An overview of lower limb lymphoedema and diabetes

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The prevalence for individuals diagnosed with lower limb lymphoedema and coexisting diabetes is unknown. However, both conditions cause significant problems that can compromise the viability of the lower limbs. An extensive search of the literature including medical databases (MEDLINE, PubMed and CINAHL), plus hand searching through diabetes journals, podiatry journals and wound care journals, was undertaken to search for published literature relating to diabetes and lymphoedema of the lower limb.

Key words

Lymphoedema
Lower limb
Diabetes
Foot

Lymphoedema is a chronic progressive condition for which there is no cure. Unless it is managed effectively, lymphoedema can gradually deteriorate and treatment can become increasingly difficult. Lymphoedema and chronic oedema are terms that are often interlinked.

Harwood and Mortimer (1995) define lymphoedema as: 'The accumulation of lymph in the interstitial spaces caused by a defect in the lymphatic system.' It is marked by an abnormal collection of excess tissue proteins, oedema, chronic inflammation and fibrosis (Harwood and Mortimer, 1995).

Chronic oedema describes oedema that has been present for more than three months (Moffatt et al, 2003).

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Prevalence

Lymphoedema/chronic oedema are significant causes of morbidity in the general population. An epidemiological study completed by Moffatt et al (2003) aimed to determine the magnitude of the problem and the likely impact on health resources, employment and patients' quality of life. The study, which was carried out in a primary care trust in south west London, identified a crude prevalence of chronic oedema of 1.33/1000. This increased to 5.4/1000 with age (>65 years) and was higher in women.

Risk factors

Many risk factors for lymphoedema have been identified, including non-accidental injury, such as venepuncture (Cole, 2006) and chronic health problems linked to obesity, such as diabetes, hypertension and cardiovascular disease (Soran et al, 2006).

Fife et al (2008) reviewed the current evidence base, including case studies in the absence of controlled trials, and found that there is increasing clinical evidence to suggest that morbidly obese patients are predisposed to secondary lymphoedema and that primary lymphoedema can induce adult-onset obesity. However, there is at present limited scientific evidence to determine the mechanisms by which these events take place, although it is known that obesity impedes lymphatic flow, leading to an accumulation of protein-rich lymphatic fluid in the subcutaneous tissues (Yosipovitch et al, 2007).

Obesity is increasingly being recognised as a major public health problem. The Department of Health (DoH) (2008) stated that, 'obesity is both a highly complex issue for society and a costly debilitating lifestyle disease'. Health survey information for England, undertaken in 2006, found that a quarter of the adult population in England are classified as obese, along with almost a fifth of all children under the age of 16 (DoH, 2008). Obesity is, therefore, one of the major public health issues in the developing world and is known to contribute to an increased risk of heart disease, some cancers and type 2 diabetes mellitus.

Diabetes mellitus

Diabetes is an escalating problem in the UK, which may contribute to an increased prevalence of lymphoedema in time, particularly when linked with obesity. There are currently an estimated 2.35 million people diagnosed with diabetes in England (DoH, 2007), while it is estimated that a further 800,000 individuals are living with undiagnosed diabetes (National Institute for Health and Clinical Excellence [NICE], 2004).

The prevalence of diabetes is predicted to rise to more than 2.5 million in England by 2010 (DoH, 2007). This rise is attributable to both

an ageing population and also an increasing prevalence of obesity, with the DoH (2007) figures suggesting that approximately 9% of the increased prevalence of type 2 diabetes will be a direct consequence of obesity. It could, therefore, be postulated that the incidence of individuals presenting with both lymphoedema and diabetes, particularly in obese individuals, will escalate over the next few decades.

An extensive search of the literature, including medical databases (MEDLINE, PubMed and CINAHL), plus a hand search of diabetes, podiatry and wound care journals, revealed that there are currently no published data detailing the prevalence of individuals diagnosed with both lymphoedema and diabetes. However, a combination of both pathologies in the lower limbs can compromise the viability of the legs and feet, placing the individual at high risk of infection, ulceration and necrosis, and, in severe cases, the loss of a limb.

This article aims to explore the current evidence-base for diabetes and lymphoedema and discuss the implications of both conditions on the lower extremities. It also considers the appropriate assessment and management strategies to aid nurses and podiatrists in clinical practice.

Implications of lymphoedema and diabetes for the lower limb

Lymphoedema in the diabetic foot is thought to be a combination form of lymphoedema with a complex pathophysiology – microangiopathy leads to increased permeability of the blood capillaries and an increased lymphatic load. The effects of diabetes can also affect the blood capillaries of the lymph nodes, resulting in a general immune deficiency. Lymphatic failure, due to inflammation following infection, insulin injections and ulceration, can also result (Földi and Földi, 2007).

It is important to identify patients with coexisting lymphoedema/chronic oedema and diabetes in order that treatment strategies can be instituted at an early stage to prevent long-term complications and deterioration.

