RECOGNITION, TREATMENT, AND PREVENTION OF CHRONIC VENOUS INSUFFICIENCY
IN PRIMARY CARE

By

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Chronic Venous Insufficiency (CVI) is often ignored or poorly managed by primary care providers (PCPs). A literature review was conducted to determine the most relevant guidelines for PCPs to treat CVI with and without ulceration. PCPs must be aware of the risk factors, early signs, and symptoms of CVI in order to promote prevention and provide early treatment, thereby avoiding the permanent consequences of CVI and costly treatment of ulcerations.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Title page</td>
<td>1</td>
</tr>
<tr>
<td>Committee Members</td>
<td>2</td>
</tr>
<tr>
<td>Abstract</td>
<td>3</td>
</tr>
<tr>
<td>Table of Contents</td>
<td>4</td>
</tr>
<tr>
<td>Introduction</td>
<td>5</td>
</tr>
<tr>
<td>Literature Review</td>
<td>5</td>
</tr>
<tr>
<td>The Anatomy and Physiology of the Lower Extremity Venous System</td>
<td>6</td>
</tr>
<tr>
<td>Pathophysiology of Venous Insufficiency</td>
<td>8</td>
</tr>
<tr>
<td>History and Physical Exam</td>
<td>9</td>
</tr>
<tr>
<td>Treatment of CVI with ulceration</td>
<td>13</td>
</tr>
<tr>
<td>Table 1: Compression Products</td>
<td>20</td>
</tr>
<tr>
<td>Treatment of CVI without ulceration</td>
<td>21</td>
</tr>
<tr>
<td>Prevention of CVI</td>
<td>21</td>
</tr>
<tr>
<td>Conclusion</td>
<td>23</td>
</tr>
<tr>
<td>References</td>
<td>26</td>
</tr>
</tbody>
</table>
RECOGNITION, TREATMENT, AND PREVENTION OF CHRONIC VENOUS INSUFFICIENCY IN PRIMARY CARE

INTRODUCTION

Symptoms of Chronic Venous Insufficiency (CVI) have been documented as early as 1500 B.C.\(^1\) Although much is known about the pathogenesis and treatment of CVI, it is often poorly managed by many medical providers.\(^1\) It is estimated that CVI affects 1-3% of the population in Western countries, although the incidence may be much higher due to lack of reporting standards.\(^2\) The most serious consequence of CVI is venous ulceration.\(^1\) It is estimated that approximately 2 million working days are lost, and 3 billion dollars spent annually on the treatment of venous ulcers in the United States.\(^3\) Venous wounds are often disguised as a comorbid condition but these chronic wounds represent a silent epidemic that poses a major threat to public health and the economy of the United States.\(^4\) CVI is the cause of 80-90% of all lower extremity ulcers\(^2\) and can result in impaired mobility, chronic pain, disability and a diminished quality of life.\(^5\) Many patients receive unnecessary and ineffective medications including antibiotics and diuretics. Primary care providers are in a unique position to provide effective disease management through education, prevention strategies, treatment of complications, and long-term edema control.

LITERATURE REVIEW

A literature review was done searching for current information on CVI evaluation, management and prevention. Initially, search engines including: Medline, CINAHL, Cochrane Library and Wiley Interscience Journals were searched using dates of 2007-2011 and the key search words of “chronic venous insufficiency” or “venous stasis ulcer.” Articles discussing non-surgical and surgical treatment or prevention of CVI were included.
Chronic Venous Insufficiency

Articles distributed by “for profit” companies were not included. Articles regarding advanced treatment of venous ulcers were not included because this paper is intended for PCPs, not specialists in the field of wound care. Guidelines for venous ulcer prevention and treatment were found through the AHRQ website as well as various other articles and the database, UptoDate.

This paper reviews the anatomy and physiology of the lower extremity venous system as well as the pathophysiology of CVI for the vital task of educating patients. Disease recognition and risk factors that contribute to CVI will be discussed to improve early treatment and promote prevention strategies. Evidenced based wound care principles for venous ulcer care are presented, including: systemic or topical antibiotic use; when diuretics are appropriate; dressing choices; and the necessity of compression. Armed with the foregoing knowledge, a PCP can improve many patients’ quality of life thereby avoiding the most severe consequences of future financial expense and physical suffering.

