Lymphovenous disease and its effects on the lower limb

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This article highlights the normal and abnormal physiology of fluid circulation in the lower limb with particular reference to venous and lymphatic flow. Consideration is given to the assessment and diagnosis of disease and its severity, particularly the impact on the patient including chronic oedema, ulceration, cellulitis and loss of mobility. Options for disease management and prevention such as compression bandaging and hosiery are also discussed, and the gaps between theory and practice are highlighted to provide community nurses with the most up-to-date information and practical solutions.

KEYWORDS: Oedema Wound care Leg ulcers Lymphovenous disease

Disease of the vascular system in the lower limbs can cause significant patient morbidity and in some cases lead to higher incidences of related mortality (Williams, 2012). Venous disease in particular also has a major impact on healthcare resources, as the leg ulcers that can develop are costly to treat and often return without careful management.

Lymphatic pathophysiology secondary to chronic venous insufficiency is often overlooked (White et al, 2014), sometimes because of a lack of awareness on the part of clinicians; a lack of understanding of its clinical relevance; or lack of time available for further investigations. This article will examine the development of lymphovenous disease of the lower limbs and the options for diagnosis and management. It will also consider the implications for the patient of poorly managed disease, particularly the development and management of wounds.

PHYSIOLOGY OF LOWER-LIMB CIRCULATION

In a healthy lower limb, hydrostatic pressure, coupled with other forces such as osmotic and oncotic pressure (a form of pressure exerted by proteins in a blood vessel’s plasma), cause fluid and solutes circulating in the arteries to diffuse across the capillary walls. This provides cells around the capillary beds with the required nutrients to remain metabolically active. Simultaneously, any fluid and/or dissolved waste products from cellular metabolism are reabsorbed from the interstitial spaces (Green and Mason, 2006), mainly by the lymphatic drainage system (Topham and Mortimer, 2002).

During normal lower limb circulation, the mechanical forces of the foot and calf muscles (the calf-muscle pump) exert graduated compression on the veins and lymph vessels, forcing fluid back towards the heart (Froniek, 2007). Simultaneously, venous and lymphatic return is aided by valves that prevent gravitational backflow of fluid to the limbs (Meissner, 2005). This muscle pump action is responsible for approximately 90% of the fluid returned to the heart from the veins of the lower leg (Meissner, 2005).

Fluid accumulates in the interstitial spaces when the lymphatic drainage rate and/or the rate of venous return is lower than the capillary filtration rate (Topham and Mortimer, 2002). In turn, oedema becomes clinically detectable when this imbalance has doubled the interstitial fluid volume (Topham and Mortimer, 2002).

Lymphoedema

The Lymphoedema Framework (2006) highlights that despite the accumulation of fluid in the interstitial space being the overriding clinical symptom of lymphoedema, the disease process may differ.

Primary lymphoedema refers to a group of conditions where there is a genetic link or congenital defect that affects the lymphatic system directly.

Red Flag Chronic oedema

Chronic oedema presents as tissue swelling, often in the lower limbs, but also in other areas such as the trunk, breasts and genitalia. Skin changes due to chronic lower limb oedema can be severe and include:
- Hyperkeratosis (thickening of the outer layer of the epidermis)
- Papillomatosis (growth of numerous papillomas or warts)
- Lymphorrhoea (leakage of lymph fluid from the skin).

These symptoms are all distressing in their own right and can contribute to ulceration if not properly managed.
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Secondary lymphoedema includes all conditions where damage occurs to the lymphatic system as a result of other causes, for example in patients who have undergone radiotherapy or experienced trauma.

Because of the effect of anatomical defects on the venous circulation (for example, valve deformity and varicosity) in up to half of cases of secondary lymphoedema, a greater awareness of combined lymphatic and venous disease has emerged over the past 15 years (Hardy and Taylor, 1999; Timmons and Bianchi, 2008). However, without any accurate estimates of lymphovenous disease prevalence, it is difficult to appreciate the impact of the disease on the wider health system.