Clinical features of lymphoedema that are particularly problematic in the lower limbs include:

- Pitting oedema in the initial stages, progressing to non-pitting tissues as the condition progresses
- Skin changes, including hyperkeratosis, papillomatosis, lymphangiomata, fibrosis (*Table 1*)
- Stemmer's sign
- Skin folds
- Distorted/misshapen limb
- Recurrent cellulitis.

Skin changes in the lower limb

Skin changes secondary to diabetes mellitus are also common, with published data suggesting that as many as 30% of all patients with diabetes will present with skin changes during the course of their disease (Ahmed and Goldstein, 2006) (*Table 2*).

The impact of obesity (a common precursor to both lymphoedema and diabetes) on the skin has received minimal attention to date, despite the fact that obesity is also associated with a number of dermatoses, including *acanthosis nigricans*, *keratosis pilaris*, hyperkeratosis and skin striae.

Furthermore, obesity can heighten the risk of skin breakdown in those with poor tissue viability, and complicate wound management, particularly for those who are morbidly obese (Fife et al, 2008). In obese patients with lymphoedema, the accumulation of fluid in the lower limbs can lead to fibrosis of the skin, decreased oxygen tension and macrophage function, which provides a culture medium for bacterial growth (Yosipovitch et al, 2007). Diabetes is also known to predispose individuals to infection, as the effects of an underlying vascular disease on the immune system can result in hyperglycaemia and tissue hypoxia (Falanga, 2005). Therefore, individuals with co-existing lymphoedema and diabetes are at heightened risk of infection of the lower limbs.

Bacterial infection

Erysipelas, a skin infection caused by beta-haemolytic group A streptococci, has been linked to both diabetes and sub-clinical primary lymphoedema — obesity with co-existent lymphoedema has also proven to be an independent risk factor for erysipelas (Yosipovitch et al, 2007).

Damstra et al (2008) undertook a small (n=40) study in which lymphoscintigraphy of both legs was performed in patients four months after their first acute event of erysipelas. Findings from this small sample demonstrated that 79% of the patients experienced sub-clinical lymphatic dysfunction of both legs, suggesting that lymphatic impairment may be a predisposing factor in erysipelas. However, while diabetes is a known risk factor for erysipelas infection, none of the participants in the study had previously had a positive diagnosis for diabetes, thus limiting the external validity of the study.

Fungal infection

Obesity is thought to increase the risk of cutaneous fungal infections, such as candidiasis. Individuals with diabetes are also known to be at an elevated risk of fungal skin infection, as hyperglycaemia has a detrimental effect on the immune system. The skin of individuals with lymphoedema and diabetes should be regularly assessed for fungal infection, particularly in skin folds or between digits, where fungi will thrive in the warm, moist environment.

Furthermore, mycologic tests for the presence of fungal species may prove beneficial in those individuals with ulceration on the legs or feet and coexisting diabetes and lymphoedema, as fungal infection can be detrimental to wound healing if left untreated (Missoni et al, 2006).

Specific manifestations of diabetes on the lower limb

Patients who have lymphoedema, or are at risk of lymphoedema, also need to be educated on the effect other conditions and treatments may have on their condition. Diabetes mellitus can have a profound impact on the lower limb – chronic hyperglycaemia can contribute to the development of peripheral polyneuropathy, peripheral arterial disease, reduced tissue viability and immunosuppression.

Peripheral polyneuropathy

Sensory loss is a major cause of diabetic foot ulceration. Frykberg et al (2006) reported that as many as 45–60% of all diabetic foot ulcers are neuropathic in origin. In the insensate foot the individual is unable to feel pain and is, therefore, unaware of trauma or repetitive stress, e.g. from ill-fitting footwear, which can contribute to the pathogenesis of foot ulceration.

Although the precise mechanisms underlying diabetic neuropathy remain unclear, there is increasing evidence that the hyperglycaemia-induced formation of advanced glycation end products (AGEs) are related to peripheral nerve demyelination, axonal atrophy and impaired regenerative activity, resulting in diabetic neuropathy (Sugimoto et al, 2008). AGEs are sugar-derived substances that are known to increase the risk of tissue damage, and are thought to play a significant role in the pathogenesis of the chronic complications of diabetes.

Table 1

Common skin changes secondary to lymphoedema

Hyperkeratosis	Over proliferation of the keratin layer of the skin produces scaly, brown patches on the skin
Papillomatosis	Firm, raised projections on the skin. This is due to dilation of lymphatic vessels and fibrosis — sometimes accompanied by hyperkeratosis
Lymphangiectasia	Also known as 'lymphangiomata' — soft, fluid filled blister-like projections on the skin caused by dilation of lymphatic vessels
Stemmer's sign	Inability to pick up a fold of skin at the base of the second digit
Lymphorrhoea	Leakage of lymph from the skin surface (*Figure 1*)

