



# Vascular HealthCare

Leading the way.

## The Guide Wire

NEWSLETTER • JULY 2020

### VENOUS ULCERS

#### What is a venous ulcer?

**Venous ulcers are a consequence of venous hypertension, usually caused by chronic deep or superficial venous insufficiency<sup>1</sup>.**

Until recently, it was believed that venous ulceration was primarily due to deep venous insufficiency following valve failure, (either primary valvular failure, or as a consequence of deep venous thrombosis causing damage to the venous valve), or as a result of failure of the calf muscle pump. However, recent studies have suggested that up to 57% of venous ulcers are due to superficial venous reflux alone, with the deep veins demonstrating normal venous competence <sup>1</sup>.

That said, it is important to note that some ulcers which may appear venous in origin can possibly be caused by other conditions such as rheumatoid arthritis or skin disorders<sup>1</sup>.

#### Pathophysiology

Lower limb venous hypertension is a result of one of two sources. The first is reflux of gravitational origin, also known as hydrostatic pressure. When venous valves fail to coapt, the

weight of the blood presses distally, and the highest pressures generated by this mechanism are expressed at the level of the ankle and foot.

“The second mechanism of venous hypertension is dynamic. The anatomic angulation of superficial to deep perforating veins and their contained valves normally prevent compartmental pressure from being transmitted to subcutaneous tissue and skin. Failure of this mechanism allows intra-compartmental forces to be transmitted directly to unsupported subcutaneous veins and dermal capillaries. There, the effective vessels elongate, dilate and lose their valve competence. Thus, venous hypertension is both hydrostatic and hydrodynamic<sup>2</sup>.”

Subcutaneous tissue and skin are the ultimate targets for chronic venous insufficiency<sup>2</sup>. The underlying cause of ulceration is still unclear, but it is thought to involve changes in the microcirculation of the skin and subcutaneous tissues. Venous hypertension causes an increase in venular and capillary pressure, in turn, leading to local oedema and reduced reabsorption of proteins and fluid from the interstitial tissue spaces. Leakage of red blood cells across the capillary wall and into the

interstitial tissue spaces produces a brawny, brownish pigmentation often associated with venous ulcers. This is due to haemosiderin deposition caused by the breakdown of the red blood cells<sup>1</sup>, and is predominantly seen in the medial lower third of the calf. Pigmentation may be followed by an itching, weeping dermatitis, in turn, possibly progressing to ulceration<sup>2</sup>. Ulceration may be either spontaneous, or as a result of minor trauma. Although the pathophysiology of the ulceration is not clear, it appears to be related to an inflammatory reaction in the tissue, fibrin cuffing and eventual lipodermatosclerosis<sup>3</sup>.

#### Risk Factors

- Previous deep venous thrombosis and subsequent post thrombotic syndrome
- Superficial venous incompetence (either primary, or secondary as a consequence of the above).
- Obesity
- Immobility
- Arthritic conditions (result in reduced movement of the ankle joint, which may lead to failure of the calf muscle pump)<sup>1</sup>.

## Role of Ultrasound

Ultrasound is a useful tool to rule out deep venous pathology which may affect the successfulness of varicose vein treatment<sup>3</sup>, and can be used for the assessment of patients with primary or secondary varicose veins, or the investigation of patients with skin changes and extensive venous ulceration<sup>1</sup>. Early detection and recognition of the pre-ulcerative leg are important factors which influence the effectiveness of treatment and the duration an ulcer may be active for.

## Clinical Appearance

Venous ulceration presents on the CEAP classification table as -

- C<sub>5</sub> - skin changes with healed ulcer
- C<sub>6</sub> - skin changes with active ulcer<sup>4</sup>

Venous ulcers usually vary in size and can be reasonably shallow.

In some cases ulcers may be circumferential, involving a large area of the lower calf.

Often, they become infected with different types of bacteria and in these instances can be extremely painful<sup>1</sup> and produce an odour<sup>5</sup>.