THE ANATOMY AND PHYSIOLOGY OF THE LOWER EXTREMITY VENOUS SYSTEM

The frequent occurrence of CVI is understandable considering the anatomy of the lower extremity venous system. The system of veins in the lower extremity function both as a reservoir to hold extra blood and as a conduit to return blood from the legs to the heart and lungs. Unlike the arteries, which possess three well-defined tissue layers, most veins are composed of only one tissue layer. Only the largest veins possess internal elastic membranes, and at best, this layer is thin and unevenly distributed, providing little buttress against high internal pressures. A large portion of the 5-10L/minute cardiac output is received by the end-capillary venules of the legs for eventual delivery back to the heart and
Chronic Venous Insufficiency

lungs. The lower extremity venous return to the heart occurs against a reverse pressure gradient, gravity, and fluctuating thoraco-abdominal pressures. Additional resistance created by elevated right atria pressure in congestive heart failure can also influence this venous return. Muscle pumps of the calf and foot propel venous return through a complex series of valves that are individually frail and prone to malfunction. As stated in Fundamentals of Phlebology: Venous Disease for Clinicians, "Considered in this light, the venous system seems almost magical in its function." 

There are three sections of the lower extremity venous system: deep, perforating, and superficial veins. The deep venous system lies within the muscle fascia, the superficial veins are in the subcutaneous tissue, and the perforating veins connect the two systems, draining venous flow from the subcutaneous area through the muscle fascia into the deep vein system. The superficial compartment is a low-pressure area while the deep compartment is a high-pressure area. During walking, the calf muscles flex (muscular systole) increasing the internal pressure within the fascial muscle compartment, forcing open the one-way valves through which the blood flows upward. During relaxation of the calf muscle (muscular diastole), the pressure within the deep vein decreases, the valves fall back to the closed position, and blood is maintained at the higher level. During muscular diastole, the pressure in the deep veins is temporarily lower than the superficial veins. The blood can then drain from the superficial system through the perforating veins into the deep venous system. Normally, perforating veins contain valves that prevent reflux of blood from the higher pressured deep veins into the lower pressured superficial system during muscular systole, thereby preventing venous hypertension. If the perforator valves stop functioning correctly, the deep vein system produces backflow into the
Chronic Venous Insufficiency

superficial system creating a high-pressure system where it does not have the structural support of the fascia. Unfortunately, there are many conditions that damage the lower extremity venous system, causing a chain reaction of events that can lead to venous dysfunction, venous hypertension, valve failure, and extravasation of vascular fluid into the interstitial area of the lower leg causing inflammation, edema, skin changes, and ultimately ulceration.

PATHOPHYSIOLOGY OF VENOUS INSUFFICIENCY

Venous dysfunction develops when venous return is impaired for any reason, and it can arise from abnormalities within the deep veins, perforating veins, superficial veins, or a combination thereof. There are two main categories of venous pathophysiology: reflux or obstruction. Causes of reflux include: primary muscle pump failure (non ambulatory or paralyzed patients), or venous valvular incompetence, which may be segmental or involve the entire length of the vein (genetics, trauma, surgery). Obstruction of the venous system is caused by thrombotic (DVT) or nonthrombotic (compression of the venous system from a mass, obesity, or lymphedema) causes. Whichever the cause, the main issue is the failure of the valves to close and direct flow of blood to the deep and central venous system which leads to venous stasis and congestion in the lower extremity venous system. Prolonged congestion leads to venous hypertension which results in vein dilatation, valvular incompetence, and reflux of blood flow further compounding the problem. Venous hypertension causes most venous pathology of the leg, including: telangiectasias and varicose veins; edema; lipodermatosclerosis; and ulceration. Chronic venous hypertension causes impairment and inflammation of the microcirculatory network of venules, capillaries and arterioles within the skin. The vessels dilate allowing leakage of
Chronic Venous Insufficiency

micro and macromolecules into the interstitial tissue causing edema and further damage to
the dermis and adipose tissue. Persistent inflammation and microangiopathy may then
manifest as a range of skin changes including: eczema (red, flaky, itchy skin a.k.a. stasis
dermatitis); hemosiderin staining (brown discoloration); atrophie blanche (white spots of
depigmentation); lipodermatosclerosis (hardening); and ulceration. These changes are
preventable, but most are not reversible once they occur. PCPs must be aware of the risk
factors, early signs, and symptoms of CVI in order to promote prevention and provide early
treatment, thereby avoiding the permanent consequences of CVI and costly treatment of
ulcerations.