Table 2

Common skin changes secondary to diabetes

Necrobiosis lipoidica diabeticorum	A collagen disorder characterised by degeneration with a granulomatous response
Granuloma annulare	A benign, asymptomatic self-limited eruption that classically presents as groups of round firm, skin-coloured papules frequently occurring on the lateral or dorsal surfaces of the feet (Cyr, 2006)
Acanthosis nigricans	Characterised by a dark, warty, hyperpigmentated thickening of the skin
Diabetic dermopathy	Characterised by small brown lesions on the shins of some patient's with diabetes which is thought to be a consequence of diabetic microangiopathy, sometimes termed 'spotted leg'
Lipoatrophy	Partial or generalised thinning of the fatty-cutaneous layer of the skin

Foot deformities

Foot deformities are known to be causal factors in the pathogenesis of diabetic foot ulceration (International Diabetes Federation, 2005). Structural changes are common in the diabetic foot, mainly due to peripheral motor neuropathy. Digital deformities, such as clawed toes, prominent metatarsal heads, *pes cavus* (high arch profile) and muscular atrophy are commonly observed in the diabetic foot. Furthermore, limited joint mobility, including restricted movement at the ankle, is a common problem and can have an impact on the individual's gait, increasing the risk of foot ulceration (McIntosh, 2007). This may be exacerbated in those with concomitant lower limb lymphoedema, where the range of motion at the ankle is restricted due to chronic oedema.

Peripheral arterial occlusive disease (PAOD)

There is a strong association between diabetes mellitus and peripheral arterial occlusive disease (PAOD), with macro- and micro-circulatory impairment being a common clinical finding. Indeed, reports have suggested that the risk of PAOD increases 20-fold in people with diabetes compared with the non-diabetic population (Shaw and Boulton, 2001). Macrovascular disease, in diabetic patients is due to the same atherosclerotic changes observed in the non-diabetic population; however, PAOD tends to occur at a younger age and is more aggressive (Dinh and Veves, 2005). Microvascular changes are thought to be primarily due to basement membrane thickening at the capillary level and endothelial and smooth muscle dysfunction (Dinh and Veves, 2005).

Peripheral arterial disease (ischaemia) is rarely a causative factor for diabetic food ulcers, but rather an underlying aetiology that impedes wound healing (Frykberg et al, 2006). However, a significant proportion of diabetic foot ulcers, approximately 30–50%, are complicated by the presence of lower limb ischaemia (Frykberg et al, 2006). Furthermore, the majority of lower extremity amputations in the diabetic population occur due to dysvascularity (Apelqvist et al, 1992).

Chronic venous insufficiency

While it is widely accepted that diabetes is associated with haemorheological disturbances that can alter arterial flow resulting in chronic tissue hypoxia, it is unclear whether these haemorheological disturbances are confined to the arteries (which can increase the risk of cardiovascular incidents and peripheral arterial disease), or whether the haemostatic impact of diabetes can also result in venous dysfunction. One retrospective population-based study, with a limited sample size (n=302), found that the age-adjusted risk for venous thromboembolism was more than two-fold higher among patients with diabetes when compared to a non-diabetic population (Petrauskiene et al, 2005). Obesity is also a recognised risk factor for the development of chronic venous insufficiency and venous thromboembolism (Yosipovitch et al, 2007). Lipodermatosclerosis and venous ulcerations may complicate venous insufficiency of the lower limbs and, for those with lymphoedema and diabetes, might lead to compromised healing and a heightened risk of infection.

Assessment strategies for the lower limb

Individuals with lower limb lymphoedema, as well as those with diabetes, require regular, comprehensive, lower limb assessments to yield baseline measures and to monitor for the presence of skin conditions that might predispose to infection and/or ulceration, peripheral vascular disease and any neurological deficit.

Vascular assessment

Vascular assessment of the lower limb is essential to quantify lower limb perfusion in patients presenting with lower limb lymphoedema and diabetes. NICE (2004) guidelines advocate regular vascular screening for all individuals with diabetes. This concurs with best practice guidelines for the management of lymphoedema (Lymphoedema Framework, 2006) in which arterial assessment, including Doppler examination and measurement of the ankle:brachial pressure index (ABPI), is advised for all patients with lower limb lymphoedema. It is imperative that arterial perfusion is quantified in individuals with lower limb lymphoedema and diabetes, as the presence of peripheral arterial disease may contraindicate compression therapy, and increase the risk of foot or leg ulceration, or in the case of established ulceration, contribute to delayed healing. However, a survey of 250 delegates at a British Lymphology Conference found no consensus in the method of vascular assessment of lymphoedema patients across practitioners, suggesting that guidelines based on best evidence are warranted (Todd et al, 2008).

