Compression Therapy

Kim Kaim, RN BNursing(Dtn) MWoundC
kaim04@gmail.com
www.woundcareresource.com

Peripheral Oedema – what it is and what causes it?
  Hydrostatic
  Oncotic
  Reduced lymph return
  Acute vs Chronic

Why do we need to address oedema?
  Healing
  Trophic changes
  Growing health concern

What is compression therapy and how does it manage oedema?
  The science of compression

Assessment

Ulcer types and compression
  Venous leg ulcer
  Lymphoedema (trophic skin changes)
  Arterial ulcer
  Diabetic foot ulcer

When would I NOT use compression therapy?
  Not Competent
  Arterial Disease
  Use with caution...
    Mixed venous and arterial disease
    Heart Disease
    The Diabetic Limb

Guidelines

References

Updated July 2021
Peripheral Oedema – what it is and what causes it?
Oedema occurs when interstitial fluid production exceeds filtration. Excess fluid accumulating in the lower legs can be related to increased hydrostatic pressure, decreased oncotic gradient and/or reduced filtration related to the functioning of the lymph system. The presence of oedema can be acute or chronic.

Hydrostatic
This refers to a fluid pressure within the capillaries. Capillary hydrostatic pressure tends to be higher than tissue hydrostatic pressure, which encouraged greater movement of fluid into the tissues. Higher pressures encourage more permeability: the vessels are described as becoming “leaky”. Higher pressures are most often related to venous hypertension, varicose veins, or previous DVT (4), but can also be related to venous outflow obstructions, body position (gravity)(5), obesity and reduced mobility(6). Co-morbidities that impact on overall fluid quantities in the body such as heart failure(7) can also result in increased hydrostatic pressures in the lower legs.

Oncotic
Oncotic pressure refers to the “pull” of proteins in circulation(8). When there is an imbalance in the concentration of proteins in the circulation vs that in the tissues, fluid will shift across the capillaries to attempt to balance the concentrations on both sides (proteins are generally too big to move across these membranes so the fluid moves instead). Therefore, the generally higher concentrations of proteins in the blood encourage the movement of fluid back into the capillaries. A lack of these proteins in circulation stops this “pull” and more fluid moves into the tissues, encouraging oedema, such as in the case of liver failure.

Reduced lymph return
There is a general net flow of fluids from the circulation into the tissues. To manage the excess fluid the body has a lymphatic system. This is a network of highly permeable vessels with one-way valves that collect the fluid and return it to the circulation. Again, this is mostly driven through hydrostatic pressures(9).

Summarizing: the hydrostatic pressure tends to encourage fluid to travel out of the capillaries, the oncotic pressure tends to encourage return of fluid into the capillaries and the lymph system cleans up and returns the excess(8). When there is an imbalance resulting in more fluid leaving the capillaries than what can be returned, we have oedema. Physical positioning, comorbidities that impact fluid balance, medications that encourage peripheral oedema as well as physical activity and genetics all play a role in this balance.
Acute vs Chronic
The skin is very good at adapting to the needs of the body it contains. Skin can stretch significantly and quickly if needed. Oedema can appear rapidly, such as when there is a deep vein thrombosis, or after trauma or surgery.

Why do we need to address oedema?

Healing
It has been recognized and supported through research that the majority of lower leg ulcers respond positively to compression\textsuperscript{10}. This is demonstrated in leg ulcer and lymphoedema guidelines around the world. We know that oedema impacts on tissue changes, inflammation and perfusion, impacting on skin health and repair\textsuperscript{11}.

Trophic changes
Chronic, impaired venous return leads to leakage of proteins into the extra-cellular spaces, inflammation, and ultimately, local skin changes. These changes include ankle flair, hyperpigmentation, lipodermatosclerosis, atrope blanche and venous eczema. Studies are varied as to the prevalence of venous leg ulcers but the general consensus is that around 1% of any given population will have, or has had, a venous leg ulcer (Australia\textsuperscript{12}, UK\textsuperscript{13}, USA\textsuperscript{14}). These skin changes and ulcerations have their own medical, financial and social implications.

\begin{center}
\begin{tabular}{|c|c|c|}
\hline
| Ankle flair | Hyperpigmentation | Venous eczema and Lipodermatosclerosis |
\hline
\end{tabular}
\end{center}

Photos courtesy of DermNetNZ.org

The use of compression therapy supports venous return and reduces the impact on the skin. The compression of the tissues reduces oedema by opposing leakage of fluid from capillaries into tissues and by encouraging lymphatic drainage. It is also believed to increase the speed of venous blood flow, which may reduce local inflammatory effects\textsuperscript{11, 15}.

