

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

LEG ULCERS

METEOR CRATER,
ARIZONA, USA



UPDATED CHAPTER
FEBRUARY 2023

‘Ulcer’ Full thickness loss of epidermis and some dermis, which will heal with scarring

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

AUTHOR: DR BRIAN MALCOLM,

BSc, MBChB, MA, DRCOG, DPD, DCH, Dip Derm (Glasg), FRCGP.

ASSOCIATE SPECIALIST, NORTH DEVON HEALTHCARE TRUST.

FORMER GENERAL PRACTITIONER AND COMMUNITY DERMATOLOGIST, DEVON AND DORSET.

CHAPTER ONE: THE ECZEMAS

CHAPTER TWO: PSORIASIS

CHAPTER THREE: SKIN INFECTION AND INFESTATION

CHAPTER FOUR: SKIN MALIGNANCY

CHAPTER FIVE: LEG ULCERS

CHAPTER SIX: ACNE

CHAPTER SEVEN: URTICARIA AND RELATED ALLERGIC DISORDERS

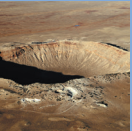
A SYSTEMATIC APPROACH TO DIAGNOSING SKIN CONDITIONS

A PREQUEL TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

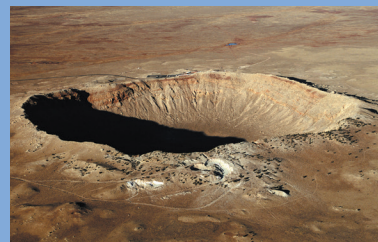
CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASES IN DERMATOLOGY

A SUPPLEMENT TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

TESTS AND TECHNIQUES – WHICH, WHEN AND WHY? A SEQUEL TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE



Front cover image: Meteor crater in the rolling plain of the Canyon Diablo region, 19 miles (30 km) west of Winslow, Arizona, USA. It is 4,000 feet (1,200 m) in diameter and about 600 feet (180 m) deep inside its rim, which rises nearly 200 feet (60 m) above the plain. (Source: *Encyclopaedia Britannica*.)



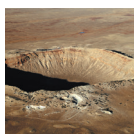
CHAPTER FIVE: LEG ULCERS	
Leg ulcers	1
Pathogenesis of leg ulcers	2
Assessment	3
Examination	4
Baseline investigations	5
Treatment	5
CRITERIA FOR REFERRAL	10
TEACHING POINTS	11
REFERENCES	11
APPENDIX 1 – LAPLACE’S LAW	12



“Your mission if you choose to accept it is to enthuse GPs about leg ulcers. This tape will self destruct in five seconds. Good Luck!”

LEG ULCERS

This is how I chose to start a teaching workshop on leg ulcer management. A cheap stunt some might say, but underlying it is my profound belief that the vast majority of doctors have abdicated responsibility to become involved with leg ulcer management, and see their role as minor and advisory. The reason for the present status quo where nurses are almost wholly responsible for chronic wound management in the community are manifold; some apparent, some more obscure and lost in the mists of time. Ulcers are associated with dressings, dressings with nurses and so on. Ulcer management is often regarded as unrewarding and unpleasant. Why, however, should leg ulcer management be any different say from peptic ulcer management? Both represent a medical condition where there is a discontinuity in a specialised physiological barrier which is multifactorial and requires full assessment. The model of care is very similar, yet one is traditionally a doctor's remit and the other a nurse's. Venous leg ulcers are recognised as a major cause of morbidity and decreased health related quality of life.¹ I make no apology therefore, for including this subject as a fundamental in the Core Tutorials series; perhaps in some small way it may help redress the prevailing imbalance both in knowledge of and attitude to leg ulcer management!



Let us now look at why this area of medicine is so important, particularly to those of us involved in primary care.

- 3.8 million patients with wounds managed in the NHS annually.²
- The annual prevalence in adults of venous ulceration is 2%, an increase of 71% between 2012 and 2018.²
- The cost to the NHS of treating wounds is estimated at around £8.3 billion annually,² costing over £7000/patient/annum and increasing at 8-9% per annum.¹
- 81% of costs are incurred in the community.²
- Wounds and dressings will perpetually appear near the top of the cost list in the regular prescribing figures for virtually all GPs.