HISTORY AND PHYSICAL EXAMINATION

Prevention, by definition, is the act of impeding an event from occurring. In order to
prevent CVI, a PCP must assess the risk factors for each patient. While interviewing the
patient or reviewing a patient’s medical history, PCPs should make note of the many risk
factors for CVI including: older age; female sex; history of pregnancy; flat feet; an
occupation or hobby that requires standing or sitting for long periods; obesity; immobility;
smoking; lower extremity or pelvic surgery; above average height; or episodes of lower
extremity cellulitis. Previous history of a lower extremity deep vein thrombosis,
coagulation problems, or injection drug use in the lower extremity, all of which would
cause vein damage and scar tissue, are other risk factors for CVI. Family or personal
history of varicose veins is also a risk factor.

Patients that already have CVI often report leg aching, heaviness, swelling, leg cramps,
itching or tingling skin on legs, restless legs, and venous claudication (leg pain in a
dependent position alleviated by elevation).
Some patients may not even realize they have edema in the legs because their legs have always been large. The leg discomfort related to CVI does not radiate as with radiculopathies nor does joint movement exacerbate the discomfort as with arthritis. Most of the symptoms are subjective, but the one common objective finding is edema. Heart failure, medications (see box below), pulmonary hypertension, malnutrition, renal or hepatic failure, and pregnancy are some of the more common systemic causes of peripheral edema. While some of the local causes of peripheral edema consist of: DVT, CVI, postoperative edema, infection, phlebitis, inferior vena cava compression or lymphedema.

<table>
<thead>
<tr>
<th>Common medications that can contribute to peripheral edema</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antihypertensive agents:</strong> Calcium Channel Blockers Beta-adrenergic blockers Vasodilators Central alpha agonists Peripheral alpha blockers</td>
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</table>

The physical examination should always include weighing the patient because peripheral edema may not be visible until there is a 10% increase in total body weight. Measurements of the ankle and calf circumferences are rough estimates of edema that can...
Chronic Venous Insufficiency

be used to gauge progress of treatment. The measurements must be standardized to the practice. For measurements in our clinic, the patients sit down with legs mildly elevated in a reclining chair. Ankle measurements are taken 5 cm up from the medial malleolus, and calf measurements are taken 10 cm down from the patient's posterior knee crease. Other practitioners take the measurements at certain distances from the floor with the patient standing up straight. Either technique works but each measurement must be done using the same parameters at each visit. If clinical signs cause suspicion of a possible DVT, draw a D-dimer level and perform a duplex ultrasound of the leg, if appropriate, for confirmation.

A detailed examination of the lower extremities will include a peripheral vascular examination of the dorsalis pedis and posterior tibial pulses. Often edema due to venous disease makes palpating the dorsalis pedis and posterior tibial pulses impossible. If unable to palpate pulses on the feet, obtain an ankle brachial index (ABI) measurement, a non-invasive assessment of arterial status or refer the patient to a vascular specialist for non-invasive or invasive testing if appropriate. For instructions on how to obtain an ABI go to http://www.youtube.com/watch?v=LvHeMiCaUdw. A Doppler is necessary for this test. If a Doppler is not available, the patient can be sent to a diagnostic center or a vascular doctor for testing. Adequate arterial perfusion (an ABI > 0.8) must be assessed before compression treatment can begin. Unfortunately, 15-20% of patients with venous ulcerations also have arterial insufficiency, making compression therapy challenging.

Assessment of the lower extremities should consist of noting:

- skin condition – intact or not; edema- pitting or nonpitting, bilateral or unilateral;
- varicose veins; dry, moist, or scaly
Chronic Venous Insufficiency

- Color- normal, hyperemic, or red streaking
- Temperature – warm, cool, hot and inflamed
- sensation test with a monofilament – intact, diminished or absent
- assessment of mobility – is the patient able to perform the calf muscle pump exercise?
  Is the patient ambulatory?