Growing health concern
As we live longer the number of comorbidities and the medications that go along with that longevity also increases. Many of these contribute to oedema. Obesity also contributes to oedema and this, too, is on the increase\textsuperscript{6}. Chronic venous hypertension itself is degenerative and rates of deterioration have been measured by several studies, but the key here is that is does get worse over time\textsuperscript{16}. It is also under-recognised. Therefore, quality of life is generally worse and costs to treat are generally higher by the time the person

<table>
<thead>
<tr>
<th>CEAP classification of chronic venous disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>C0 No visible or palpable signs of venous disease</td>
</tr>
<tr>
<td>C1 Telangiectasies or reticular veins</td>
</tr>
<tr>
<td>C2 Varicose veins</td>
</tr>
<tr>
<td>C3 Edema</td>
</tr>
<tr>
<td>C4a Pigmentation or eczema</td>
</tr>
<tr>
<td>C4b Lipodermatosclerosis or atrophie blanche</td>
</tr>
<tr>
<td>C5 Healed venous ulcer</td>
</tr>
<tr>
<td>C6 Active venous ulcer</td>
</tr>
</tbody>
</table>

Produced by Kim Kaim, 2021
seeks help. Increased strain on healthcare resources means that options for managing the condition need to be assessed versus their evidence-based efficacy as well as cost and applicability to the individual patient\(^{(16)}\).

**What is compression therapy and how does it manage oedema?**

Compression therapy is the application of a product that applies an external force to a body area with the aim of supporting venous and lymphatic return. This support results in reduction of oedema\(^{(17)}\), healing of venous leg ulcers\(^{(18)}\), reduction in trophic skin changes related to chronic venous hypertension\(^{(19)}\), and reduction in immune-complex deposition in patients with vasculitis\(^{(20)}\).

The force can be accomplished in a number of ways including:

<table>
<thead>
<tr>
<th>Unna Boot</th>
<th>General name given to a gauze bandage impregnated with zinc paste under a cohesive inelastic bandage.(^{(18)})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short-stretch bandages</td>
<td>Bandages with minimal or no elastomers. Low extensibility and high stiffness.. Low resting pressure and high working pressure.(^{(18)})</td>
</tr>
<tr>
<td>Long-stretch bandages</td>
<td>Highly extensible (elastic) bandages that expand or contract to accommodate changes in leg geometry during walking resulting in only small pressure changes. Instead the bandage sustains applied pressures for extended periods, even when the patient is at rest.(^{(21)})</td>
</tr>
<tr>
<td>Tubular bandages</td>
<td>A straight, elastic, tubular bandage that is applied in three layers of separate lengths creating a pressure gradient that is greatest at the foot and ankle.</td>
</tr>
<tr>
<td>Mutliple layer bandaging</td>
<td>A compression system that uses one or more layer and may consist of a combination of short and long stretch bandages.</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Compression garments</td>
<td>Specially manufactured garments that need to be individually measured and fitted. Highly variable levels of compression and stiffness.</td>
</tr>
<tr>
<td>Velcro garments</td>
<td>Segmented wraps for use on foot, lower leg and thigh, often over a garment that protects the leg but can also provide some compression. Highly variable levels of compression and stiffness.</td>
</tr>
<tr>
<td>Pneumatic compression</td>
<td>Pressure is applied via an inflatable garment, continuously, intermittently or in sequential cycles.(^{(18)})</td>
</tr>
</tbody>
</table>

*The images in the above table are only one example in each area. There are many other options and the ones shown above are given no particular endorsement.*

“In general, bandages are most commonly used for the treatment of active VLUs; compression stockings are generally used to prevent recurrence once the ulcer has healed.” – “Principles of compression...” pg 1\(^{(3)}\)

“Compression Therapy” is a therapy and the use and application of such needs to be done by health professionals who understand the underlying principles and their importance. The practitioner should seek to earn competency in the application of different forms of compression therapy. You must also work with the patient for a mutual understanding of why the therapy is required and to also explore the best form of the therapy for that patient to ensure optimum compliance.