**PATHOPHYSIOLOGY**

Lymphovenous disease is thought to be precipitated by disease of the venous system. Meissner et al (2007) described changes to the endothelium (thin layer of cells that lines the interior surface of the veins), particularly in the legs and feet that result from one of three main causes:

- Primary venous hypertension
- Obstruction of venous outflow
- Inefficient muscular activity in the calf and foot.

Whichever pathology is present, the changes to the venous vasculature are largely similar. Increased vessel dilation causes the valves in the veins to become incompetent, meaning that they cannot fully close during normal venous return (MacGregor, 2013). Fibrosis or scarring of the venous endothelium can also occur, which reduces the vein's elasticity and ability to tolerate increased pressures during venous return. According to Meissner et al (2007), this is particularly problematic in cases where venous thromboembolus (a blood clot blocking the vein) has caused a past obstruction and remnants of the clot have adhered to the endothelium.

In one prospective multicentre cohort study by Kahn et al (2008), 43% of patients from a sample of 387 with acute deep vein thrombosis (DVT) had some degree of post-thrombotic syndrome — a venous disease following acute DVT and which may account for the fibrosis and lumen changes (the lumen is the inside space of the vein) that develop in some patients with lymphovenous oedema.

The overall effect of incompetent venous circulation is to increase local hydrostatic pressure, which in turn increases permeability in the capillary beds (Meissner et al, 2007). This outflow of excess fluid from the circulatory system overwhelms local lymphatic drainage mechanisms and results in fluid accumulation.

As the pressure of interstitial fluid builds, lymphatic vessels become compressed and fluid uptake is further reduced (Bianchi, 2013). A diagnosis of chronic oedema is made if the oedema does not spontaneously reduce or is otherwise managed by correcting a primary cause (such as congestive cardiac failure) within three months.

**Chronic oedema**

When the oedema becomes chronic, early changes to the structure of the dermis and subcutaneous tissues can be detected, including brown hyperpigmentation caused by haemosiderin deposits, redness caused by vascular dermatitis and atrophie blanche (Timmons and Bianchi, 2008).

If left untreated, these changes may become irreversible, even if the oedema is managed, and can result in significant morbidity, including (Lymphoedema Framework, 2006):

- Recurrent cellulitis
- Ulceration
- Loss of mobility
- Negative self-image.

**DIAGNOSIS**

Although effective treatment of lymphovenous disease relies on accurate diagnosis of the underlying pathology, patients often do not present until they experience symptom-related morbidity, such as increasingly limited mobility as a result of heavy legs; uncontrollable leaking in the lower limbs; (lymphorrhoea); or ulceration. Alternatively, signs of lymphovenous disease/oedema are often identified incidentally when patients present to health professionals with other conditions (Timmons and Bianchi, 2008).

Topham and Mortimer (2002) recommended that community nurses should obtain a detailed clinical history, taking account of any pre-existing medical conditions that may have increased the patient’s capillary filtration rate, such as DVT, cardiac failure, reduced serum albumin and infection. It is also important to consider impaired drainage to the lymphatic system caused by primary or secondary lymphoedema.

Timmons and Bianchi (2008) recommend simple methods of assessment in the first instance, including thorough physical examination of limb, measure of limb circumference and the skin’s condition. Lower limb swelling that is ‘pitting’ or skin with a ‘woody’ texture should be recorded in the patient’s notes. Similarly, any flaking, dry or scaly skin, brown discoloration (haemosiderin deposits), or chronic ‘eczematous’ redness should also be recorded.

An assessment of the vascular status of a patient’s limbs using hand-held Doppler (ultrasound assessment of arterial flow to determine the ankle brachial pressure index [ABPI] and elasticity of the artery walls) might also preclude the need for further, more invasive and expensive investigations such as lymphoscintigraphy (lymph node mapping), or magnetic resonance imaging (MRI), and provide enough information to safely start treatment (Griffin, 2014).