Signs of venous pathology include:

- Reticular or spider veins - they appear as reddish or bluish “broken” veins usually near the medial malleolus of the ankle
- Red and flaky skin characteristic of venous eczema (dermatitis)
- Hyperpigmentation (brown discoloration) due to hemosiderin deposits in the dermis
- Atrophie blanche (areas of depigmentation) that appear in the gaiter area (between the ankle to mid-calf)

Patients with severe CVI almost universally have lipodermatosclerosis, which is the progressive hardening of the skin and subcutaneous tissue of the lower extremities. Edema is difficult to detect if lipodermatosclerosis is present because the skin is so scarred it cannot stretch, but the venous hypertension is still present. Venous ulcers are the most severe consequence of CVI and are usually irregularly shaped, tend not to be deep, and drain a large amount of drainage. Spontaneous venous ulcerations generally occur on the medial or lateral ankle area, just above or below the malleolus. This is the location of the greatest venous hypertension. Unfortunately, with a grossly edematous leg, any minor trauma can quickly develop into a venous ulcer without proper treatment. Once skin ulceration develops, the standards of care for venous ulcers should be followed:
  debridement of devitalized tissue if applicable, infection control, best dressing for the
Chronic Venous Insufficiency

wound, and compression.¹²

TREATMENT OF CVI WITH ULCERATION

As with all localized problems, any contributing systemic factor that can affect the outcome should not be overlooked. Systemic factors that influence the healing of a venous ulcer include: obesity, immunosuppression, malnutrition, diabetes mellitus or congestive heart failure.¹ Addressing systemic factors is vital as well as the local treatment to a venous ulceration. For most PCPs, a referral to a wound care center would be the best use of resources. While the PCP is addressing the systemic issues, a wound care team can provide the patient with the most effective treatment provided by the most knowledgeable health care providers to treat the wound. If a wound care center is not an option, the following guidelines can help a PCP in the treatment of a venous ulcer.

The treatment of venous ulcerations requires patience. It is a slow process. The cause of the problem, venous hypertension, has to be managed before healing can begin. This is why compression is the main treatment modality for CVI. Consistent compression of the limb will eliminate edema by counteracting the venous hypertension.¹³ A common misconception in the treatment of CVI is that diuretics will reduce the edema. The pitting edema of CVI is caused by the shift of vascular fluid to the interstitial spaces and out of the vascular circulation. Diuretics will only work to reduce the intravascular fluid volume and therefore will not help unless compression therapy is used in conjunction with diuretics. Compression therapy will help reduce venous hypertension so that the interstitial fluid can return to the venous system. Diuretic use can lead to hypovolemia and should be used judiciously and only for temporary intervals when treating CVI.¹⁰

Compression, when applied properly, will help reduce the pain and inflammation
caused by excessive edema. The process of reducing chronic edema includes these key elements to promote venous return: compression wraps; elevation of the legs to heart level when the patient is sitting; and flexion of the calf muscle. Walking and calf muscle pump exercises activate the calf muscle pump to propel venous return back towards the heart reducing the pressure within the lower extremity venous system. Venous hypertension occurs when the pressure is not reduced during exercise. The compression wraps transform the superficial vein system into a higher-pressure system, like the deep venous system enclosed by the muscle fascia, allowing for increased blood flow upward during muscle contraction. External compression to the lower leg creates high-pressure peaks during muscle contraction causing short, intermittent venous occlusions, which are thought to reduce venous reflux and lower venous hypertension. External compression will also prevent extravasation of fluid into the interstitial area by increasing the external pressure. Slowly the interstitial fluid returns to the venous system. When sitting or standing still, the effects of gravity will continue to influence distal edema with swelling against the wrap and discomfort to the patient. This is why it is key to educate the patient about the process so they know what is happening and what to do. Elevating their legs above their heart when sitting will facilitate venous return. Many patients get frustrated when told they have to “go home and elevate”, thinking they cannot elevate their legs all the time. Constant elevation is neither practical nor healthy for the patient. The patient can continue with their activities, but they need to work in 1-2 hour periods of elevation between activities.