This article focuses on lower leg compression therapy, but the reader should be aware that compression therapy is not restricted to the lower limb.
The science of compression
So with an understanding of oedema and why it needs to be managed, let’s look a little closer at the science behind compression.

Veins are the blood vessels that return the flow of blood to the heart. The venous system is a low pressure system that utilizes valves to stop retrograde flow. Being upright during the day encourages blood to pool, especially in the lower limbs, due to the pull of gravity. This pooling increases pressure within that vein, which, in turn, puts back-pressure on the smaller veins, venules, and capillaries that feed into it. Over time the thin walls of these veins can become distorted causing their valves to no longer be able to function, increasing the incompetence of the system and further increasing the pressures. (1, 3)

To get the blood moving when in an upright position, blood in the lower limb is squeezed upwards by the contraction of the surrounding thigh, calf and foot muscles. This is referred to as the Calf Muscle Pump. About 90% of venous return from the legs is through this action. This is most effective during walking and ankle movement. As a result any impairment to normal calf muscle activity such as reduced ankle mobility, an abnormal gait or neurological deficit, decreases the effectiveness of this pump. Age also plays its part with calf muscle function as a result of reduced muscle bulk. (3)

Changes in pressure (measured at the ankle) in the venous system in legs with healthy and defective venous valves during lying, rising, standing and exercise

Copied from “Principles of compression ...” (3)
There are two principles that we can apply to compression systems: Pascal’s Law and Laplace’s Law.

Pascal’s Law relates to rigid compression systems such as those provided by short-stretch bandaging systems and multi-layer systems that incorporate a short-stretch component. Pascal’s Law states that pressure applied to an enclosed system of an incompressible fluid is distributed evenly. See the image on the right where pressure is applied to one area of the tube of toothpaste but the toothpaste itself is extruded from all of the holes at the same rate, no matter how far away they are from the applied pressure(3). (Feel free to try this at home!)

The short-stretch bandaging acts like the rigid container. When the muscles in the leg contract they increase in circumference but are trapped within a rigid bandage. This muscle movement creates a pressure wave that is distributed evenly under the bandaging throughout the lower limb. This has a compressive effect, reducing the diameter of the veins within the lower leg and forcing the venous blood to return to the heart, producing a more normal venous pressure profile in the leg(3).

This is particularly useful in the ambulant patient where there is a more pronounced effect (high peak pressures) during exercise. In the purely short-stretch system, with no elastic to constantly ‘squeeze’, it also means that when the muscles are not being used the resting pressures are lower, which may improve comfort. The rigid system has been shown to quickly reduce oedema, but again, without the elastic’s ability to conform, as the oedema reduces the bandage quickly becomes loose and can change its position on the leg. This can result in an unwanted distribution of pressure, possibly even dangerously increasing pressure in specific areas like the ankle. Laplace’s law can help to explain what happens to pressures when the bandaging slips and bunches up at the ankle.

Laplace’s Law relates to how we can understand what kinds of pressures are being exerted under the bandage(22). From the equation itself we can get an idea of the basic tenets. With mathematical equations, the value on the left is directly affected by the value on the right or inversely affected if the value on the right is inverted.

\[
\text{Pressure (Pascals)} = \frac{Tension \text{ (Newtons)} \times n}{Radius \text{ (metres)} \times \text{Bandage width (metres)}}
\]

So to put it simply:

- Pressure increases if Tension increases
- Pressure increases if \( n \) (number of bandage layers) increases
- Pressure decreases if Radius of the leg increases
- Pressure decreases if Bandage width increases

The first point relates to how much tension is in the bandage. For elastic bandaging this will vary greatly depending on how far the bandage is stretched before application. If we don’t
stretch it enough the pressure will be lower, if we stretch it too much the pressure will be higher.