PATHOGENESIS OF LEG ULCERS

One fundamental principle has to be established before we proceed. A leg ulcer is not a diagnosis but a manifestation of an underlying disease process; 95% of patients have at least one co-morbidity with a mean of 4.1 comorbid conditions.¹ This is why we doctors must continue to be involved. It is in this sphere that the skills and training to look at the greater picture through our 'wide-angled lenses' and not just focus on the leg ulcer, but the 'patient WITH the leg ulcer'. I will develop this concept more under the section on Assessment.

It is important to define what we mean by a venous leg ulcer. Definitions vary from an area of epidermal discontinuity lasting in excess of 2-6 weeks, occurring as a result of venous hypertension and calf muscle insufficiency. Such ulcers will comprise the majority estimated at between 70-80% of all leg ulcers, 10% will be of an arterial aetiology and 10-20% of a mixed aetiology. Recent research demonstrates that a diagnosis was not recorded in 25% of cases!

Although many theories abound about the pathogenesis of venous ulceration at a cellular level, there is a general agreement that the fundamental problem is one of failure of the calf muscle pump due to venous incompetence, paralysis or immobility. Valve failure in either of the deep or superficial venous systems allows reflex of blood at high pressure into the vulnerable superficial veins and capillaries. This results in chronic venous hypertension which, if not corrected, leads inexorably to the process termed lipodermatosclerosis, consisting of both fibrin and haemosiderin deposition, induration, venous flare and atrophie blanche, the term used for the stellate macular scars as a result of capillary damage and very characteristic of this pathological process. The end result of the chronic process is the classic 'inverted champagne bottle' or 'bowling pin' leg. Arguments about the exact nature of the microcirculatory impairment in venous disease may appear obscure and academic, but more effective treatment must surely follow once pathogenesis is more clearly defined.

The need for high quality research is self evident!



ASSESSMENT

A number of good comprehensive assessment tools exist; these are usually administered by nurses. Links to a doctor with a clear understanding of leg ulcer pathology are often tenuous at best. Although excellent local models exist, in other areas the lines of responsibility often remain fudged and there is evidence of suboptimal practice, inadequate diagnosis, poor recognition and management of co-morbidities and a lack of education and training for health professionals.¹ This, I feel, is partially due to the fact that leg ulceration is multi-agency, involving district nurses, practice nurses, residential nursing homes, community clinics, tissue viability specialists, physicians, vascular surgeons, general surgeons, plastic surgeons, chiropodists, dermatologists, care of the elderly physicians, general practitioners, Uncle Tom Cobley and all! There are many hiding places for the unenthused!

All assessment tools should address the following:

Relevant past and present medical history to include:

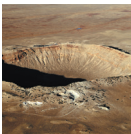
- Trauma
- Deep vein thrombosis
- Pregnancy
- Positive family history of leg ulceration
- Ischaemic heart disease
- Peripheral vascular disease
- Diabetes
- Congestive cardiac failure
- Thyroid disease
- Rheumatoid arthritis
- Autoimmune disease
- Inflammatory bowel disease
- Neoplasia
- Haemoglobinopathies
- Contact allergy
- Radiotherapy

Social history to include:

- Smoking (healing rates of 38% versus 55-58% in non/ex-smokers)
- Nutritional status
- Mobility
- Foreign travel (if relevant)

Drug history – especially:

- Non-steroidal anti-inflammatories
- Corticosteroids + other immunosuppressants
- Beta blockers
- Nicorandil



