MANAGING ULCERATION CAUSED BY OEDEMA

Lower limb oedema can result in ulceration, lymphoedema and poor quality of life for patients. This article examines the mechanics of oedema and how nurses can take steps to manage patients, the importance of effective early interventions, compression bandaging and adequate skin care.



Figure 1. Ulceration of the toes, which is extremely painful and difficult to treat.

The relationship between leg oedema (abnormal accumulation of fluid beneath the skin) and leg ulcers has been well documented for many years, as has the relationship between compression (in the form of bandaging) and healing rates (Myers et al, 1972; Cherry et al, 1996). Less clear for many practitioners is the pathophysiology of leg oedema, how to diagnose the underlying cause(s) of oedema, when it is appropriate to apply compression and what advice to give patients.

This article will describe the normal cycle of tissue fluid, the mechanisms that can disrupt this leading to oedema and ulceration, and the factors leading to lower limb oedema.

Normal flow of tissue fluid in the legs.

In the healthy person there is a continuous flow of fluid from the arterial portion of the capillary bed from where it is filtrated into the tissues. The arterial pressure in the capillaries is sufficient to force 20–30mls per hour through the capillary walls. This would cause rapid swelling of the feet were there not contrary mechanisms in place. These are:

 Pre-capillary vasoconstriction (narrowing of the blood vessels)

- Reabsorbtion of fluid and protein molecules into the lymphatic system
- Return of lymph to the venous system via lymphatic vessels.
 This is dependent on a healthy lymphatic system and enhanced by muscular activity, which massages the lymph vessels promoting lymphatic return.

Factors causing leg oedema

According to Mortimer and Levick (2004) 'All oedema is due to an imbalance between capillary filtration and lymphatic drainage.'

Lower limb oedema can be caused by one or more of the

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following, and it is essential that a differential diagnosis is made of the cause of the swelling as it may be life-threatening (Green and Mason, 2006):

- Raised capillary pressure caused by venous incompetence and cardiac failure
- Raised permeability in the capillaries meaning fluid can leak. This is due to hormones, medication or vasodilatory influences, such as heat and inflammation
- Changes in the oncotic pressure gradient (pressure exerted by blood plasma that pulls water into the circulatory system) as seen in renal failure, malnutrition
- Lymphatic blockage, malfunction, damage or absence. This may be due to congenital abnormality, surgery, radiotherapy, infection or tumour growth and is aggravated by immobility and/ or obesity.

Venous oedema

In the developed world, the most frequent cause of leg oedema associated with ulcers is chronic venous insufficiency (CVI). This is mainly due to valve damage and/ or calf muscle pump failure (each time the calf muscle contracts, it squeezes the deep veins, emptying these veins of blood).

Deficient or damaged valves in the veins lead to a backflow of blood causing venous hypertension (high pressure in the veins of the leg). This is exacerbated by immobility, which inactivates the pumping mechanism of the calf, and obesity which may compromise the drainage of the venous and lymphatic system in the groin causing a vicious circle of increased swelling, heaviness of the leg and immobility.

Skin changes

The high pressure in the veins is transmitted to the capillaries whose walls become leaky so that fluid, fibrin (protein involved in the clotting of blood) and cells are forced into the tissues causing oedema, lipodermatosclerosis (a condition that affects the skin just above the ankle), and heamosiderin staining (a brown or rusty discoloration of the skin) (*Figure 2*).

Venous eczema may also develop secondary to an inflammatory response to the deposits in the skin. Skin problems such as blistering, leakage and maceration may occur as a consequence of accumulated fluid in the tissues. The presence of tissue fluid also blocks the microcirculation causing tissue anoxia (extremely low levels of oxygen) and is a contributory cause of ulceration.

The progression from venous oedema to lymphoedema

If venous oedema is left untreated lymphatic changes may lead to lympho-venous oedema. There is no clear cut-off point between venous oedema and lymphoedema. The progression from venous oedema to lymphoedema is not inevitable and obesity would seem to be a major factor. As the limb becomes filled with fluid it becomes increasingly immobile as does the whole patient.

If oedema is not controlled, the lymphatic system becomes



Figure 2. Patient's legs featuring haemosiderin staining.

overwhelmed and ineffective. The leg may become more swollen with increased levels of fluid and the oedema becomes more solid so that it no longer 'pits' (a dent is not formed when pressed with a finger) and will not reduce after long periods of elevation.