The second point takes into account the method we use to apply the bandage. A spiral application with 50% overlap will result in effectively applying 2 layers of the bandage, doubling the pressure. If we did a 66% overlap we would be applying 3 layers, triple the pressure! For this reason care should be taken when applying highly elastic bandages to not excessively overlap the edges.\(^{(22)}\)

The third point says that where the radius of the leg is smaller (ie- ankle) the pressure applied by the dressing will be greater. In a perfectly shaped leg (calf:ankle = 2:1) this will result in a graduated compression from higher at the ankle to lower at the calf. But this also applies to sub-shapes within the leg. The law applies to a cylinder, but the leg is not a perfect cylinder; the tibia or malleolus may protrude. These become sub-shapes of a different (smaller) circumference than the main shape of the leg. Pressure on these areas will be higher. This is why padding of the limb is so important.

Finally, the bandage width itself will have an impact on the pressures applied. There is a mathematical formula that explains the relationship between pressure, force and area, however I think an analogy that most of us are familiar with will make more sense. Imagine you have two bags of groceries. They both have exactly the same weight of groceries in them. One is a plastic bag with those thin handles that seem to stretch and get thinner as you carry the bag home (you know the ones!). The other bag has a wider handle to carry it by. The bag with the thinner handle will tend to ‘cut in’ to your hand (or arm) so that by the time you get to where you can put them down you have red stripes where they dug in. The bag with the wider handle does not dig in. This is because the force is distributed over a wider area, effectively reducing the pressure on your hand. Where bandage kits are available in different widths, it is very important to apply it as directed.

**Assessment**

There are many causes of oedema including hypertension and heart failure, severe protein deficiency, and renal failure. It is important during your assessment to determine the underlying cause to be able to create the best management plan.\(^{(1,3)}\) This involves a thorough examination of the patient’s presenting complaint, history (medications, comorbidities, social, financial), mobility, diet and physical and mental health. Clinical assessment of the patient’s neurovascular status and review of any pathology and imaging will help to make a diagnosis of the kind of wound and how best to address the oedema.

Caution is required where there is significantly reduced arterial supply or neuropathy. Being able to complete this assessment thoroughly and accurately requires training and practice
and should be done by health professionals competent in this area. If you are not certain, refer.

The patient’s financial status and options for funding also need to be included in your assessment. Different forms of compression not only have differing efficacy but also a vast range of costs. Some costs may be covered by a patient’s health insurance.

The patient’s physical capability is also incredibly important to assess. I have interviewed many patients who received compression garments that live in their draw because they are too hard to get on/off. They do not reduce oedema sitting neatly folded in the draw.

Some patient’s will have excellent support systems or family members. The capabilities of those support systems and/or family members also need to be understood. Non-government organisations often have limitations on how often they can attend to a patient and may or may not have nurses skilled in the application of certain forms of compression. Family members may also want to provide care and it is important to ensure they are trained and assessed in their application and removal of the required compression.

In my experience motivation and understanding are key to the successful use of compression therapies. My patients have often seen specialist before coming to me. They have been prescribed a great plan with the right compression, and yet they are still not healed when they see me 6 months later. I have found this relates to their understanding of what is important and why. There is such an incredible focus on the dressing that goes onto the wound that the compression therapy – which is the part that which actually heal the wound – is forgotten. While it is easy to describe to someone the importance of compression, I find it is more motivating to PROVE it. In my practice I aim for a 30-40% reduction in wound size in 3-4 weeks. I take digital photos and the wound is measured electronically (digital planimetry). I discuss the changes in the wound size with the patient, showing them the photos (proof) of healing. By being able to actually measure improvement or deterioration is not only a mandatory clinical tool, it is also a way to improve motivation.

**Ulcer types and compression**

**Venous leg ulcer**

VLUs are the most common type of chronic lower limb wound (40-85%) and are due to disease or disrupted function of the veins leading to a chronic venous insufficiency. There can also be some arterial insufficiency adding to the wound’s inability to heal and this is considered a mixed aetiology leg ulcer (10-20%)\(^{(15)}\). Both of these types of ulcerations respond well to compression therapy where compression is not contra-indicated during your assessment.
### Lymphoedema (trophic skin changes)

Oedema that develops as a result of a failure in the lymphatic system is referred to as lymphoedema. Chronic oedema is oedema that has been present for more than three months. Chronic oedema may be a result of a failure in the lymph system or it may have a more complex underlying aetiology[23]. Impairment of the lymph flow can be related to pressure (capillary pressure, negative interstitial pressure, interstitial fluid colloid osmotic pressure, and plasma colloid osmotic pressure), effects on the extrinsic or intrinsic propulsion mechanisms (fibrosis impeding muscle movement) or damage to/removal of lymphatic structures (radiation or dissection of nodes)[24].