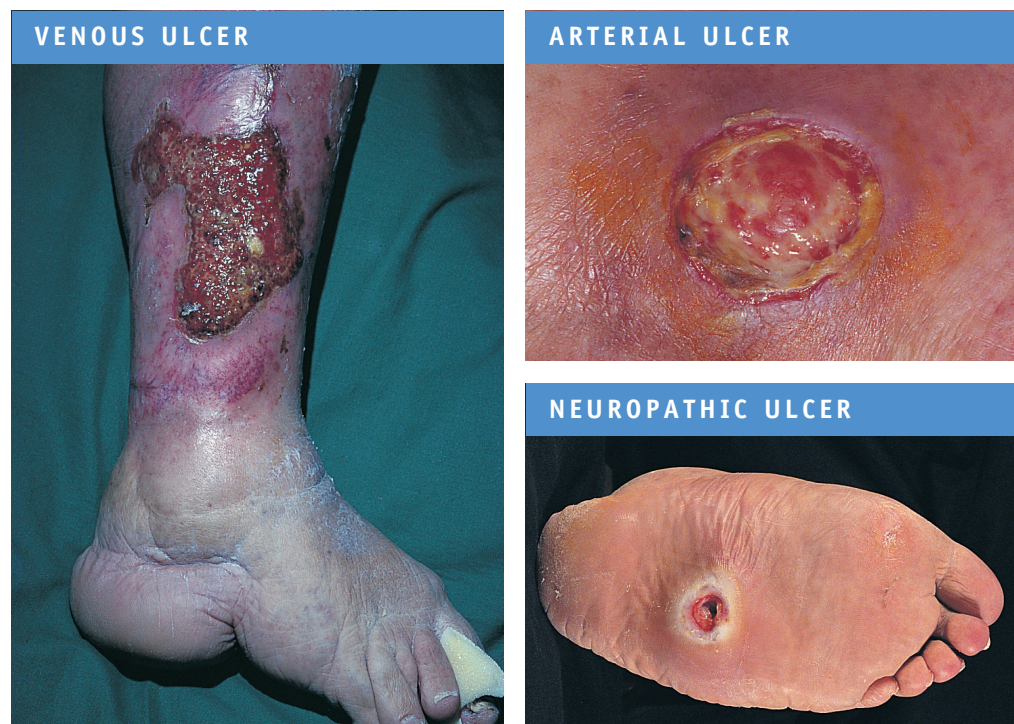
EXAMINATION

This requires very little sophisticated equipment in the community setting. Indeed, the physician or appropriately trained nurse can make a very accurate assessment of the type of ulcer on clinical grounds alone. Examination should be comprehensive to exclude previous undiagnosed underlying pathology e.g. evidence of anaemia, CCF, obesity, arthritis, diabetes, thyroid dysfunction etc. Traditionally, this is the doctor role but how many of us are involved in the assessment of leg ulcers? History and examination specific to leg ulcers includes:

1). **Site** – 88% of venous ulcers occur in the so called ‘gaiter’ area. Most commonly on the medial aspect; however, extensive venous ulcers can extend to adjacent areas of the leg. If an ulcer lies exclusively outside of the gaiter area, then you must question whether it is of venous origin. Arterial ulcers commonly occur on the dorsum or plantar aspect of the foot or on the toes.

2). **Appearance** – look for the changes of lipodermatosclerosis previously described. It would be very unusual to have a venous ulcer without background changes of venous hypertension involving the surrounding skin. Venous ulcers are normally ragged, shallow and sloping in contrast to the deeper punched out so-called ‘cliff edge’ appearance of arterial or vasculitic ulcers. Raised, rolled edges may be indicative of malignancy. Is there evidence of arterial compromise – pallor, loss of hair, nail dystrophy, coldness, poor capillary return? Are the peripheral pulses palpable?

NB: The dorsalis pedis pulse is congenitally absent in 10% and impalpable in a further 10% of cases.

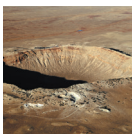


3). **Symptoms** – is the ulcer painful? Even when extensive, venous ulcers can be relatively painless unless infected, although patients do report pain as a symptom in up to two thirds of cases, while arterial ulcers are characteristically painful especially on elevation of the leg. An important exception is the neuropathic ulcer most commonly associated with diabetes.