Compression is measured in mmHg and that measurement reflects the amount of external pressure applied to the leg. The amount of pressure (sub-bandage pressure)
Chronic Venous Insufficiency

necessary relies on the underlying pathologies of the patient but must also be tolerated by the patient. Compression treatment for venous ulcers with the use of a high-compression (>40mmHg at the ankle) bandage, i.e. 3-layer, 4-layer, short-stretch or paste containing bandages, has been shown to significantly reduce healing time. A trained clinician must apply these bandages. The compression is to be graduated, meaning a 20-30% reduction of pressure from the ankle to just below the knee. This occurs naturally when compression is applied equally to a limb of “normal” proportions, due to the principles of Laplace’s Law, the smaller the circumference at any given point, the greater the pressure. In this case “normal” proportions means the circumference increases from ankle to knee. When a leg is not of “normal” proportions, it may require reshaping with padding prior to the application of the compression. The provider who applies the bandage should always have the patient’s ankle flexed to 90 degrees during application to allow for optimal ankle movement and reduce “bunching up” of layers at the ankle during flexion. Compression bandages can be single layered or multi-layered. The latest Cochrane Review of Compression for venous leg ulcers has stated that multi-layer bandages are most effective for treating venous leg ulcers. There are many types of these bandages on the market. The bandages may be made of elastic or inelastic materials and each has its own advantages and disadvantages. A provider must understand how the different pressures can be achieved by using the properties of each bandage system. For ambulatory patients a stiff bandage system, through the use of inelastic materials or multiple layers of elastic materials, provides the higher working pressure necessary to prevent venous reflux and reduce venous hypertension. Inelastic bandages, such as zinc paste bandages, i.e. Unna’s boot, and short-stretch wraps, i.e. Comprilan, can achieve resting pressures of 30-
Chronic Venous Insufficiency

60mmHg. This pressure is not sustained though and decreases over the first 24 hours with movement or decreased edema, resulting in lighter resting pressure but maintains an effective working pressure. Due to the loss in pressure, more frequent bandage changes may be required to prevent slippage and maintain an effective bandage pressure.

Elastic materials or long-stretch bandages in multiple layer bandage systems can sustain the pressure for up to a week due to their ability to accommodate movement and changes in limb circumference. The use of a single elastic bandage, i.e. an ace wrap, to apply strong compression is not recommended and can be dangerous due to risk of pressure damage. Multi-layer bandage systems provide protective layers and are safer. These materials sustain pressure even at rest, and therefore may be more effective for immobile patients or those with an ineffective calf muscle pump. The staff that applies the bandage systems should acquire the knowledge and skills necessary for safe application while also providing effective compression. It is a delicate balance with many other factors contributing to the challenge. Many patients have difficulty tolerating compression or have had a bad experience already and are reluctant to try again. The treatment team needs to remember, “some compression is better than no compression” and a gradual increase in compression may improve concordance for some patients. Another important point to remember is that there is always more than one path to get to a result. During the “therapy phase” when venous hypertension and edema is being reduced with compression, there are other important wound-healing factors to consider.

Debridement is necessary if the wound bed is covered with necrotic tissue. The body’s defenses will handle a small amount, but if there is a thick layer of slough, the body’s defenses will be overwhelmed, and it will significantly slow wound healing. Also, the
devitalized tissue provides a breeding ground for bacteria. Sharp debridement with a curette works well. Sharp debridement is the quickest debridement method. The slough has no sensation but the patient’s leg may be too tender for debridement so the provider may need to delay debridement until compression has been applied for a week or two then perform the debridement after the inflammation is reduced. There are many other forms of debridement including: mechanical, biological, enzymatic, or autolytic.

