A leg ulcer can generally be defined as a wound between the knee & ankle joint that is not demonstrating significant progress towards healing in 4 weeks (i.e. reduction of wound size by at least 30%)

'Leg ulcer' is not a diagnosis!
The underlying pathophysiology is the diagnosis

- Most leg ulcers are due to circulatory insufficiency
  - Venous leg ulcer – due to chronic venous insufficiency (CVI) approximately 50-70%
  - Arterial leg ulcer – due to peripheral arterial disease (PAD) approximately 10-20%
  - Mixed leg ulcer – i.e. co-existing CVI and PAD approximately 10-20%
  - Leg Ulcers of other causes 10-20%

Venous Disease
- This is the result of a lack of movement or standing or sitting for long periods of time. This lack of movement causes a pooling of blood in the lower limbs and swelling (oedema)
- Venous thrombosis may occur. This is commonly evident when travelling by car, bus, train or plane for long trips commonly and incorrectly called economy class syndrome.
Venous Disease

Venous ulcers result from the breakdown of the venous circulation of the leg, and are an association of the inability of the leg to force the passage of blood through the various connecting veins via the bicuspid valves by muscular contraction. Venous ulcer may also be the end result of venous stasis, a lack of venous return.

Venous Disease – Clinical Signs

- They are most often found in the gaiter area, the lower 1/3rd of the leg, and where there is a history of varicose veins and oedema. They are usually irregular in shape, not painful, and oedema is often present. The skin is stained around the ulcer area, there may also be other skin changes including eczema, atrophie blanche (white stippled scars on the skin). There may be ankle flare, distended small veins on the medial aspect of the foot present.
**Venous Incompetence - Clinical Signs**
- oedema
- staining - haemosiderin deposition
- lipodermatsclerosis
- lower 1/3 of leg
- often painless
- irregular shape
- may be copious exudate

**Venous Disease - Treatment**
- Identify and treat the underlying disorder to ensure venous disease
- controlling and treating infection
- debride non-viable tissue either autolytically with hydrogels or enzymes or surgically.
- applying the appropriate dressings.

**Diagnosis**
The most essential issue is to ensure a diagnosis of venous disease. It is often the case that if some venous signs are found eg. Oedema then it is assumed that the wound is venous. Peripheral oedema may be caused by a other issues including:
- Heart Failure
- Nephrotic Syndrome
- Liver Disease
- Drugs
  Medications may cause, or exacerbate, peripheral oedema antihypertensive drugs such as calcium channel blockers and direct vasodilators are most frequently implicated

**Venous Disease - Treatment**
- Apply graduated compression bandages or stockings to ulcers if true venous pathology is demonstrated. toe to knee, 30-40mmHg at ankle
- Address factors that may delay wound healing eg. Nutrition, smoking, exercise, working environment eg standing for long periods.
- surgery in some cases
- must exclude arterial involvement

**Venous Disease - Surgical Treatment**

**ATROPHIE BLANCHE**

with complements of Mi-tec Media
Venous Ulcers

- Inverted Champagne Bottle Legs
- Typical Venous Ulcer

Arterial Ulcers

- Arterial ulcers are very painful, especially at night. This is as marked in small ulcer as in larger ulcers. Their edges are sharply defined, and the ulcer is 'punched out'. The base is often covered with slough.

- A history of intermittent claudication (pain on exercise), dependant foot (dusky foot) white on elevation and a history of peripheral vascular disease. The skin is often shiny and friable. Uncontrolled diabetes and smoking are significant factors causing arterial insufficiency. Healing is often slow and may depend on control of the underlying cause.

- Healing is often slow and may depend on control of the underlying cause.
- Wounds in bed-ridden patients, general conditions of the skin in elderly patients which is often associated with malnourishment.
- It is important to note that between 10 to 15 percent of leg ulcers are of mixed aetiology.
Arterial Ulcers

- Management of Arterial wounds usually involves improving blood flow through angioplasty, stenting, bypass grafting.
- Often results in amputation of a digit or limb.

Arterial Ulcers Treatment

Mixed Venous Arterial Ulcers

It is important to note that between 10 to 15% of leg ulcers are of mixed aetiology.