Vascular assessment should include a comprehensive medical history in addition to vascular testing. It is important to establish control of diabetes control and other arterial risk factors including hypertension and dyslipidaemia. The presence of vascular symptoms such as arterial pain should also be explored, for example, intermittent claudication. The Edinburgh Claudication Questionnaire (ECQ) (Leng and Fowkes, 1992) is touted as a validated questionnaire to diagnose intermittent claudication in clinical practice. However, practitioners should be aware of its limitations. One large observational study (n=4527) set in general practice in the Netherlands, found that the ECQ alone has an inadequate diagnostic value in general practice in patients with symptoms suggestive of intermittent claudication, with the sensitivity of the ECQ found to be 56.2% (Bendermacher et al, 2006). The ECQ should, therefore, not be used as a single method to diagnose intermittent claudication and other tests, such as the ankle: brachial pressure index (ABPI), should be performed to diagnose peripheral arterial disease (Bendermacher et al, 2006). Epidemiologic cross-sectional studies show that in the general population asymptomatic PAOD is more common than symptomatic PAOD, with less than half of all individuals with PAOD having symptoms of intermittent claudication (Hooi et al, 2001; Bendermacher et al, 2006).

A large cross-sectional survey of 3650 participants in 18 general practices across the Netherlands explored risk factors for asymptomatic PAOD (Hooi et al, 1998). It found that 8.6% of participants had asymptomatic PAOD, while 3.8% had symptomatic PAOD, which concurred with later published work (Hooi et al, 2001) and suggested that asymptomatic PAOD is more commonly observed than symptomatic PAOD in clinical practice. Risk factors that were reported to be significant for asymptomatic PAOD included younger age categories, smoking status, male gender, hypertension and diabetes. Therefore, individuals with diabetes may remain asymptomatic despite the presence of PAOD. Whether diabetic somatic polyneuropathy contributes to asymptomatic PAOD is unclear, however, practitioners must be aware that patients may not experience arterial pain. They must also be able to differentiate between arterial pain and painful sensory diabetic neuropathy in those who are symptomatic, with the latter commonly causing parasthesias, allodynia, burning and lacinating pain in the lower limbs.

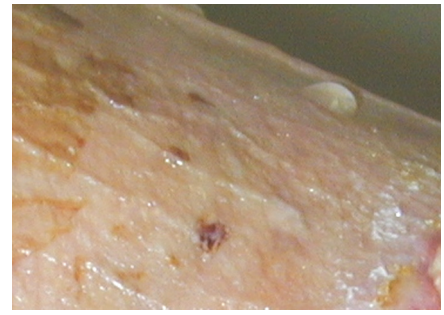

Figure 1: Lymphorrhoea.

Current diabetes guidelines suggest that basic vascular examination should include palpation of foot pulses (dorsalis pedis and posterior tibial pulses), testing capillary return time and temperature gradient at annual review (NICE, 2004; International Diabetes Federation, 2005). Pulse palpation may prove difficult in the presence of oedema and, therefore, is inappropriate as an assessment strategy for individuals with lymphoedema or chronic oedema. Doppler examination is particularly useful for locating non-palpable pulses, providing audible signals of the strength, phasic nature and pitch of the signal, as well as doppler waveforms to aid the practitioner in the interpretation of vascular status.

The Lymphoedema Framework (2006) advocates the ABPI as an objective measure of the patency of the arteries supplying perfusion to the foot. Essentially, this test involves measuring systolic pressure in the upper and lower limbs to arrive at a ratio. The ratio is obtained by dividing the highest ankle pressure by the highest brachial pressure for each limb separately (Marshall, 2004). There are, however, limitations to this test due to lymphoedema and diabetes (*Table 3*).

While there is minimal evidence to support its use, the Pole test has been advocated as a useful, non-invasive, alternative method to the ABPI for diagnosing peripheral arterial disease, particularly in individuals with diabetes (Lazarides and Giannoukas, 2007).

Other alternative assessment strategies to the ABPI include toe brachial pressure indices (TBPI) (*Figure 2*) and transcutaneous oxygen measurement (TcPO2) (*Figure 3*). TcPO2 provides a physiologic measure of tissue oxygenation that has been found to be highly predictive of wound healing failure at levels below 25mmHg (Frykberg, et al 2006). However, transcutaneous oxygen measurements may be falsely low in the presence of infection, as this impairs oxygen diffusion in the neuroischaemic foot. After the infection has resolved, values may be seen to rise even without vascular intervention (Edmonds, 2005).

Venous assessment

Venous photoplethysmography (VPPG) is a simple, non-invasive test that can be used to assess venous competency in the lower limb by measuring venous refill times (*Figure 4*).

VPPG investigation is useful to identify patients with chronic venous insufficiency and is recommended particularly for those presenting with the following symptoms:

- Post-thrombotic syndrome
- Swelling
- Varicose veins
- Varicose eczema
- Venous oedema
- Venous ulcers.

Interpretation of results:

- If refill time is >25 seconds, venous insufficiency is **not** present/significant
- If refill time is <20 seconds, venous reflux is present. A tourniquet cuff can then be applied at appropriate positions to determine the level of venous insufficiency
- If refill time is <10 seconds, deep venous insufficiency or obstruction is indicated.