As well as removing the necrotic tissue, there are other guidelines regarding infection control in venous ulcer treatment. Patients with weeping, edematous legs are at increased risk for cellulitis according to the Wound Healing Society (WHS). The WHS states the plasma leaking from the legs actually neutralizes the fatty acids of sebum, which inactivates the bactericidal properties of the skin. If infection is suspected in a previously debrided ulcer, or if epithelialization from the margin is not progressing within 2 weeks of debridement and initiation of compression therapy, the provider must determine if infection is the cause of delayed healing in the debrided ulcer by obtaining a tissue biopsy or by a validated quantitative swab technique. UpToDate suggests injecting 1-2 mL of sterile saline into the dermis surrounding the ulcer then quickly withdraw the fluid then send it for culture. The WHS states “systemically administered antibiotics do not effectively decrease bacterial levels in granulating wounds; however, topically applied antimicrobials can be effective.” The WHS recommends treating any ulcer with \( \geq 1 \times 10^6 \text{ CFU/g} \) of tissue or any tissue level of beta hemolytic streptococci, following adequate debridement, with topical antimicrobial therapy. The recommendation is then somewhat vague by stating the topical antibiotic should be discontinued when the wound is back in “bacterial balance” to avoid bacterial resistance to the antibiotic or cytotoxic effects of the
topical antibiotic.\textsuperscript{15}

Often the dermatitis caused by extreme edema is mistaken for acute cellulitis.\textsuperscript{2} It is very difficult to tell the difference between the two conditions in as they both have pain, edema, warmth, and erythema as classic signs. The history of the timing of onset of symptoms and the patient’s usual state of leg health are important facts in this situation. Compression will reduce the symptoms associated with dermatitis. Systemic antibiotics should be reserved for patients showing signs of a significant infection: increase in pain, leg edema, local heat and tenderness; increasing erythema of the surrounding skin; lymphangitis (red streaks traversing up the leg); rapid increase in the size of the ulcer; or fever.\textsuperscript{10} For applicable patients, the WHS recommends treating with a systemic gram-positive bactericidal antibiotic to eradicate the most common causes of skin and subcutaneous infections: streptococci or staphylococci.\textsuperscript{15} During systemic treatment for infection, compression must continue, but the wound should be inspected more frequently to ensure resolution of symptoms. If topical antimicrobial therapy is used, it will be part of the primary dressing for the wound.

Along with compression and infection control, the choice of dressing for the wound is also very important. There is a plethora of dressing choices on the market. Almost every dressing product can be impregnated with a topical antimicrobial agent. All dressing classes will not be discussed here due to the exhaustive list of options. As venous wounds drain copious amounts, absorptive dressing supplies are recommended. The dressing under the compression wrap is necessary to keep the wound bed moist but not saturated.\textsuperscript{15} A dressing should be selected according to the amount of drainage expected and the duration before the next dressing change.\textsuperscript{12} The amount of drainage will decrease as the
edema decreases. An alginate or hydrofiber dressing will work well to absorb the copious amounts of drainage with an absorbent pad over it to wick away the overflow. Aquacel by Convatec, a hydrofiber, is specially formulated to wick away excess moisture from the wound bed straight up into the absorbent pad and does not allow the drainage to travel laterally onto the peri-wound skin to cause maceration. Of course, a dressing that will not stick to the wound is optimal, but it also needs to be absorbent: ABD pads, Exudry or even a sanitary napkin work well. Reevaluation of the choice of dressing should happen frequently to keep the moisture level of the wound bed optimal.

Once edema has been sufficiently reduced by regular changes of compression wraps and the wound is healed, the maintenance phase begins. The patient can be transferred to a self-applied compression stocking or wrap – see table 1. At our clinic, Occupational Therapists work with patients at donning/doffing their stockings or wraps until the patient is independent with their use. Once the patient’s legs are pain free, wound free, and lighter weight due to loss of fluid volume, adherence to compression therapy is greater. Of course, CVI is a chronic condition and ulcer recurrences will occur. The patient will then return to the treatment phase. Positive reinforcement regarding use of compression stockings or wraps by the PCP is helpful for patient compliance. Compression stockings or wraps not only help reduce edema, but they also protect the leg from trauma. Measuring for compression stockings or wraps is a specialized skill. There are many different products on the market. These items can be ordered from a durable medical supply company or specialty shop with professionals trained in compression therapy.