BASELINE INVESTIGATIONS

Full blood count, U+Es, TFTs and urinalysis would suffice as a basic minimum with other tests as indicated from the medical history and examination. There is no place for routine swabs. These should only be taken when infection or presence of a significant biofilm is clinically evident, either by excessive exudate and malodour, sudden deterioration or acute onset of pain or cellulitis. There must be a clear distinction between infection and contamination defined as the presence of bacteria without multiplication and colonisation defined as the presence of bacteria with multiplication, but no host reaction. Swabs should be taken from the deep base of the ulcer after appropriate cleaning/debridement.

Best practice requires a Doppler assessment by an adequately trained professional, preferably within six weeks of the onset of the ulcer. The equipment is inexpensive and should be readily available to any health professional dealing with ulcers. This contrasts the brachial and ankle pressure which in healthy individuals should equate. A reading of 1 or above indicates normal arterial flow; 0.8 would be the lowest level that full compression could be considered safe. Below this reading there is likely to be significant arterial disease and a vascular opinion should be considered. Modified compression can be used in experienced hands. Spuriously high and falsely reassuring Doppler indices can be obtained in poorly compressible, calcified vessels most commonly evidenced in diabetics. The paradox of leg ulcer treatment is that the best possible treatment for venous ulcers is the worst possible treatment for arterial ulcers, and the road to effective ulcer healing is littered along the way with avoidable medicolegal catastrophes! Worryingly however, only 15% of venous leg ulcers have a Doppler assessment recorded and of the 85% with no recording, 29% were treated with compression!²



DOPPLER ASSESSMENT

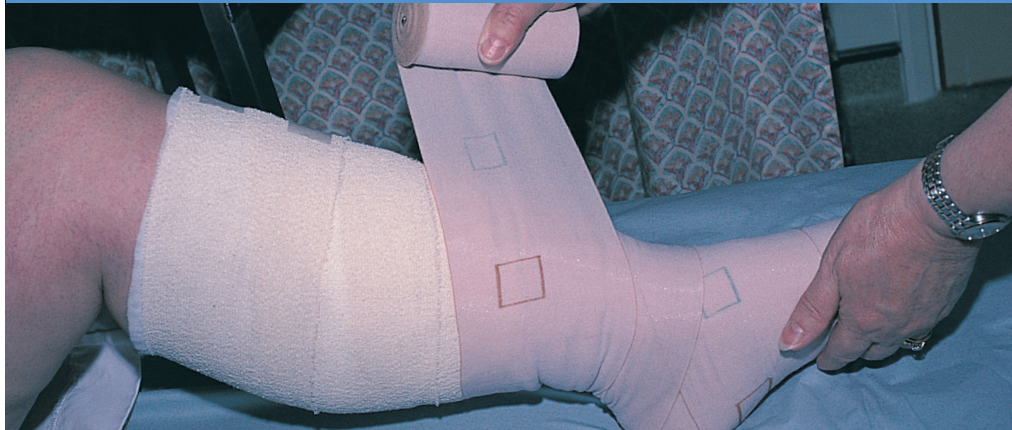


TREATMENT

The four cornerstones of wound management are:

- 1). The definition and treatment of underlying cause
- 2). Strict control of factors affecting healing
- 3). Appropriate dressings
- 4). Maintaining successful wound healing

LAYER COMPRESSION BANDAGING



Compression treatment, either in the form of bandaging systems or hosiery, is the accepted first line treatment for venous leg ulcers occurring in the absence of significant arterial pathology. No one bandaging system is clearly superior although evidence favours high compression and multi-component systems over low compression systems.^{3,4} Four layer systems appear to have an edge over short stretch bandaging⁵ but the quality of research in this important disease area remains poor and well designed randomised controlled trials incorporating economic evaluation are long overdue to arrive at optimal strategies and best components. The dangers of inappropriate compression have already been mentioned. One study reported 147 cases of compression damage over a five year period; seven of which required arterial reconstructive surgery and 12 amputation.⁶ Inappropriate compression can lead to pressure necrosis even in the absence of arterial disease, especially in patients with abnormally narrow ankles or thin calves.