The International Lymphology Society (ILS, 2003) suggest that duplex ultrasonography of the venous system may be useful and the Lymphoedema Framework (2006) identifies a multitude of other diagnostic tools that can help in obtaining an accurate diagnosis. However, due to the varying recommendations about diagnosis and the lack of a single, clear
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diagnostic test for lymphovenous disease, it is not surprising that management is often misdirected. For example, in the author’s experience, patients with peripheral oedema are often treated with diuretics, regardless of the cause.

Inappropriate disease management can lead to unnecessary complications and a poor outcome for the patient. A diagnosis of lymphovenous disease should only be arrived at when there is compelling clinical evidence that a combination of increased capillary filtration rate (often secondary to a specified pathology such as heart failure or post-thrombotic syndrome), with a suspicion or confirmation of impaired lymphatic drainage (due to primary or secondary lymphatic dysfunction), has led to the development of a chronic oedema (oedema present for greater than three months, which does not resolve on limb elevation) (Green and Mason, 2006; Lymphoedema Framework, 2006).

DISEASE-IMPACT ASSESSMENT

Disease management should be directed by consideration of specific elements of underlying pathology in conjunction with the disease stage (Bianchi, 2013). For example, if a patient has signs of mild disease with low levels of oedema and no history of associated complications, lifestyle advice regarding exercise, leg elevation, skin care and weight loss may be enough. However, a patient presenting with more advanced disease (long-term dense oedema, very dry skin, recurrent ulceration) would require more aggressive treatment.

As the disease progresses, the lymph vessels and veins become increasingly less amenable to simple management strategies and may require very intensive emollient therapy, treatment with antifungals and high levels of compression.

Disease staging is useful for guiding further investigations or directing referral to other specialists. For example, the ILS (2003) has a four-tiered staging approach that considers the behaviour of interstitial fluid and its impact on soft tissues.

Timmons and Bianchi (2008) recommend a continuum of disease staging that moves from mild to severe based on a whole spectrum of signs and symptoms seen during disease development, ranging from spider veins to severe hyperkeratosis. This type of guidance can be useful when determining the intensity of management required.

The Lymphoedema Framework (2006) has acknowledged the lack of clarity around the stages of lymphovenous disease and recommends a pragmatic approach in which clinicians produce a clear account of the signs and symptoms of disease through assessment of the following:

- Limb circumference
- Limb volume: using strategies such as bioimpedence testing (the use of electrical current to assess the body’s water content)
- Vascular system status: using Doppler ultrasound and/or duplex scanning.

Having a clear record of any observations and/or objective clinical tests that were carried out will help clinicians to gauge the effectiveness of any treatment and monitor disease progression in the future. Treatment is likely to be multimodal and dependent upon both the clinical signs of disease and the patient’s expectations and requirements (Lymphoedema Framework, 2006).

PREVENTING ULCERATION

Ulceration is a common problem in patients with lymphovenous disease and develops primarily as a result of problems with inadequate venous return. Venous leg ulcers are a sign that disease has progressed to a moderate or severe stage (Timmons and Bianchi, 2008).

There is not a huge amount of evidence to suggest conclusively that disease progression and the development of venous ulcers are entirely preventable in this patient group. However, in one venous leg ulcer prevalence study, Moffatt et al (2004) found that 42% of the leg ulcer patients in the study had some form of lymphatic-related oedema. This highlights that chronic oedemas may play an important part in the development of future ulceration.

Vandongen and Stacey (2000) provide some compelling evidence that compression can reduce some of the features of mild-to-moderate lymphovenous disease such as ulceration. Their randomised controlled trial focused on reduction of lipodermatosclerosis and preventing re-ulceration in patients who had already experienced a venous leg ulcer. In their study, 153 patients were randomised to use compression hosiery or no hosiery at all in the period after their ulcer had healed. The study reported a significant improvement in lipodermatosclerosis (inflammation of the layer of fat under the epidermis) and a lower incidence of ulcer recurrence in the hosiery group compared to the non-hosiery group.
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Similarly, Mîlc et al (2010) used a randomised control trial to demonstrate that venous leg ulcer recurrence can be reduced using higher class compression. Participants wearing British standard class 3 hosiery had an ulcer recurrence rate of 20.6%, compared with a rate of 36.4% in patients wearing British standard class 2 hosiery.