See Table 1 below for multiple compression choices
### Chronic Venous Insufficiency

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<tr>
<th>PRODUCT</th>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
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| Unnaflex - 3 layer wrap | - Relatively inexpensive  
- Multilayer compression | - Must be applied by a trained professional due to no measurable gradient tool |
| Profore - 4 layer wrap | - Reduces edema quickly | - Slips down easily  
- Must be applied by a trained professional  
- Slightly more expensive |
| Surepress- elasticized bandage | - Measurable gradient  
- Can be applied by a caregiver | - Single layer compression  
- Can be applied incorrectly by caregiver |
| Comprilan - short stretch bandage | - 100% cotton  
- Can be applied by a caregiver  
- Can be used in a multilayer system or on its own | - Single layer compression  
- Can be applied by a caregiver incorrectly |
| Tubigrip - elasticated tubular bandage, comes in a 10m roll  
*Easy initial compression to start with in the office* | - Comes in many sizes  
- Measurable gradient  
- Can increase compression by using multiple layers  
- Can be donned independently  
- Soft  
- Easily laundered  
- Can be used in conjunction with other forms of compression | - Contains latex  
- Wears out within 3-4 weeks  
- Can roll down causing a tourniquet effect  
- Light compression |
| Juxta-lite by Circ-aid – NEW | - Easy to apply  
- Gradient  
- Soft, breathable  
- Multiple sizes  
- Long lasting  
- Independent donning  
- Washable  
- May be paid by Medicare b/c of the potential for 30-40mmHg compression | - Improper donning – usually too loosely applied  
- Needs training to don correctly |
| Off the shelf Compression stockings- many companies including:  
Jobst  
Medi  
Juzo  
Sigvaris | - Open or closed toe  
- Many different sizes/colors/styles  
- Inexpensive  
- Multiple measurable  
- Multiple measurable gradients | - Wrinkles can cause wounds  
- Increase in mmHg compression makes more difficult to don  
- Wear out at 4-6 months and need to be replaced |
| Custom measured and fit compression stockings or wraps  
Juzo  
Elvarex  
Jobst | - Consistent measurable gradient  
- Minimizes risk of pressure injury  
- Long lasting  
- Zipper can be added to easy with donning by caregiver | - Expensive  
- Can be measured incorrectly  
- Can be difficult to don/doff |
Appliances to assist with donning/doffing stockings:

- Easy-slide
- Butler (metal cage)
- OT modifications (loops)
- Wear gloves for better grip
- Cotton-liner if contact dermatitis
- Knee-high nylons to secure dressing while stocking applied

TREATMENT OF CVI WITHOUT ULCERATION

The treatment of CVI without ulceration is much the same as for CVI with ulceration. The purpose of treatment is to prevent ulceration and improve leg health. A study done in San Diego related to venous disease and quality of life created evidence showing that even modest venous disease causes functional limitations and limitations in daily activities.\(^5\)

The venous hypertension requires treatment first with compression. If legs are grossly edematous, the PCP can use multilayer compression wraps to reduce the edema. Once the edema is reduced, the patient can be measured for compression stockings or wraps that will prevent the edema from recurring. The use of compression should be daily and forever.\(^6\) It is customary to treat CVI with conservative treatments first, but evidence suggests that surgery aimed at preventing venous reflux decreases recurrence of ulcers.\(^8\) Therefore, it seems reasonable to speculate that such treatment could reduce the risk of ulcers if performed early in the course of CVI.\(^8\) Ultimately, prevention of CVI in all patients is the goal of the PCP and awareness of the condition starts the process of prevention.

PREVENTION OF CVI

Prevention of CVI begins with an assessment of risk factors and eradicating any modifiable risk factors. A PCP can encourage at-risk patients to adopt a schedule of leg
elevation throughout their day to promote venous drainage. The patient must understand that a traditional recliner chair does not sufficiently elevate legs to promote venous drainage. The patient would obtain better elevation by lying on a sofa with their legs on pillows at one end. Patients need to walk and perform calf muscle pump exercises to facilitate venous blood flow. A physical therapy referral may be appropriate for instruction in leg exercises to strengthen the calf muscle pump. Most importantly, patients need to understand the importance of wearing compression daily for the rest of their lives. Just like a pair of glasses that are used every day for vision, compression stockings or wraps are also needed every day. Providing education to the patient so that they understand the pathophysiology of the disease and how they can treat themselves to prevent future problems is the goal. Of course, education is an ongoing process.