In the absence of infection, venous

ulcers are usually painless.

Ulcers may be large and shallow, with a red base sometimes covered by yellowing tissue (See Figure Two). Exudate fluid can vary in its degree<sup>6</sup>.

Venous ulcers usually demonstrate uneven borders, and the surrounding skin may be tight, shiny, warm and discoloured<sup>5</sup> (See Image One).

Before ulceration occurs, venous hypertension typically presents clinically as chronic leg swelling and ankle pigmentation (in the form of reddish brown pigmentation-

the aforementioned haemosiderin deposition) and is frequently associated with local skin irritation or itching<sup>1</sup>. Ultimately, ankle ulceration in the gaiter zone (located in the lower calf and ankle)<sup>3</sup> occurs.

In this region, the ambulatory superficial venous pressures are the highest, leading to oedema, pigmentation, and ulceration.

The skin, after years of oedema, is difficult to examine for venous incompetence (both clinically and with ultrasonography) because of extensive fibrosis<sup>3</sup>.

### Clinical\*

- C<sub>0</sub> - No clinical sign
- C<sub>1</sub> - Small varicose veins
- C<sub>2</sub> - Large varicose veins
- C<sub>3</sub> - Edema
- C<sub>4</sub> - Skin changes without ulceration
- C<sub>5</sub> - Skin changes with healed ulceration
- C<sub>6</sub> - Skin changes with active ulceration

### Etiology\*

- E<sub>C</sub> - Congenital
- E<sub>P</sub> - Primary
- E<sub>S</sub> - Secondary (usually due to prior DVT)

### Anatomy\*

- A<sub>S</sub> - Superficial
- A<sub>D</sub> - Deep veins
- A<sub>P</sub> - Performing

### Pathophysiology\*

- P<sub>R</sub> - Reflux
- P<sub>O</sub> - Obstruction

"Early application of compression should be performed to correct swelling and progressive scarring and to initiate the healing process by improving the venous microcirculation."

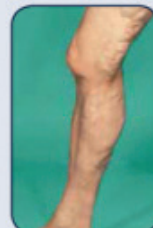
Kistner R. Specific Steps to Effective Management of Venous Ulceration, Supplement to Wounds June 2010.

\*Fronek HS, Bergan JJ, et al. The Fundamentals of Phlebology: Venous Disease for Clinicians, 2004. pg 151.

### Clinical Classifications with examples



C<sub>1</sub> - Telangiectasias or reticular veins



C<sub>2</sub> - Varicose veins



C<sub>3</sub> - Edema and corona



C<sub>4</sub> - Lipodermatosclerosis and eczema



C<sub>5</sub> - Ulcer scar



C<sub>6</sub> - Active ulcer



Image One - Venous Ulcer with obesity as co-factor. Image courtesy of Wound Source<sup>6</sup>.



Image Two - Venous Ulcer. Image courtesy of Wound Source<sup>6</sup>.

## Complications

It is important to remember that some venous ulcers are associated with arterial disease and patients with mixed venous and arterial ulceration can pose a challenging diagnostic problem for treating physicians. Therefore routine measurement of the ABI in all patients with ulceration and risk factors for peripheral artery disease may be recommended to exclude a significant arterial component<sup>1</sup>.

## Treatment

Treatment of superficial venous incompetence, either through ablation (thermal or chemical) or surgical options results in the majority of ulcers healing due to reduction of venous hypertension.

Venous ulcers that are caused by significant deep venous insufficiency are not treated as above, as the underlying venous hypertension will not be corrected. Instead, compression bandaging which reduces oedema and venous hypertension has proved to be an effective method of healing ulcers. Different compression grades can be used dependant on the clinical situation. An ABI of >0.9 is required for the application of four layer compression dressings in order to prevent arterial compromise of tissues under the bandaging<sup>1</sup>.