Neurological assessment

The international consensus on the diabetic foot practical guidelines (International Working Group on the Diabetic Foot, 1999) advocate testing for neuropathy with a 10g monofilament and 128Hz tuning fork. The use of the 10g monofilament, to detect light touch, is a reliable method to discriminate between patients who are at increased risk of developing foot problems from those in lower risk categories (Baker et al, 2005). Vibration perception testing (VPT) is a further useful measure to identify diabetic peripheral neuropathy and predict long-term complications of diabetes, such as foot ulceration (Baker et al, 2005). VPT can be undertaken with a 128Hz tuning fork. Combining the two modalities, VPT and monofilament, is reported to increase specificity (Armstrong et al, 1998). However, in the presence of hyperkeratosis (abnormal thickening of the skin), sensation can be diminished and, thus, the ability to perceive the monofilament can be reduced.

Management strategies

Management of both lymphoedema and diabetes starts with an attempt to control the underlying disorder.

Recommended management of lymphoedema

The Lymphoedema Framework Project and British Lymphology Society (BLS) and Lymphoedema Support Network (LSN) (2007) are working to raise awareness of the condition and promote best practice for the management of this chronic, incurable condition.

Management is based on four main components:

- Skin care and preventative measures
- Exercise, movement and positioning
- Lymphatic drainage
- External compression and support (hosiery/multilayered bandages).

Treatment is based on a programme of physical therapy and there are a number of issues that need to be addressed for each patient. These include; reduction of the swelling, prevention of skin changes, prevention of infection and treatment of specific problems, e.g. lymphorrhoea, cellulitis, lymphangiomata and papillomatosis (Gordon and Mortimer, 2007). A two-phase approach to treatment is recommended: an intensive treatment phase aimed at reducing limb volume and improving limb shape and skin

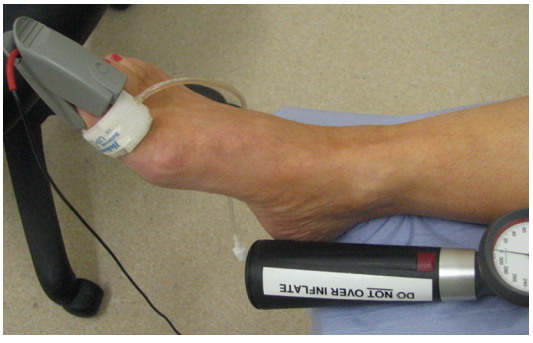

Figure 2. Toe pressure measurement.

Figures 3a and b. Transcutaneous oxygen measurement.

Table 3

Limitations of the ABPI due to lower limb lymphoedema and diabetes

Lymphoedema	Diabetes
▸▸ Tissue thickening, oedema or hyperkeratosis can make it difficult to find pedal pulses with a 8 MHz Doppler probe; the use of a 4 or 5 MHz probe may be more appropriate in these circumstances (Lymphoedema Framework, 2006)	▸▸ Calcification of the arterial wall occurs in approximately one third of patients with diabetes causing the vessel to become resistant to compression. Therefore, the ratio obtained is often abnormally high (>1.3), as it is the stiffness of the vessel wall that is measured not blood pressure (Marshall, 2004), which can lead to inaccurate results

conditions, followed by a maintenance phase where the patient is shown self-management techniques to control the swelling and prevent further complications (Földi, 1994).

Skin care

Skin care is essential in the management of lymphoedema. The general principles of skin care aim to preserve skin barrier function through washing and the use of emollients (Lymphoedema Framework Project, 2006). In patients with lymphoedema and diabetes this is especially important as the risks associated with skin damage are increased. Regimens including daily inspection of the legs and feet, and appropriate skin care should be taught to the patient. Cellulitis is a significant risk for patients with lymphoedema, which is magnified in those with concomitant diabetes. Hyperglycaemia (elevated blood glucose levels), associated with diabetes, is known to impair neutrophil function and subsequently suppress the immune system, increasing infection risk in patients with diabetes (Edmonds and Foster, 2006). Infection plays a major role in delayed healing of wounds, hospitalisation and the incidence of lower extremity amputation. Prompt recognition and management of infection in the diabetic foot is imperative. If infection is left undetected or treatment is delayed, diabetic foot ulcers can become limb- and life-threatening (Sheppard, 2005). The cause of most episodes of cellulitis in patients with lymphoedema is believed to be Group A b-haemolytic streptococci or other bacteria (Lymphoedema Framework, 2006). Symptoms may vary from patient to patient and may come on over minutes or weeks. Symptoms may include:

- Redness, inflammation or rash on the skin
- Pain
- Increased swelling
- Fever, chills, rigor.

Predisposing factors may include *tinea pedis* (athlete's foot), venous eczema, scratches or insect bites. The BLS and LSN (2007) have devised guidelines on the management of cellulitis/erysipelas in lymphoedema. These guidelines suggest management strategies, including indications for antibiotic therapy for acute and recurrent infections in patients with lymphoedema.