However, a recent study by Ashby et al (2014) found that there was no difference in healing outcomes between patients managed with hosiery and four-layer bandaging, concluding that two-layer hosiery was as effective in healing venous leg ulcers in suitable patients as compression bandaging.

One of the problems with much of the literature is the rate of non-concordance with compression therapy. This can make it difficult to accurately assess the benefit of any therapies used. However, Williams (2009) has identified the need for appropriate treatment for oedema in patients who have a leg ulcer or are at risk of developing one. As a method of preventing lymphovenous disease advancing to a point where ulceration is inevitable, compression therapy may be the most accessible option available to community nurses.

COMPRESSION THERAPY

Bianchi (2013) describes compression therapy as a central therapeutic option in the management of chronic oedema. She highlights the importance of early management and achieving a level of therapy that corresponds with the extent of the individual’s disease. Compression therapy improves chronic oedema by reducing the vessel size of both superficial and deep veins while supporting the calf muscle pump to exert additional pressure during muscle relaxation (Moffatt, 2007).

Further to improving venous circulation, by exerting external pressure, compression also encourages fluid uptake into the local lymphatic system. Coupled with a massaging effect on soft tissues, these multiple modes of action of compression also reduce limb swelling, improve venous and lymphatic circulation, and reduce the density of sclerotic tissues (Foldi et al, 2006).

In practice

Kolluri (2011) has described the need to have clear goals when determining the type of compression to be used. The primary treatment goal will often be to reduce limb volume, as this is a measurable outcome and something that has a direct patient benefit. Improvement in limb volume will also quickly indicate the degree of success of any prescribed treatment. However, the mechanism by which compression therapy is delivered may vary — similarly, any benefits of compression therapy can only be realised if patients are concordant with treatment. Compression can be delivered in a number of ways at a range of pressures and includes bandaging and hosiery.

Bandages

Compression bandages can use both elastic and inelastic materials and are available from a variety of manufacturers. Bandages are often used in the early stages of treatment, either when skin ulceration is present, or when large volumes of fluid reduction are required in a short period of time (Partsch et al, 2001).

Although both elastic and inelastic bandages result in compression, there are differences in the way they achieve it. Elastic bandages apply a constant pressure following application and the pressure is dictated, in part, by the extent to which the bandage is stretched — a standard pressure of 40mmHg can be achieved using a four-layer elastic bandage system.

Inelastic bandages apply a variable level of pressure as they exert the majority of their force when the calf muscle pump is engaged — this leads to pressures between 20mmHg at rest and 60mmHg when walking (MacGregor, 2013).

There is much debate about which type of bandage works best. Nelson et al (2004) completed a randomised controlled trial of both forms of compression bandage, using 387 patients who were randomised to either a long-stretch or short-stretch bandage system for treatment of venous leg ulceration. The study showed no statistical difference between ulcer rates in either study arm.

Meanwhile, an earlier study by Ukat et al (2003) demonstrated improved healing with elastic bandages. However, this contrasted with studies by Partsch et al (2001) and Scriven et al (1998), who demonstrated independently that ulcer healing was faster using inelastic bandages.

To date, there are no studies that convincingly demonstrate that either system is universally better than the other. The studies highlighted here, like most into the use of compression bandaging, are mainly concerned with leg ulcer healing. In practice, however, the choice of system is often dictated by the patient’s level of mobility and arterial competence, and whether he or she finds it acceptable.

Hosiery

The Lymphoedema Framework (2006) provides guidance on the use of hosiery as both a management
and maintenance treatment. It is recommended that under normal circumstances, bandages should be used to reduce oedema. However, using hosiery, though slower, may be the only way to achieve limb volume reduction in patients with poor concordance.

In recent years, the development of more variety in hosiery systems has helped to improve their acceptability to patients, both in terms of comfort and aesthetics. This means that compression hosiery can be a viable alternative to bandaging (Amsler et al, 2009; Ashby et al, 2014). Hosiery is also often much easier to apply, particularly for those patients wishing to self-care (Kolluri, 2011).