The skin becomes thickened (hyperkeratosis) with raised papules (papillomatosis). The skin may also break down with lymph exuding through the skin breaks (lymphorrhoea) often in copious amounts leading to maceration, ulceration (sometimes even of the toes which is extremely painful and difficult to treat) (*Figure 1*) and increased risk of infection.

Lymphoedema is associated with recurrent cellulitis (skin infection caused by bacteria), known as acute infective episodes (AIEs), which further damage the lymphatics leading to a vicious circle of worsening oedema. AIEs are probably the most serious complication of lymphoedema (Mortimer, 2000).

Ryan (2001) has described the progression of skin changes in severe lymphoedema: 'Increased fibrosis in the subcutaneous tissues resist further swelling but gives way in some sites to produce gross swelling in others





Figure 3. Extreme swelling showing ulceration.

which thickens ... in severe lymphoedema there is a huge unevenness in the size and hardness of the tissues' (*Figure 3*).

Early intervention to interrupt these worsening cycles can have a huge impact on disease progression, patient quality of life and costs.

Management

Hygiene, skin care, exudate management, weight loss, exercise and compression are the components of effective management.

Hygiene and skin care

Hygiene and skin care are crucial to prevent deterioration and ulceration (Linnitt, 2000). Regular washing with a soap substitute, paying particular attention to fissures and areas between the toes will help prevent infection and tissue breakdown.

Application of emollients will help keep the skin supple. If patients wear compression bandages they will be unable to carry out their own care, therefore nurses must be allowed time in primary care to carry out this essential task. Ten minute appointments are not sufficient.

Weight loss and exercise

Weight loss and exercise are

very important. Patients must be encouraged to take responsibility for their own health. Small amounts of exercise, fidgeting, and dorsiflexion (bending backward) of the ankle will all aid lymphatic return.

Deep breathing may help drain the central lymphatic system. A gradual increase in exercise programmes should be introduced in conjunction with weight loss. Many patients will need a lot of support and encouragement to tackle this aspect of their care.

Exudate management

Where there is uncontrolled oedema, lymphorrhoea will occur as lymph leaks out of highly stretched skin. Ulceration in conjunction with oedema will be like a 'hole in a bucket' and fluid will pour out. Reducing exudate will prevent further skin breakdown. Exudate leakage and skin damage may be reduced by:

- Leg elevation: periods of rest with the leg elevated (where possible higher than the heart) will help drain fluid from the limb
- Topical treatments: application of topical steroid (i.e. Trimovate® cream [GlaxoSmithKline]) in conjunction with absorbent dressings will help dry an area of lymphorrhoea and will help to treat superficial infection. If there are any heavily leaking ulcers the surrounding tissue will need to be protected with highly absorbent dressings, i.e. Kerramax[®] (Ark Therapeutics) Sorbion[®] Sachet S (H&R) Healthcare) is useful if an even higher absorbency is required. Sorbion Sana (H&R

Healthcare) is useful for painful ulcers where adherence is a problem. The expense of such dressings may be offset by fewer dressing changes. In the author's experience, foam dressings are not able to handle copious amounts of exudate

 Compression bandage therapy is a crucial part of oedema and exudate reduction.
Prior to application, vascular assessment must be carried out to rule out arterial disease.

Vascular assessment of oedematous limbs

The RCN (2006) states that 'measurement of the ABPI [anklebrachial pressure index] is essential to rule out arterial insufficiency in patients with an ulcerated limb' prior to applying compression.

However, in patients with significant leg oedema this may prove difficult. In a patient with foot oedema, a 5MHz probe may be helpful in picking up the signal, but Doppler examination will be inaccurate in patients with very enlarged legs with tense oedema as it will be impossible to compress the artery with a cuff. This does not mean that it is impossible to carry out vascular assessment.

If there are strong palpable foot pulses the ABPI will be normal. The Doppler signal is also an indicator of arterial disease with biphasic and triphasic signals suggesting healthy elastic arteries. If the signals are monophasic, compression should be applied with caution. Arterial flow can be further assessed by wave form analysis and toe pressures. Some practitioners will not feel competent to make a decision about applying compression without an ABPI result and such patients should be referred for expert advice from tissue viability nurses or vascular units.