Patients should be instructed in daily care of their legs. Daily skin care includes washing using a mild, pH balanced soap to remove any crusts that may form, rinsing well, drying, then applying a moisturizing lotion or petroleum-based product. It is best if patients shower at night before bed so that their legs can be elevated during the night. Then they can don their compression stockings or wraps first thing in the morning before they get out of bed and their legs start to swell. For support in donning/doffing the compression stockings or wraps, an occupational therapy referral may be valuable.

As well as the above recommendations, referral to a vascular surgeon early in the disease process is another WHS guideline. A procedure known as subfascial endoscopic perforator surgery (SEPS) is the procedure of choice to address the underlying pathologic etiology of venous ulceration. The SEPS procedure prevents backflow of the deep venous system into the superficial venous system by severing the perforating veins. The
Chronic Venous Insufficiency

procedure is not effective if the patient already has severe deep venous disease.\textsuperscript{16} Therefore, evaluation by the vascular MD earlier in the disease process may benefit the patient. There are also less extensive procedures than deep ligation of multiple perforating veins that can be performed by a vascular surgeon to decrease venous hypertension when combined with compression.\textsuperscript{16} Some of the procedures include: saphenous vein stripping, radiofrequency ablation, endovenous laser ablation (EVLA) or foam sclerotherapy.\textsuperscript{1} Treating the underlying cause of the venous hypertension is the best way to prevent future chronic, painful problems.

CONCLUSION

Established CVI with ulceration is already a problem for approximately 1\% of the population.\textsuperscript{2} Considering the risk factors, i.e. being female or pregnant for example, the potential for CVI rises considerably. Taken together with, the prevalence of obesity and an aging population, CVI is likely to become even more prevalent. The most serious consequence of CVI is skin ulceration, which is expensive to treat and slow to resolve. CVI also negatively affects a patient's mobility and comfort, which in turn leads to a reduced quality of life. The delicate nature of the lower extremity venous system potentiates the risk of system failure. Chronic venous hypertension causes inflammation of the microcirculation causing further damage to vessels and the surrounding dermis and adipose tissue. CVI cannot be treated without consistent compression or the physical and financial stress of surgery. All of the above are reasons why awareness is so important and prevention is the key.

For those patients that have current venous hypertension with ulceration, treatment is available at wound care centers worldwide. For providers without easy access to a
Chronic Venous Insufficiency

wound care center, the guidelines for treatment of venous ulcers have been provided. Although these guidelines are certainly not exhaustive, they provide the basics of care that may be used to initiate treatment and heal uncomplicated wounds. Reducing venous hypertension is vital for treatment of CVI with or without ulceration. Compression is the mainstay of treatment but elevating legs while sitting and exercising the calf muscle pump are also important factors. If ulceration is present, there are also wound care issues that will need to be addressed as well as treatment to reduce venous hypertension. Wound care principles such as: debridement of devitalized tissue; infection control, topically or systemically; and choice of dressing to cover wound and maintain appropriate moisture balance of the wound bed. Unfortunately, even with appropriate treatment of venous ulcers, they can take months to heal. Time necessary for treatment is another reason why prevention of CVI is so important.

A PCP's awareness of CVI and its risk factors is the beginning. Assessing for risk factors and initiating preventive strategies for those patients at risk is the next step. Checking patient's legs during the physical exam will alert a PCP to the early signs of CVI. Educating the patient regarding the importance of daily skin care and compression, exercise, and leg elevation to reduce the damaging effects of venous hypertension will allow the patient to be proactive in disease prevention. Consideration for early referral to a vascular surgeon for evaluation and treatment to prevent CVI may be beneficial.

CVI is not considered a life threatening condition and therefore is often ignored as an important health problem (7). Patients and health care providers frequently disregard early signs of CVI. The condition is usually only addressed when it cannot be overlooked any longer. Treatment begins only after severe venous hypertension has caused
permanent damage to the lower extremities. Awareness and prevention can help solve the problem of CVI saving many health care dollars and increasing a patient’s quality of life - the two bottom lines in health care.
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