Leg Ulcers are mostly due to either Venous or Arterial Disease about 80%.

However, there is a group of Leg ulcers that are difficult to heal due to a wide range of causes.

About 20%
Simple leg ulcer may develop as complications of the other causes that may ultimately result in leg ulcers include:

- Infections, which may lead to ulceration especially if the necrotising type of bacteria is present.

Leg ulcers may also result in patients with lymphoedema, caused by a reduction in the function of the lymph vessels to drain extracellular fluid.

**Lymph Node**

- What is This?

**Primary Lymphoedema**

1. Primary, not related to a malignancy
   - 1.1 Malformations
   - 1.1.1 Hypoplasia
   - 1.1.2 Lymphangiectasia
   - 1.2 Lymph nodes

2. Secondary, not related to a malignancy
   - 2.1 Traumatic
   - 2.2 Lymphangitis or Lymphangiosis
   - 2.2.1 Bacterial
   - 2.2.2 Fungi
   - 2.2.3 Parasites
   - 2.2.4 Insects
   - 2.2.5 Chemical irritants

3. Tumours:
   - 3.1 Block dissection
   - 3.2 Irradiation
   - 3.3 Sequelae of surgical treatment
     - 3.3.1 Lymph node removal, liposuction, etc.
   - 3.3.2 Stripping veins

4. Self-mutilation (Lymphoedema factitia)
Secondary Causes of Lymphoedema

3. Secondary, caused by malignancy
   3.1 Direct metastasis
   3.2 Intra/lymphatic propagation of tumour
   3.3 Compression of lymphatics
   3.4 Stewart–Treacy syndrome (angiosarcoma)

3.2 Recurrent malignancy (after operation and/or radiation)
   3.2.1 Intra/lymphatic propagation of tumour
   3.2.2 Compression of lymphatics
   3.2.3 Stewart–Treacy syndrome (angiosarcoma)

Skin Changes Lymphoedema

- Thickening of the Skin
- Build up of scale and Keratin
- Worsening hyperkeratosis producing a warty appearance
- Difficult to pick up skin fold between the fingers
- Skin creases deepen
  - Around the ankle
  - Base of the toes
Elephantiasis Lymphoedema

Due to Filariasis (Filarial Worms) from a mosquito, fly or similar insect bite

Post Treatment using a combination of Washing, Oiling, Massage, Diet, Yoga, Ayurvedic Medicine and Bandaging

Indian Case Study

- Vasculitic ulcers may develop as a result of other medical conditions, such as rheumatoid arthritis and polyarthritis, Polycythemia and skin conditions like Scleroderma including infection, eczema and irritant dermatitis, haemorrhage and neoplasia the most common of these being squamous cell and basal cell carcinomas.
- Also inflammatory like wounds eg. pyoderma gangrenosum

Vasculitic ULCERS

Rheumatoid Arthritis
Systemic Lupus Erythematosus SLE
Scleroderma
Vasculitic like Ulcer
Pyoderma Gangrenosum
VASCULITIS

Definition:
An inflammation of blood vessels commonly associated with auto-immune or immune mediated disease.
The vessel lumen is usually compromised.

VASCULITIS [Mechanism]

Vasculitis

- Affects blood vessels of any size
- In many cases the etiology is unknown.
- Approximately 20 different disorders
- No universally accepted classification system

Pathogenesis

- Immune Complex Deposition
- Complement Activation and Consumption
- Lymphocytic Infiltration
- Granulomatous Inflammation and Giant Cell Formation

Major Histopathological Features

- Fibrinoid necrosis of the affected vessel wall and inflammatory cells within the vessel wall.
- Extravasation of RBC’s is frequent and responsible for palpable purpura.

Minor Histopathological Features

- Thrombosis of vessel lumen
- Inflammation and damage to surrounding tissue
- Direct immunofluorescence staining shows IgG, IgM, IgA and C3 deposition within the vessel wall.
Ulcer [RA Patient]

is this a Venous Ulcer?