Patients should be advised to protect their skin when carrying out certain activities such as gardening, walking around barefoot (especially at swimming pools/changing rooms), and to take extra care when cutting toe nails. This is of paramount importance in the presence of any neurological or arterial deficit (Linnitt, 2000; Mortimer and Todd, 2007). Diabetic patients should be made aware of the risk of infection through blood sugar analysis (finger pricking) and injecting insulin due to puncture of the skin. It is important that patients with lymphoedema and diabetes are assessed and closely monitored for foot problems. In the authors' opinion, all individuals with lower limb lymphoedema and/or diabetes who present with skin pathologies on the feet (e.g. callus or corns), or toe nail pathologies should be referred to a Health Professions Council registered chiropodist/podiatrist for assessment, advice and treatment.

Exercise and movement

Exercise, movement and limb positioning are essential in the management of lymphoedema. The main aim is to maintain joint mobility, especially at the ankle joint, which will enhance lymphatic and venous flow through calf muscle contraction. Godoy and Godoy (2001) suggested that passive movement of the foot can cause significant volume change in the lower limbs. Foot and ankle exercises should be encouraged to promote calf pump action, and thigh and hip exercises can help with fluid movement from the thigh (Green, 2007). Patients with diabetes-related complications may not be able to carry out vigorous exercise due to claudication symptoms, but they may be able to swim or carry out gentle stretching exercises. Patients should be encouraged to exercise within their limitations. Swimming in itself will need to be carefully monitored due to risks of infection from dry skin, and the risks of foot infection from communal changing rooms. Wearing protective footwear when walking to and from the pool will minimise risks.

Lymphatic drainage

Lymphatic drainage is designed to stimulate lymph flow from areas of congestion to areas with functioning lymphatics. It is a specific, gentle, rhythmic massage that is designed to work on the superficial lymphatics in the skin and encourage the interstitional fluid to be reabsorbed by the initial lymphatic vessels. The aim is to build collateral drainage routes and alternate drainage pathways, helping to relieve congestion in the limb (Leduc and Leduc, 2000).

External compression and support comprises multilayer

inelastic lymphoedema bandaging and compression hosiery. Multilayer lymphoedema bandaging (MLLB) is a key element of intensive lymphoedema management. MLLB uses inelastic bandages that produce high working and low resting pressures. Thus, they produce a massaging effect and stimulate lymph flow (Lymphoedema Framework, 2006). *Table 4* summarises the indications and contraindications for the use of MLLB.

Assessment

Careful and thorough assessment of the patient is required before instigation of compression therapy as detailed in the algorithm (*Figure 5*), and illustrated in the case report presented. Any patient presenting with a diabetic foot ulcer and lymphoedema should be referred urgently to the specialist foot care team in line with NICE (2004) guidelines. Effective communication between the diabetes and lymphoedema teams is essential to improve patient care. For those without foot ulceration, the shape of the limb, extent of swelling, skin and tissue condition must all be considered when deciding on the type of compression to apply to a lymphoedematous limb. Vascular assessment, including Doppler assessment and measurement of the ABPI must be carried out to ascertain if the patient is suitable for compression therapy. The Lymphoedema Framework (2006) has identified an algorithm for the long-term management of lower limb lymphoedema. Following ABPI

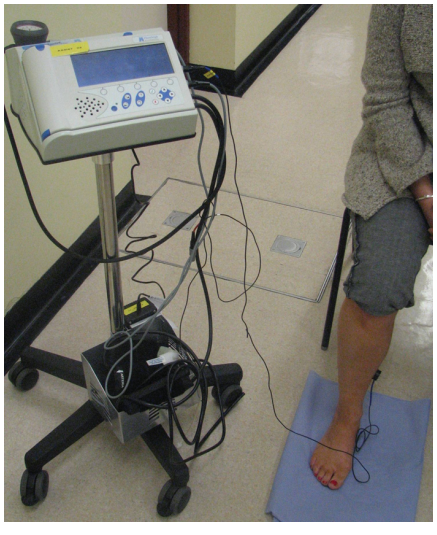

Figure 4. Venous refill testing.