Primary lymphoedema is considered to be genetic and can be congenital lymphoedema (present from birth), lymphoedema praecox (swelling developing around puberty) or lymphoedema tarda (where it develops later in life – although it then becomes unclear if this is truly primary or a secondary lymphoedema). Secondary lymphoedemas can be caused by cancer and cancer-related treatments, and non-cancer-related conditions like chronic venous disease, trauma, heart failure, inflammation, infection, pulmonary hypertension and conditions such as arthritis. A 2005 article by Williams attempting to determine the extent of the condition cited an overall prevalence of 1.3 to 1.4 per 1000[23].

Compression is a major component in treatment and management of lymphoedema, along with skin care, manual lymphatic drainage and physiotherapy[17].

### Table: Ulcer Characteristics

<table>
<thead>
<tr>
<th>Type</th>
<th>Location</th>
<th>History</th>
<th>Characteristics</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous leg ulcer</td>
<td>Gaiter region of the leg; most commonly around the medial malleolus</td>
<td>Varicose veins, DVT, Other venous disease, Trauma, Surgery</td>
<td>Irregular sloping margins, Usually shallow, Fibrinous, granulating base, Variable size: from small to encircling the leg, High exudate levels, May be painful; pain relieved by elevation of the limb</td>
<td>Periwound/lower limb oedema, Ankle flare, Varicose veins, Varicose eczema, Lipodermatosclerosis, Hyperpigmentation, Atrophic blanche</td>
</tr>
</tbody>
</table>

*Photo copied from http://jamanetwork.com/journals/jamasurgery/fullarticle/394351*

*Table copied from “Simplifying Venous Leg Ulcer Management”[15] and photo from DermNetNZ.org*
Arterial ulcer

Arterial ulcers account for 5-30% of leg ulcers and are due to a disruption of the function of the arteries. The point at which microcirculation and nutrient blood flow to tissues are severely disturbed is known as critical limb ischaemia\(^\text{[26]}\). These types of wounds contra-indicate the use of compression. Revascularisation, risk-factor modification and administration of antiplatelet therapy are the cornerstones of treatment for these patients. Risk reduction includes smoking cessation and treatment of hyperlipidaemia while arterial hypertension and diabetes mellitus should be adequately treated.\(^\text{[27]}\)

<table>
<thead>
<tr>
<th>Type</th>
<th>Location</th>
<th>History</th>
<th>Ulcer Characteristics</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial Ulcer</td>
<td>Toes, feet or lateral or pretibial aspects of the lower leg</td>
<td>Intermittent claudication/rest pain Cardiac or cerebrovascular disease</td>
<td>Punched out, sharply demarcated edges Painful Small and deep Necrotic wound base Dry/low exudate levels Gangrene may be present</td>
<td>Surrounding skin is often dry and shiny with loss of hair Weak or absent foot pulses</td>
</tr>
</tbody>
</table>

Table copied from “Simplifying Venous Leg Ulcer Management”\(^\text{[15]}\) and photo from the Primary Care Dermatology Society (http://www.pcds.org.uk)

Diabetic foot ulcer

The management of diabetic foot ulcers (DFUs) focuses on the two main reasons for ulceration: neuropathy and ischaemia. As such, a multi-disciplinary team is very important, Podiatrist and/or Orthotist can look at off-loading of the DFU and the Vascular Surgeon can determine/ensure adequate blood supply. Regular debriding of wound and surrounding callous is also important as well as appropriate management for the wound itself\(^\text{[28]}\). However, the person who has diabetes is not automagically protected from venous insufficiency. Where there is oedema or venous stasis that requires management:

“Compression may be used safely in patients with controlled diabetes. Thorough assessment of peripheral perfusion and neuropathy is essential in determining the level of risk and in selecting an appropriate compression system”. WUWHS, p10 \(^\text{[29]}\)

<table>
<thead>
<tr>
<th>Type</th>
<th>Location</th>
<th>History</th>
<th>Ulcer Characteristics</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic Foot Ulcer</td>
<td>Pressure bearing areas of the sole of the foot (neuropathic) Margins of the foot, e.g. over first or fifth metatarsophalangeal joints (neuroischaemic)</td>
<td>Diabetes</td>
<td>Sensory loss when neuropathy is present Variable depth: may be deep +/- sinuses, and may involve tendons and bones</td>
<td>Neuropathic: foot may be warm; ulcer often surrounded by callus Neuroischaemic: foot may be cool and foot pulses may be absent</td>
</tr>
</tbody>
</table>

Table copied from “Simplifying Venous Leg Ulcer Management”\(^\text{[15]}\) and photo from DermNetNZ.org

When would I NOT use compression therapy?