Ulcer [RA Patient]

NO This is Vasculitis

Classic Vasculitic Ulcer

Purple Halo

Dark margin

Auto-Immune Ulcer (SLE)

Clinical Presentation

- Distribution of Skin lesions
  - Often start on dependent areas, become generalized
  - Hydrostatic force on the post-capillary venules leads to deposition of immune complexes in these sites

Clinical Presentation

- Course of skin lesions
  - Depends on the etiology
    - Acute - resolving within several days to weeks
    - Chronic / recurrent - persisting from months to years.
  - Successive crops, evolving from papules and nodules to palpable purpura
  - Significant post-inflammatory hyperpigmentation
Classification of Systemic Vasculitis
Based on Caliber of Blood Vessel

- **Small Vessels**
  - Leukocytoclastic vasculitis
  - Henoch-Schönlein Purpura
  - Urticarial vasculitis
  - Mixed cryoglobulinemia
- **Small to medium vessels**
  - Buerger Disease
  - Central Nervous System Vasculitis
  - Wegener’s granulomatosis
  - Churg-Strauss syndrome
  - Microscopic polyangiitis
- **Medium vessels**
  - Kawasaki syndrome
  - Polyarteritis nodosa
- **Large vessels**
  - Giant Cell Arteritis
  - Temporal arteritis
  - Takayasu’s arteritis
- **Vessels of any size**
  - Behcet’s Disease
  - Vasculitis related to connective tissue disease
    - Rheumatoid Vasculitis
    - SLE
    - Primary Systemic Sclerosis
    - Sjogren’s syndrome
    - Polymyalgia Rheumatica

Clinical Presentation

- **Morphology of Skin Lesions**
  - Petechiae
  - Non-palpable Purpura
  - Palpable Purpura
  - Ecchymoses
  - Erythematous macules
  - Papules
  - Nodules
  - Vesicles
  - Pustules
  - Bullae
  - Eschar / Gangrene
  - Ulcers
  - Urticaria
  - Livedo reticularis
  - Nail fold telangiectasia

The many faces of Vasculitis

Livedo vasculopathy

Livedoid vasculopathy (LV) is a coagulation disorder classified as a vasculopathy different from inflammatory vasculitis. The diagnosis of LV was based on the following established clinical and histological criteria. Clinical criteria included painful purpuric macules, typically on the lower extremities, that ulcerated and then slowly healed with atrophic stellate scars and surrounding telangiectasia.

Systemic Lupus Erythematosus

SLE is a systemic auto-immune disorder characterised by circulating auto-antibodies to nuclear and cytoplasmic antigens

The disease is female predominant and is potentially fatal. The skin lesions may be a defined ulcer that is difficult to heal due to the systemic Drug treatment of SLE

Including Steroids, Cytotoxic agents eg. Cyclophosphamide and Azathioprine

All having a retarding effect on healing
Systemic Lupus Erythematosus

The skin lesions may be scarring plaques.

Systemic sclerosis (Scleroderma)

Scleroderma is a chronic multi-system disorder of unknown aetiology characterised by thickening of the skin caused by an accumulation of connective tissue and by involvement of visceral organs including the GI tract, lungs, heart and kidneys. Vascular abnormalities of the microvasculature are a prominent feature. Scleroderma may appear as pale indurated plaques often surrounded by a violaceous halo.

Diagnosis is by examination and blood tests. Treatment of scleroderma is difficult as there is no cure. The use of d-penicillamine, immuno-suppressives, and cytotoxic drugs e.g. Azathioprine, Methotrexate, Cyclophosphamide are used. Non-steroidal anti-inflammatory drugs for pain.

Topically as with many of the unusual Skin ulcer covered a moist environment With Hydrogels and the use of Foam Dressings eg. Lyofoam or Allevyn is Indicated.
Pyoderma Gangrenosum

PG is an inflammatory skin disease resulting in painful, enlarged, ulcerated nodules. The ulcer is irregular, raised, with reddish borders and undermined edges with necrotic base. A Rare condition destructive, non-infective ulceration of the skin. It is associated with Inflammatory Bowel diseases and immune system abnormalities. The ulcers are painful rapidly Enlarging with undermined bluish and purplish red margins.

Pyoderma Gangrenosum

PG is difficult to diagnose and is Mostly obtained by exclusion. Wound Biopsy will often help to exclude other cases. It is often the Case that if a biopsy is taken that the Wound will enlarge (Pathergy).