The gold standard was the Charing X 4 layer bandage with studies reporting 70%+ healing rates in 12 weeks.⁷ The present median duration of a venous leg ulcer in the community is nine months. A study from Cardiff then demonstrated using a 3 layer measured tubigrip system achieved 66% healing in the same period.⁸ Further outcome data from this study demonstrated 51% of venous ulcers were relatively easy to heal, but 33% were particularly difficult to heal with responses correlating inversely to both the chronicity of the ulcer and its size at the onset of treatment. The application of multi-layer bandaging systems requires a significant degree of nursing expertise. The normal leg shape leads naturally to graduated compression by even application of compression bandaging by Laplace's Law (Appendix 1). Ulcers do not heal well in the presence of oedema of whatever aetiology.

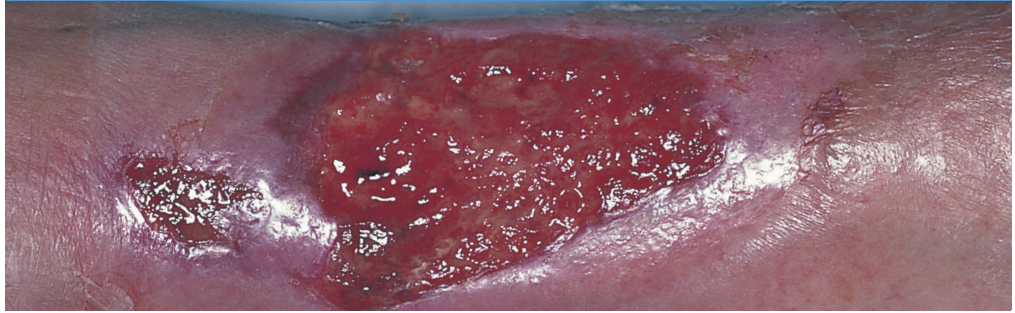
Of much less import to the rate of healing is the wound contact layer. **Dressings don't heal wounds!** There are myriad regimes but very little scientific validity. This cannot be over emphasised. The frustration engendered by attempting to heal recalcitrant ulcers can be well understood but this cannot justify the constant interchanging of dressings, which has been demonstrated in research, with little regard to evidence or expense. This process is often driven by commercial pressures and unsubstantiated claims from various pharmaceutical companies. 15% of the overall costs of wound care relate to use of dressings.

Some understanding of the different broad categories of dressings and their indication is advisable for the physician; after all, it is his or her name at the bottom of the prescription! The common goal of all dressings, however, is to create an optimal environment for healing:

- moist but not macerated
- optimal temperature and pH
- free of toxins and irritants
- free of clinical infection