Given that compression therapy has such immense benefits to the majority of lower leg ulcers, why would I not use it???
Not Competent

The application of compression and the use of compression therapy is complex. As you can see by the science behind the bandaging, small changes in application can result large under-bandage pressure changes, potentially causing harm. Also, your assessment needs to be thorough to ensure you do not miss subtle signs that indicate the patient will not be able to tolerate the compression\(^{(30)}\).

![Pressure damage over the anterior tibia on a thin leg](image1)

![Rupture of the distal anterior tibial tendon due to pressure damage](image2)

Photos copied from Beldon, 2008\(^{(30)}\)

Arterial Disease

Where arterial supply is already compromised, adding in additional pressure (be it compression therapy or anti-thromboembalism stockings) we are exacerbating the problem. But without thorough assessment we may not be aware of the problem. Also, we can not always rely on the patient to tell us if there is a problem with the bandaging, such as:

- where the person is neuropathic,
- on strong pain medication,
- already in so much pain from their ulcer they may not notice a pressure injury forming,
- or not willing to speak up due to the “power difference” or perception that “Doctor knows best”

Perrin’s article in Phlebolymphology (2008)\(^{(2)}\), while it is citing somewhat older data, is valid in it’s concerns that thorough assessment is required prior to application and that application of any compression be closely monitored.

Use with caution...

Mixed venous and arterial disease

Once you have conducted your clinical review and determined that the ulcer has both a venous and arterial component, do you compress? General rule of thumb based on ABPI is that anything between 0.8 and 1.3 is compressible with full-strength compression. Outside of this range (from 0.5 to 0.8) compression can still be considered but should be reduced\(^{(31)}\). My comment here is to understand that an ABPI (or TBI) is only ONE tool in your clinical toolkit and the data should not be used to independently inform your decision. Many factors can skew the results of your ABPI so before you start compression do a full clinical assessment,
including the ABPI, then decide. If you are not certain, ensure there are checks in place to confirm the compression is being tolerated (in hospital this might be circulation obs for the first 24 hours).

Heart Disease
With compression the fluid from the tissues is returned to the venous system and back into circulation. In the patient with heart failure this can lead to a risk of excessive pre-loading of the heart. It is important to introduce compression in a staged fashion, lower pressures (short-stretch) and unilaterally. Work with the Cardiologist and primary Physician to closely monitor and manage the heart failure. If the heart failure is well controlled, slowly increase compression therapy to optimum, again, in conjunction with close monitoring by their medical team\(^{(29)}\).

The Diabetic Limb
In the patient with diabetes there is a risk of damage to the foot from lack of sensation or lack of blood supply to the foot that can lead to ulceration, infection and worse. Thorough neurovascular assessment is required. Where there is critical limb ischaemia – do not apply compression, refer to Vascular ASAP. Explore possible vascular investigations/interventions. Where there is neuropathy – consider using mild to moderate compression with extra padding and intermittent pneumatic compression. Always ensure diabetes is well controlled, involve the Diabetic support team and Podiatrist\(^{(29)}\). There may be other advanced therapies (negative pressure wound therapy, biological dressings, bioengineered skin equivalents, hyperbaric oxygen therapy, platelet rich plasma and growth factors) that can also be explored where there are no other options for improving arterial supply and wounds are not improving. However these are expensive therapies and there is insufficient evidence to determine their effectiveness\(^{(32)}\).

Guidelines
Guidelines are not linked to here as they change regularly with updates in evidence. But I highly recommend you see what your service has with regards to general skin care protocols as well as leg ulcer management protocols. Other guidelines may be available to you at a State or Local level and there are National and International/Cooperative guidelines that are evidence-based, available online.
References