2. Dry scaly hyperkeratosis, prior to the use of Debrisoft.**

**Figure 3. A patient’s legs after using Debrisoft.**

Case report

Many case studies show very dramatic results in extreme cases. The case study below highlights that intervention should be made early to prevent problems occurring later.

This female patient had previously experienced leg ulcers and was managing in compression hosiery as prescribed, however, she developed cellulitis and was dismayed as she had been scrupulous with her self-management regimen.

On assessment, although she was wearing compression stockings, they...
were ill-fitting and the pressure levels (18-21mmHg) were inappropriate. She had also been advised to apply cream daily, which she duly did, but the nurse recommending this had not appreciated that she only washed her legs once a week.

An accumulation of emollient, coupled with the ill-fitting low pressure stockings had contributed to her cellulitis. Dry scaly hyperkeratosis had built up (Figure 2). This is a very common picture but is often enough to cause infection.

On this occasion, she was not hospitalised as she came to the author early enough to receive appropriate care. This care regimen was:

- Complete showering of the limb
- Use of Debrisoft (once only)
- Reapplication of the appropriate amount of emollient.
- The use of an Actico® (Activa) bandage with Cellona® (Activa) undercast padding and Actiwrap® (Activa) to toes.

This was completed with six appointments over two weeks after which 23–32mmHg compression stockings were fitted. Figure 3 shows the legs directly after using the Debrisoft and light application of emollient.

**Conclusion**

Lymphoedema is not a ‘static’ disease and should be revisited regularly to ensure that the patient is on track with their self-management regimen.

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- Use of Debrisoft (once only)
- Reapplication of the appropriate amount of emollient.
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References