Effective compression

Effective compression aids oedema reduction and ulcer healing because it:

- ▹ Reduces capillary filtration
- Moves fluid to a noncompressed area
- Increases venous and lymphatic return
- Aids the breakdown of fibrotic tissues (Foldi et al, 2005)
- Early application of compression in the presence of leg oedema can prevent its progression into irreversible oedema and leg ulceration.

Choice of bandage system

Hosiery alone is not an effective tool for reducing oedema. In order for a stocking to be effective, oedema must be reduced first by bandaging. Stockings alone are useful in the ongoing management of patients whose venous ulcers have healed to prevent recurrence. In some patients, oedema recurs despite them wearing hosiery. This causes the stockings to become too tight and short periods of compression bandaging should be re-introduced.

All too often ulcer recurrence is a result of patients ceasing to wear compression hosiery because they can no longer pull them on when their leg is swollen. While the ulcer is active, bandaging is more effective except where friction from the bandage may exacerbate the ulcer or there are other contraindications.

Short stretch versus long stretch?

Randomised controlled trials showing the advantage of one bandage system over another are inconclusive, but it has been reported that inelastic (short stretch) bandages achieve more pronounced haemodynamic effects than elastic material (Partsch, 2006).

Short stretch bandages are used by lymphoedema specialist nurses in the intensive phase of lymphoedema management and can continue to be effectively used once oedema has been reduced through to ulcer healing. In the author's opinion, cohesive short stretch bandaging has the advantage over some other bandage systems in that it is less bulky than four-layer bandaging.

If applied to an oedematous limb, the rapid reduction in oedema will cause the bandage to slip so that it should be checked within a day or two after application.

As with any bandage system, a cotton tubular bandage should be used as the contact layer to protect leg from potentially itchy padding, followed by sufficient padding to protect against injury and to shape the leg.

Misconceptions

Over the years, some misconceptions have arisen about bandaging that can have an adverse effect on optimal care for patients with leg oedema, for example, that short stretch bandaging is ineffective in the management of oedema in the immobile patient (Vowden, 2005). It would seem that this 'myth' originally arose because of promotional material from the companies stating that inelastic material would work only during exercise, leading to the assumption that if the calf muscle was inactive, the bandages would be ineffective. This has subsequently been perpetrated in the literature (Marston and Vowden, 2003). However, in the author's opinion this has been demonstrated not to be the case in practice and anecdotal evidence shows marked reduction of oedema in immobile patients. Moreover, short stretch bandages have always been used by lymphoedema specialist nurses with good success even in patients with restricted mobility.

Another statement in the guidelines is that compression bandaging should not be applied if a patient has an ABPI lower than 0.8 (RCN, 2006). However, the presence of lower limb oedema constricts capillary circulation whereas compression bandaging reduces oedema and improves arterial inflow. Logically, therefore compression bandaging should not routinely be withheld for patients with leg oedema and low ABPIs as withholding compression will result in increased oedema and further constriction of capillary circulation with tissue death and an extension of ulceration. Patients with mixed aetiology ulceration should be under the care of a tissue viability team.

In addition, uncontrolled oedema will lead to 'leaky legs' and *pseudomonal* colonisation. Specialist nurses have been managing patients with ABPIs lower than 0.8 with short stretch compression bandaging for many years and reporting their results (Hopkins, 2008).

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Working groups are currently discussing the way forward in developing guidelines for this difficult-to-manage group of patients.

Conclusion

The provision of education for practitioners who care for patients with lower limb oedema is vital and should form part of the student nurse's curriculum together with chronic wound management. The rationale for compression bandaging should be clearly understood and practitioners should be encouraged to gain expertise in application as inappropriately applied bandages can result in tissue injury, which may be catastrophic. Withholding compression from patients in whom it is indicated can also have severely deleterious results.

Patients should be given clear information about how they can help themselves in order that they understand the importance of skin hygiene, emollients, weight loss exercise and compression.

Further research needs to be undertaken regarding compression bandaging in patients with mixed aetiology disease. Meanwhile, these patients should have specialist input into their management as this chronic condition not only has an impact on healthcare resources, but also causes longterm misery for patients (Woods, 2000). WE

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Key points

 There are many causes for chronic leg oedema and a patient may suffer from more than one of these. Before treatment all possible causes should be identified.

- Early intervention can prevent leg oedema from progressing to lymphoedema.
- >> Weight loss (where necessary), exercise and skin care are all important aspects of oedema management together with compression bandaging.
- Misconceptions about methods of bandaging need to be further discussed and addressed by appropriate research.

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