## Take Home Message

### Predisposing Factors

- History of DVT
- Incompetent perforators
- Varicose Veins
- Obesity

### Associated Changes to the Lower Limb

- Firm "brawny" oedema
- Reddish brown discolouration
- Evidence of healed ulcers
- Dilated and tortuous varicose veins
- Limb may be warm

### Ulcer Location

- Anterior to medial malleolus
- Pretibial area
- Generally lower 1/3 of leg

### Ulcer Characteristics

- Uneven edges
- Ruddy granulation tissue
- No necrotic tissue

### Pain

- Moderate to no pain
- Discomfort relieved by leg elevation

### Surrounding Area

- Leaking oedema may result in maceration, pruritus and scale

### Pulses

- Normal leg and foot pulses

### Compression Bandaging Guidelines

- Compression bandages over padding with/without tubular stretch bandage over compression bandages

Table One- Indicators for the Assessment of Venous Leg Ulcers – Courtesy of Carville 2005<sup>9</sup>.

## Reference List

1. Thrush A & Hartshorne T. *Peripheral Vascular Ultrasound: How, why and when*. 2nd Ed. Elsevier Churchill Livingstone. Philadelphia, Pennsylvania. 2005. pp170-2
2. Dean
3. Zwiebal WJ & Pellerito JS. *Introduction to Vascular Ultrasonography*. 5th Ed. Elsevier Saunders. Philadelphia, Pennsylvania. pp479-80
4. Myers K & Clough A. *Making Sense of Vascular Ultrasound*. Oxford University Press. New York City. New York. 2005. pp211
5. Sobel, M. Venous Ulcers- Self Care. Medline Plus. *U.S National Library of Medicine*. [Internet] 2018. [Cited 23rd April 2020]. Available: <https://medlineplus.gov/ency/patientinstructions/000744.htm>
6. Wound Source. Venous Insufficiency Ulcers. *Kestral Health Information*. [Internet] n.d. [Cited 23rd April 2020]. Available: <https://www.woundsource.com/patientcondition/venous-insufficiency-ulcers>
7. CPD for General Practitioners. Ulcer Assessment. *Revalidation Support Unit (RSU)*. [Internet] n.d. [Cited 24th April 2020]. Available: <https://gpcpd.heiw.wales/clinical/venous-leg-ulceration/ulcer-assessment/>
8. Coville K. *Wound Care Manual*. 5th Ed. Silver Chain Foundation. Osborne Park, Australia. 2005.97



Vascular HealthCare

### Dr Naomi Hunter

B. Med FACP



**Phlebologist, The Hunter Vein Clinic.**

Specialist in non-surgical varicose vein treatments – thermal ablation and sclerotherapy.

**Phone** 4933 0431

**Fax** 4933 0470

**Email** [info@hunterveinclinic.com.au](mailto:info@hunterveinclinic.com.au)

[www.hunterveinclinic.com.au](http://www.hunterveinclinic.com.au)

### Dr Nicole Organ

FRACS (Vascular)



**Vascular and Endovascular Surgeon**

**Phone** 4946 2310

**Fax** 4920 6476

**Email** [surgeon@nicoleorgan.com](mailto:surgeon@nicoleorgan.com)

[www.nicoleorgan.com](http://www.nicoleorgan.com)

### Dr Mathew Sebastian

FRCSEd, MMed (Surg), FRACS (Vascular)



**Vascular and Endovascular Surgeon**

**Phone** 4942 1035

**Fax** 4920 6476

**Email** [msebastian@vascularhealthcare.com.au](mailto:msebastian@vascularhealthcare.com.au)

[www.mathewsebastian.com.au](http://www.mathewsebastian.com.au)

### For More Information



Vascular HealthCare

**Richard Rounsley**

Mobile 0408 558 798

Email [richardr@vascularhealthcare.com.au](mailto:richardr@vascularhealthcare.com.au)

**We look forward to working with you and your patients.**