Philippine Society of Cutaneous Medicine, Inc.

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Skin ulcerations of the legs have a significant impact on the quality of life and work productivity of an individual. The long-term care and costs of these chronic wounds can create serious psychological and social problems as well. Hence there is a great need for us to understand the pathophysiology that has led to the development of new approaches to the management of lower extremity ulcers.

There are three main types of lower extremity ulcers. They are venous, arterial, and neuropathic. Venous ulcers constitute about 80% of all leg ulcers, while foot ulcers are more likely to be arterial or neuropathic in nature. Co-existing rheumatologic disease occurs in 10% to 15% of patients, whereas diabetes mellitus is present in 5% to 12% of patients. Less commonly, trauma, pressure, and infectious agents are causes of leg ulcers.

EPIDEMIOLOGY

Venous leg ulcers have an overall prevalence ranging from 0.06% to 2%. There is slight female predominance, with a female-to-male ratio of 1.6:1. Venous ulcers are more common with increasing age, with a peak prevalence between 60 to 80 years. It has been reported that 72% of persons have their first ulcer by age 40 and 15% before 30 years of age. Recurrence rate is as high as 72%.

PATHOPHYSIOLOGY

“Chronic venous insufficiency” which is synonymous with venous hypertension is the venous pressure deep in the system upon ambulation that may either fall minimally or not at all. It may occur by one of four pathophysiologic mechanisms: 1) dysfunction of valves in the superficial and/or communicating veins because of congenital or acquired incompetence 2) dysfunction of valves in the deep system because of congenital absence, inherent weakness, or thrombotic damage 3) deep venous outflow obstruction rather than valvular incompetence and 4) muscle dysfunction and calf muscle pump failure from inflammatory conditions of the joints or muscles, fibrosis, or neuropathies. There is no general rule on the sequence of pathogenic steps leading from venous hypertension or venous insufficiency to venous ulceration.

PATIENT HISTORY

The lack of specific symptoms characterize the history of patients with venous ulcerations. The discomfort varies from patient to patient. The surface area of the ulcer does not correlate well with the presence of pain. It has been noted that deep ulcers, especially around the malleoli, or small venous ulcers surrounded by atrophic blanche are the most painful. Patients complain of swelling and aching of the legs, often worse at the end of the day, exacerbated by dependency and improved leg elevation. If the pain is so severe, might as well consider the possibility of either infection or a different cause of the ulceration, such as vasculopathy. Odor and copious drainage from the wound and pruritus of the surrounding skin are common associated findings. The patients can have a history of phlebitis, deep vein thrombosis, or silent thromboses, commonly during or after pregnancy or surgical interventions.

RISK FACTORS

Age, obesity, history of significant leg injury as broken leg, stab or gunshot wound or a crus injury as well as phlebitis are the potential risk factors for chronic venous insufficiency.

The type of employment and the lifestyle can alter the course of venous ulcerations. Patients with jobs requiring long hours of standing tends to heal slowly.

GENERAL MANAGEMENT OF LEG ULCERS

The primary goals of the treatment for Chronic venous insufficiency are: diminution of edema, alleviation of pain, improvement of lipodermatosclerosis, healing of ulcers and prevention of recurrence.

A. Bed rest with leg elevation (above the heart level for at least 30 minutes 3-4x a day)
B. Compression therapy (Cornerstone of therapy)
   First Phase
   1. Rigid inelastic bandages
      Example.
      A. Unna boot (moist zinc-impregnated paste bandage)
      B. Short stretch bandage
   2. Long-stretch bandage - a type of elastic system
CPM 5TH EDITION

WOUND HEALING - LEG ULCERS

(higher resting pressure than short stretch bandage)
3. Multi-layered bandage system
4. Pneumatic devices based on an intermittent inflation of air bags
5. Orthotic compression
Second Phase
1. Elastic material ex. Graded compression stockings or compression bandages

C. Wound Debridement
1. Autolytic (Occlusive dressings) - hydrogels, alginites, foam, films, hydrocolloids
2. Chemical - (Enzymes debriding agents) - collagenase, papain, trypsin
3. Mechanical
   a. wet to dry dressings
   b. hydrotherapy
   c. irrigation (or whirlpool)
   d. destronomers
4. Growth factors -
   a. Human recombinant epidermal growth factor
   b. Platelet derived growth factor (for diabetic ulcer)
5. Surgery
   a. Graft (Split thickness skin graft)
   b. Pinch grafting
   c. Superficial vein surgery (ligation or sclerosis of the long and short saphenous system, with or without communicating vein ligation or sclerosis)
   d. Radical excision of the ulcer bed, the fibrotic suprafascial tissues and the diseased superficial and perforating veins