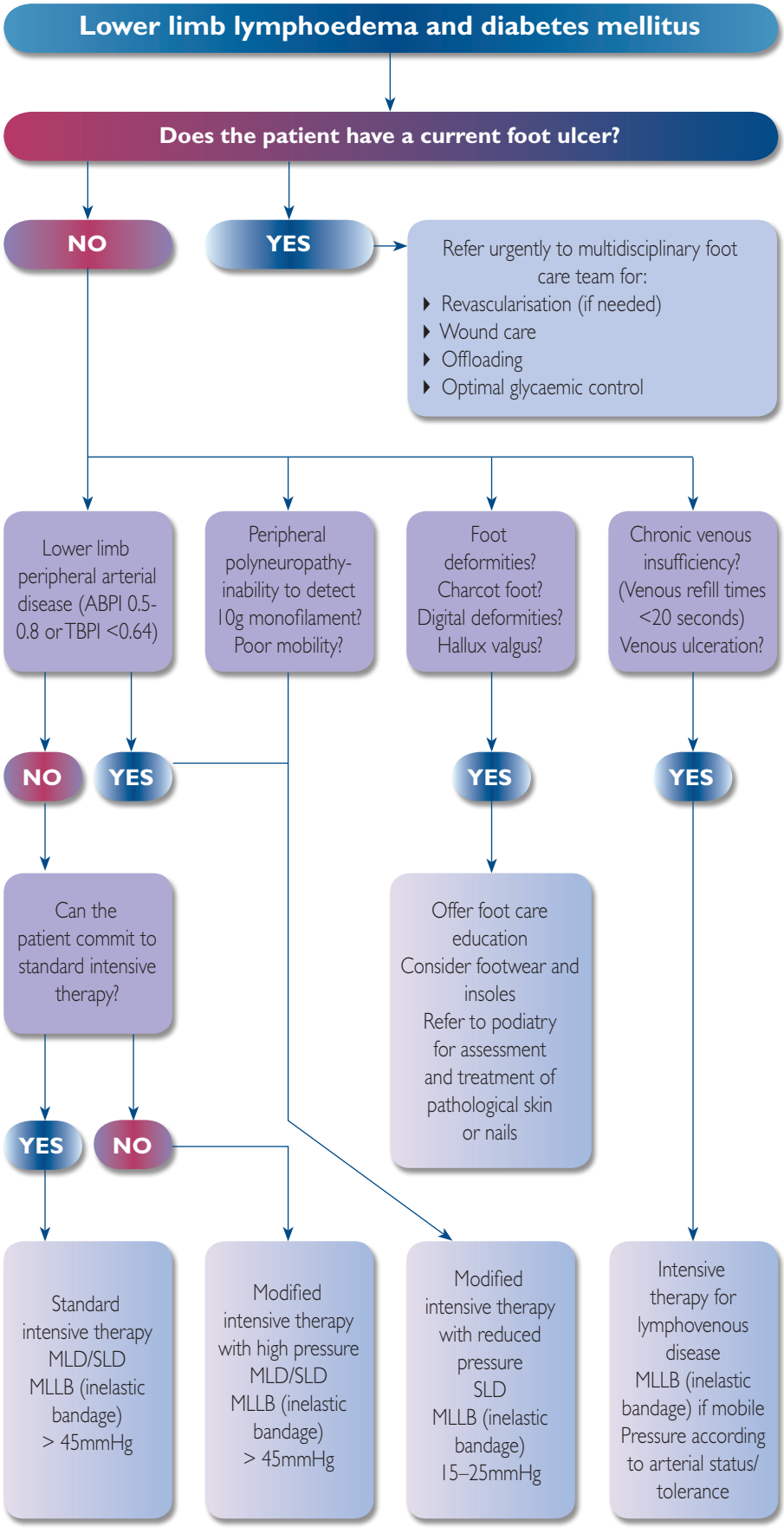

Figure 5. Management strategies for individuals with lower limb lymphoedema and diabetes (adapted from NICE, 2004; Lymphoedema Framework, 2006).

assessment (ABPI) or doppler ABPI, the flowchart guides the practitioner through a suitable regime of compression for the patient. Recording the ABPI is essential to ensure that patients with lower limb peripheral arterial occlusive disease are identified. The reliability of the readings need to be considered, particularly in patients with a lymphoedematous limb and diabetes. This, however, should always be used in conjunction with other assessment methods, bearing in mind that patients with diabetes may have calcified arteries and, along with oedema, there is scope for the assessment to provide a false high or false 'normal' reading. It may be necessary to apply reduced compression to patients with diabetes.

When applying compression bandages for the management of lymphoedema it is important to follow the law of Laplace. This indicates the pressure profile of a bandage and the sub-bandage pressure achieved through the correct tension and bandage overlap (Thomas, 2003). Poor fitting or badly applied garments/bandages can lead to skin damage. In patients with diabetes this could have severe consequences. Patients may develop allergies to components of hosiery and it is important to regularly monitor the patient's skin for any deterioration. It may also be necessary to alter the compression garment or apply a stocking liner for protection. Care must be taken to avoid any creases in either garment, as this could potentially cause skin damage and ulceration.

Patients must be given strict instructions on what to look for and what actions to take in case of problems with compression therapy. They should be made aware of the risks associated with the application and wearing of the bandages or compression garments. All wrinkles must be removed from the stocking and they must not be turned over at the top, as this can lead to a tourniquet effect restricting the flow of lymph and circulation, leading to pressure damage. Patients with diabetes are particularly vulnerable to pressure damage to the foot, due to poor tissue viability as a consequence of ischaemia and an insensate foot due to neuropathy. The Lymphoedema Support Network (LSN, 2006) have produced a patient leaflet, *The use of compression hosiery in the management of lymphoedema.* This written information supports the verbal instructions given to patients.