Hosiery provides compression within a narrow range of pressures predetermined in the manufacturing process. Different hosiery manufacturers and brands use a variety of different standards by which they classify the level of compression. Table 1 shows two of the most common used in the UK (British Standard and European Class), but there are many more available worldwide and this is not an exclusive list.

Hosiery is available in different colours, sizes, lengths and shapes and can often be customised to suit the individual patient. This means that hosiery prescriptions need to be considered thoroughly to ensure both the correct fit and correct pressures are achieved (Doherty et al, 2009).

**SURGICAL MANAGEMENT**

There are few surgical options with proven value in the management of lymphovenous disease and most are targeted at managing any primary pathology that might be affecting the venous system, such as a tumour obstructing the venous and lymphatic outflow (Lymphoedema Framework, 2006).

National clinical guidelines recommend treatment either by intravascular catheterisation or surgery for varicose veins (National Institute for Health and Care Excellence [NICE], 2013), or occlusion of veins and arteries by thrombus (blood clot) (NICE, 2012).

Surgery for a lymphatic obstruction is generally focused on the removal of tumours or the correction of external obstructions such as an inguinal hernia (Campisi et al, 2001). Surgery to improve lymphatic flow directly can take one of two forms, either ablative (where the vessels are destroyed, often by laser or foam injection), or reconstructive (where vessels are surgically repaired or new vessels grafted into place).

Microsurgical techniques are also available and aim to reconstruct lymphatic flow when damaged nodes or vessels are involved, or to bypass obstructions (Lee et al, 2011). In addition, autologous lymph node transplant surgery (transplanting lymph nodes to facilitate restoration of normal lymph transport in the affected limb) may prove beneficial in some cases (Tobbia et al, 2009).

Though surgical intervention may form part of a treatment plan for some individuals, further evaluation of its clinical effectiveness is required. Conventional management of lymphovenous disease is well established in the literature and is based around comprehensive health promotion, self-management and compression therapy (Williams, 2012).

**CONCLUSION**

Historically, lymphovenous disease has been an under-diagnosed condition due in part to the misdiagnosis of chronic oedema. Overlooking chronic oedema purely as a symptom of cardiac disease, renal disease or hypertension can mean that only a part of the underlying disease is managed. Poor diagnosis has meant that there is a lack of strong prevalence data. This, in turn, means that the extent of chronic oedema and, more specifically, the prevalence of lymphovenous disease, remain largely unknown.

However, there is a knowledge base around the pathology of both venous and lymphovenous disease and a range of therapies can be used to support disease management. It is the responsibility of the clinician to determine the underlying disease process and select appropriate management options in collaboration with the patient. Whether surgical or non-invasive management options such as compression are used, the therapy goals should be evaluated with the patient and monitored to ensure success. Where wounds are present or where treatment is used specifically with the prevention of ulceration in mind, strategies for improving patient concordance should be considered, such as intensive patient education, the use of hosiery application aids and kits, or in some cases simply offering a choice of hosiery colour and/or texture.

While the disease process is fairly well understood, further work to establish the cost and clinical effectiveness of different types of compression therapy would be beneficial. It would also be useful to understand the degree to which both surgery and compression can correct underlying pathology in the lymphovenous circulation. This would enable clinicians to better explain the pathology and support patients to make improved treatment choices.

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KEY POINTS

This article has highlighted the normal and abnormal physiology of fluid circulation in the lower limb with particular reference to venous and lymphatic flow.

Consideration must be given to the assessment and diagnosis of disease and its severity, particularly the impact on the patient including chronic oedema, ulceration, cellulitis and loss of mobility.

While the disease process is fairly well understood, further work to establish the cost and clinical effectiveness of different types of compression therapy would be beneficial.

Historically, lymphovenous disease has been an under-diagnosed condition due in part to the misdiagnosis of chronic oedema.

Options for disease management include compression bandaging, and compression hosiery.

It would also be useful to understand the degree to which both surgery and compression correct underlying pathology in the lymphovenous circulation.