# Pathophysiology, clinical assessment, and investigations involving leg ulcers

Sir,

The article venous leg ulcers: Pathophysiology and classification by B Vasudevan[1] was interesting, and the author has dealt very eloquently with the subject of pathophysiology, classification, clinical assessment and investigatory approach to the patient with a leg ulcer. There are many causes for leg ulceration. However, the majority (>90%) of chronic leg ulcers have a vascular etiology, with chronic venous hypertension thought to be the primary cause of around 70% and a significant contributory factor in a further 15% that has mixed arterial and venous etiological factors. Approximately, 5% occur due to arterial insufficiency. The rest may be due to diabetes, vasculitis, malignancy, hematological disorders, rheumatoid arthritis, pressure and other trauma.[2]

The pathophysiology of venous ulceration, including the clinical, etiologic, anatomical and pathophysiological (CEAP) classification, so important for clinical assessment and

management, has been discussed in detail by the authors. A brief mention of pathophysiology of other common ulcers needs to be made. Arterial leg ulcers occur as a result of reduced arterial blood flow and subsequent tissue perfusion. Three mechanisms involved in the pathophysiology are (a) extramural strangulation, (b) mural thickening or accretion, and (c) intramural restriction of blood flow.

Diabetic foot ulcers are common and estimated to affect 15% of all diabetic individuals during their lifetime. The etiology of diabetic foot ulcers usually has many components. Peripheral neuropathy has been demonstrated to be the single most common contributing cause of foot ulceration. Ulceration can be attributed to a triad of peripheral neuropathy, biomechanical deformity, and superimposed minor trauma.[3] In general, lack of sensation from neuropathy is responsible for unrecognized repetitive trauma and loading that leads to skin and soft tissue breakdown, creating an entry point for infection. Therefore, understanding the cause, presentation, and treatment of diabetic peripheral neuropathy is paramount for prevention of diabetic lower extremity ulcerative disease and amputation. To add to the woes, an inflammatory environment contributes to diabetic microangiopathy, worsening local ischemia.[4] Other factors in ulceration are trauma, deformity, callus formation, and edema.[5]

Pressure ulcers are as their name implies, caused primarily by unrelieved pressure. They usually occur over bony prominences such as the sacrum, trochanter or the heel but can occur on any part of the body subjected to pressure. They develop due to the compression and localized damage of skin and the underlying tissue between a bony prominence and an external surface.

A detailed history may provide clues to the etiology of the ulcer and should be confirmed by physical examination and appropriate investigations. Venous ulcers most commonly occur above the medial or lateral malleoli. Arterial ulcers often affect the toes or shin or occur over pressure points. Neuropathic ulcers tend to occur on the sole of the foot or over pressure points.

Ankle brachial pressure index is of immense value, as has been aptly brought out by the authors. The newer modalities of investigations include color duplex ultrasound scanning which has become the gold standard for evaluation of venous obstruction, but is also used to assess the location and extent of reflux in venous ulcers. A lot of stress is often laid on wound swab cultures and immediate initiation of "appropriate" systemic antibiotics. The clinician should desist from such deleterious actions. Antibiotics are to be started only if the patient is febrile and exhibits overt signs of sepsis that may include leukocytosis and in the peri-operative period to cover against a bacteremia following debridement. A quantitative bacterial culture is more specific and should be performed once wound infection is suspected.[6] This is performed by curetting or biopsying the bed of the ulcer. The quantitative biopsy is the current favorite for assessing microbial pathogens within the wound.[6,7] Quantitative biopsies containing $<10^5$ organisms/g of tissue are considered significant, and systemic antibiotic therapy should be then considered. Future applications in investigating the patient may include gene variant analysis in patients with venous leg ulcers. This implies that the high-risk minority of patients could be identified in advance by means of a simple blood test that could act as a genetic screening device.[8]

An ideal management plan for patients with leg ulcers should involve an early strategic and coordinated approach to delivering the correct treatment option for each individual patient, based on the accurate assessment of the underlying pathophysiology.

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