Table 4

Indications and contraindications for the use of multilayer lymphoedema bandaging (MLLB)

Indications	Contraindications
Fragile damaged or ulcerated skin	Severe arterial insufficiency
Distorted limb shape	Uncontrolled heart failure
For limbs which are too large to fit into compression garments	Severe peripheral neuropathy
Areas of tissue thickening or fibrosis	
Lymphorrhoea	
Pronounced skinfolds	

Footwear

Ill-fitting or inappropriate footwear is an established risk factor for diabetic foot ulceration (International Working Party on the Diabetic Foot, 1999). It is important to assess footwear at each consultation and offer advice on appropriate footwear for each patient. Finding appropriate footwear can prove difficult for individuals with lower limb lymphoedema, which may or may not need to accommodate bandaging. Therapeutic footwear has a beneficial effect in the primary and secondary prevention of diabetic foot ulceration (Maciejewski et al, 2004), therefore, referral to an orthotist for footwear advice or fitting should be considered.

Case report

Mrs A is a 61-year-old retired child minder. She developed lymphoedema in both legs following surgery and radiotherapy for carcinoma of the cervix in 2004. She is hypertensive and was diagnosed with type 2 diabetes in 2007. She is obese with a body mass index (BMI) of 58.

Bilateral leg swelling was present extending from the toes to involve the thigh and buttocks. Stemmer's sign was positive and there were skin folds at the base of her toes and ankles. Limb volume measurements, using circumferential limb measurements, showed that there was 11.6 litres of fluid in her right leg and 13.1 litres in her left, with an excess limb volume of 13.5% compared to the right leg.

Mrs A's skin was dry and patchy with small areas of hyperkeratosis around the ankles and lower leg. Tissues were pitting, although thickened, and she had poor ankle movement. She denied any intermittent claudication or peripheral neuropathy and doppler ABPI readings were reduced at 0.84 and 0.85 (from 1.06 and 1.04). As the tissues were thickened it was difficult to ascertain if this was an accurate reading and, in light of her medical history, a vascular review was requested. The vascular consultant felt that she should undergo a magnetic resonance (MR) arteriogram to examine her arterial supply. MR angiography is said to be a reliable method for investigating peripheral artery disease in selected patients with diabetes with critical limb ischaemia (Kreitner et al, 2000). MR angiography visualises lower extremity vessels that are not seen on conventional angiography (Lapeyre et al, 2005). This was reported as showing no significant peripheral vascular disease and she was thus safe to proceed with compression therapy to manage her lymphoedema.

In view of her history and symptoms of hypersensitivity to the lower left leg, it was agreed that her treatment would start with gentle compression and modified intensive therapy, with reduced compression incorporating skin care, exercise, and cohesive short-stretch bandages (Moffatt et al, 2005). Mrs A carried out simple lymphatic drainage on a daily basis to aid drainage from the buttock and thigh areas. For the first two days Mrs A was unable to tolerate the bandages overnight so the padding and bandaging technique was reviewed. The bandages were changed in terms of padding and reducing the pressure applied, by reducing the number of layers. Mrs A was then able to tolerate the bandages for the next two weeks.

Limb volumes reduced by 500mls in the left leg and 300mls in the right, more importantly she was able to flex and bend her knee and she felt that the heaviness from her legs had lessened. She is now wearing class 1 (18-23mmHg) thigh with waist attachment stockings on both legs and is tolerating the pressures well. She continues to be followed up at regular intervals, when her diabetic and arterial status is reviewed.

Multidisciplinary team approach

Individuals living with lower limb lymphoedema and diabetes require the support of numerous healthcare professionals for the management of acute problems. The need for a multiprofessional team approach in diabetes is outlined within NICE (2004) guidelines. Similarly, the Lymphoedema Framework (2006) outlines, in standard 6, the need for the provision of multi-agency health and social care. These guidelines offer best practice guidance to practitioners. By following these principles, people with lower limb lymphoedema and diabetes should receive integrated evidence-based health care from a range of health care providers to achieve optimal individualised care.

Conclusion

Lymphoedema and diabetes, as separate disease entities, can significantly compromise the viability of the lower limb due to dermatological change, increased risk of infection, arterial and neurological deficit. When these two conditions are in co-existence, the risk of such chronic complications can be magnified, predisposing the individual to recurrent infection, ulceration and potential amputation.

There is currently limited published literature that considers the impact of both conditions on the lower limbs, and the prevalence for those with both conditions is unknown. Careful assessment, particularly of vascular and neurological status, must be undertaken before developing and implementing care management plans, especially if this involves compression therapy. A multidisciplinary team approach is essential in the management of patients with lower limb lymphoedema and diabetes in order to provide regular assessments, comprehensive care to preserve the integrity of the limb and prevent adverse outcomes. JL

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Key points

- Diabetes is an escalating problem which may contribute to an increased prevalence of lymphoedema, particularly when linked to obesity.
- There is currently no published data detailing the prevalence of individuals diagnosed with lymphoedema and diabetes.
- A combination of lymphoedema and diabetes can compromise the viability of the lower limbs.
- It is important to identify patients with co-existing lymphoedema and diabetes in order that treatment strategies can be instigated at an early stage.
- Patients with co-existing diabetes and lymphoedema are at a heightened risk of infection and ulceration of the lower limbs.