D. Topical Antibiotics
Use of topical antibiotics is controversial. Cadexomer-iodine preparation has been recommended.

E. Systemic Therapy
1. Stanozolol - androgenic steroid with fibrinolytic properties
2. Ifetroban, a thromboxane receptor antagonist
3. Pentoxifylline - to decrease leukocyte adhesion of the vascular epithelium
4. Aspirin - inhibits platelet aggregation
5. Daflon - a micronized and purified flavonoidic fraction to decrease the white blood cell plugging to endothelial cells (1000 mg per day)
6. Sulodexide - heparin like molecule with profibrinolytic and antithrombotic activity

New therapies
1. Tissue engineered skin equivalent (Apligraft)

2. Topical application of granulocyte macrophage colony stimulating factor
3. Non contact radiant heat bandage
4. Monochromatic infrared energy

CONCLUSION

We should all remember that we are dealing with a patient and not just a leg! The medical history will give vital clues as to the origin of the ulcer. We have to look for the presence of Diabetes Mellitus, major operations like childbirth, and other signs like phlebitis, leg injuries, malignancy, ankle and hip arthrodesis, heart failure, hypertension, obesity, hypertension, immobility and allergies. Social and psychological factors like motivation, isolation, psychological dependence, depression, loneliness, anxiety, poverty, nutrition and ignorance if left untreated will lead to treatment failure.
## Clinical Presentation: The Three Common Types of Leg Ulcers

<table>
<thead>
<tr>
<th>Venous</th>
<th>Arterial</th>
<th>Neuropathic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cause</strong></td>
<td>Postphlebitic Syndrome Arteriovenous Shunt</td>
<td>Large vessel: Arteriosclerosis obliterans; thromboangiitis obliterans Small vessel: Raynauds Phenomenon; Vasculitis, Atherosclerosis emboli</td>
</tr>
<tr>
<td><strong>Ulcer location</strong></td>
<td>Medial malleoulos, trauma or infection may localize ulcers laterally or more proximally</td>
<td>Distal, over bony prominences; trauma may localize ulcers proximally</td>
</tr>
<tr>
<td><strong>Ulcer appearance</strong></td>
<td>Shallow, irregular borders; base may be initially fibrinous, but later develops granulation tissue</td>
<td>Pound or punched-out, well-demarced border, fibrinous yellow base or true necrotic eschar; bone and tendon exposure may be seen</td>
</tr>
<tr>
<td><strong>Characteristics</strong></td>
<td>Red base of granulation tissue; surrounding pigmentation, induration, edema, warm foot</td>
<td>Large Vessel: Black or grey base; shallow, irregular; no granulation tissue; cold foot with dependent rubor Small Vessel: Irregular, inflamed edges, whitish base</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>Absent to mild</td>
<td>Large vessel: Severe Small vessel: Severe</td>
</tr>
<tr>
<td><strong>Physical</strong></td>
<td>Varicose veins, leg edema, atrophie blanche, dermatitis lipodermatosclerosis, pigmentary changes, purpura</td>
<td>Loss of hair, shiny, atrophic skin, dystrophic toenails, cold feet, femoral bruitt, absent or decreased pulses, prolonged capillary refilling time</td>
</tr>
<tr>
<td><strong>Frequent symptoms</strong></td>
<td>Pain, odor, and copious drainage from the wound, pruritus</td>
<td>Claudication, resting ischemic pain</td>
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<td><strong>ABI</strong>&lt;br&gt;Ankle to Brachial Index (bl. pressure index)</td>
<td>&gt;0.9</td>
<td>ABI &lt;0.7 suggests arterial disease; calcification of vessels gives falsely high Doppler readings</td>
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<tr>
<td><strong>Risk Factors</strong></td>
<td>Deep venous thrombosis, significant</td>
<td>Diabetes, hypertension, cigarette smoking,</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>Allergic Contact Dermatitis, Cellulitis</td>
<td>Gangrene</td>
</tr>
<tr>
<td><strong>Treatment Pearl</strong></td>
<td>Compression therapy, leg elevation</td>
<td>Pentoxifylline, vascular surgery assessment if necessary if ABI &lt;5</td>
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Drugs Mentioned in the Treatment Guideline

This index lists drugs/drug classifications mentioned in the treatment guideline. Prescribing Information of these drugs can be found in the Philippine Pharmaceutical Directory (PPD) and the Philippine Pharmaceutical Directory Review (PPDr).

Systemic Therapy

**Diosmin/Hesperidin**
- Daflon 500

**Aspirin**
- Asaped
- Aspilets
- Astrix
- Bayer Aspirin
- Cor-30
- Cortal

**Pentoxifylline**
- C Vex
- Trental

Topical Antibiotics

**Bacitracin/Neomycin sulfate/Polymyxin B sulfate**
- BNP Ointment
- Trimycin

**Framycetin sulfate**
- Sofra-Tulle

**Fusidic acid/Sodium fusidate**
- Fucidin Cream/Ointment

**Gentamicin sulfate**
- Bactiderm Ointment
- Garamycin 1% Cream/Ointment

**Mupirocin**
- Bactroban

**Sodium fusidate**
- Fucidin Intertulle
- Fusoderm 2% Ointment

Occlusive dressings

**Deproteinized calf’s blood extract**
- Solcoseryl

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