Pyoderma Gangrenosum

PG is Difficult to treat this involves Pain management, Moist Environment, Systemic use of Steroids, Cyclosporin, Dapsone. Other topical treatments include the use of topical monoclonal antibodies, and topical immuno-suppresives eg Tacrolimus.
Pyoderma Gangrenosum (RA)

Pyoderma of the Face

Infliximab in pyoderma gangrenosum

“Improvement of idiopathic pyoderma gangrenosum during treatment with anti tumor necrosis factor alpha monoclonal antibody”

Dini V, Romanelli M, Bertone MS et al.

Case

The patient is a 85yr Lady with a 5 year history of multiple bilateral leg ulcers. She was referred to the wound clinic and a diagnosis of PG was made. She was treated with steroids, and Mycophenolate (an immunosuppressant) for three months with little Progress.

Permission was obtained for a trial use of s/c Adalimumab (Humira) 40mg second weekly. Her pain was significant
Case 09/04/2009
Her largest wound on first presentation

Case 08/10/2009
First day of Humira Injections

Case
Within four weeks there was a marked reduction in pain and a significant reduction in wound size. The s/c administration of Humira was continued secondly weekly.

Case 28/01/2009

HAEMATOLOGICLAL
Polycythemia Rubra
Raynaud’s Disease

Polycythemia
This is a Haematologic disease resulting in a number of factors that may result in a Leg ulcer. The wounds are purpuric lesions, painful and difficult to heal. On healing they tend To leave an irregularly shaped white scar. The cause may be cryofibrinogenemia and Antiphospholipid syndrome.
Polycythemia (Antiphospholipid Syndrome)

This woman with systemic lupus erythematosus had the antiphospholipid syndrome. In addition to the leg ulcer, she had a history of recurrent abortions. Sclerosed was used in the hope that the thrombolytic activity of this medication would lead to the intravascular fibrin thrombosis of this condition. Her pain resolved and the ulcer improved considerably but did not heal completely with unsclerosed therapy.

Polycythemia - Cryofibrinogenemia.

This painful, non-healing ulcer in a middle-aged woman with systemic lupus erythematosus was due to cryofibrinogenemia. She had detectable plasma levels of cryofibrinogen, and intravascular fibrin thrombi were present histologically. The area to be biopsied is marked to ink on the superior border of the ulcer. The ulcer healed with unsclerosed therapy.

Raynaud's Syndrome Ulcer

Complication of Leg Ulcers
- Neoplastic Development
- Calcification
- Cellulitis
- Infection
- Haemorrhage
- Dermatitis/ Eczema
- Suppuration
- Gangrene

MISCELLANEOUS

Post radiotherapy for the treatment of cancer may result in a painful desquamation burn of the skin through which the radiation passes

MISCELLANEOUS

SELF-INFLICTED ULCER (FACTIOUCIOUS)
A WOUND THAT REMAINS UNHEALED DUE TO INTERFERENCE BY THE PATIENT OFTEN A PSYCHO-SOCIAL PROBLEM
IV DRUG USERS wounds Results from extravasation of the drug into the soft tissue resulting in a Tissue Necrosis.
Neuropathic Ulcer in Non-Diabetic

Bullous pemphigoid

- Immunologic disease of the skin (Type 2 hypersensitivity)
- Cytotoxic Abs to basement membrane
- Tm:
  - Topical corticosteroids
  - Oral corticosteroids
  - Tetracyclines
  - Aspirate blisters + wet dressing (towel or gauze)

Blistering disorders

- Diverse group of diseases characterised by fluid accumulation
- Caused by damage to epidermis and upper dermis
  - Bullous impetigo
  - Insect bites
  - Burns
  - Drug reaction
  - Bullous pemphigoid
  - Pemphigus vulgaris
Epidermolysis Bullosa

Fig. 22.7 Level of blister in epidermolysis bullosa at the epidermal–dermal junction.

Epidermolysis Bullosa

Fig. 22.9 Autosomal recessive dystrophic epidermolysis bullosa: note large blood-filled blisters. Scarring has led to fixed deformity of the fingers and loss of nails.

E B Treatment

GOUT

Uric Acid Tophi
CALCINOSIS

Cutaneous Calciphylaxis