ULCER CHARACTERISTICS

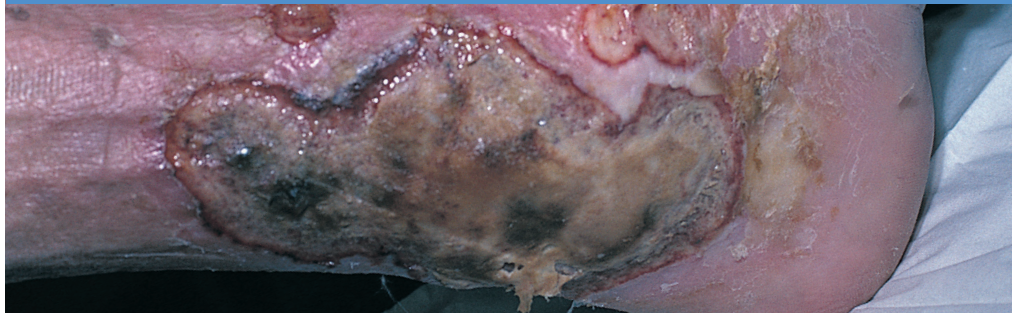
EXUDATION



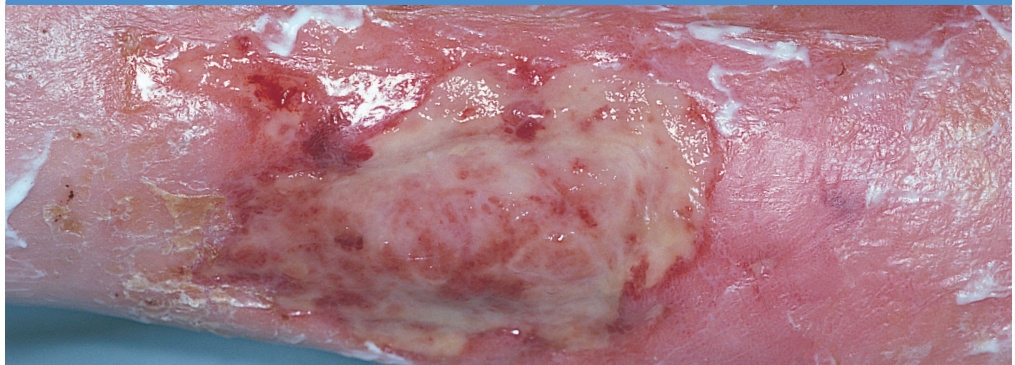
GRANULATION



SLOUGH



INFECTION



DRESSINGS TYPES

- 1.) **Films** e.g. Opsite: These are polyurethane membranes, waterproof, transparent, flexible and permeable to gas and water vapour. They are non absorptive and thus not suitable for heavily exuding wounds.
- 2.) **Foams** e.g. Lyofoam: Heat treated polyurethane, very absorbent and insulating. Suitable for a wide range of granulating wounds both flat and cavity.
- 3.) **Hydrogels** e.g. Intrasite: Organic polymers with an increased water content, hydrating and absorbent and capable of absorbing large amounts of exudates. These promote autolysis.
- 4.) **Alginates** e.g. Kaltostat: Derived from seaweed, very absorbent, hydrating and haemostatic. These can be used in moderate or heavily exuding wounds.
- 5.) **Hydrocolloids** e.g. Granuflex and Comfeel: Polymers in fine suspension, absorbent and promote autolysis. Available in pastes, granules and wafers.
- 6.) **Inert** e.g. any dressings and gauze: Secondary dressings with a primarily protective role. These provide an optimal environment for moist wound healing.
- 7.) **Protease-modulating matrix**: As stated in the BNF, these dressings alter the activity of proteolytic enzymes in chronic wounds.

Where compression bandaging techniques are positively beneficial to wound healing and wound contact layer essentially neutral, inappropriate cleansing and other topical agents can be positively detrimental! Antiseptics have been implicated in retarding wound healing and locally applied antibiotics are notorious skin sensitisers and should be avoided. Don't forget that contact sensitivity is also not uncommon with steroid preparations and impregnated paste bandages. The commonest skin sensitisers in regard to leg ulceration are neomycin, lanolin, parabens and, less commonly, colophony and rubber. Many a time I have had the experience of patients having protracted treatment for 'cellulitis' when the real problem is one of contact sensitivity!

NB Cellulitis is highly unlikely to be bilateral, the process should be dynamic and the patient is often symptomatic or at very least should have a significant elevation in CRP. If these factors are borne in mind, then this common mis-diagnosis should be avoided.

CELLULITIS



CONTACT SENSITIVITY

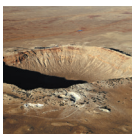


Adequate mechanical debridement of contaminated wounds is also important. Irrigation of ulcers with normal saline or soaking in warm water are usually adequate for cleansing. Potassium permanganate soaks, however, can be very useful particularly for extensive areas of weeping, exudative skin for short periods. Be aware that there is a health warning related to accidental ingestion of both liquid and tablet formulations.

Systemic antibiotics are only indicated in clinically infected ulcers and where there is evidence of cellulitis, lymphadenitis or septicaemia. Appropriate antibiotics should be used in high doses and for periods of at least one week to be effective. The presence of beta haemolytic *Streptococcus* always requires consideration for treatment.

Overgranulation is traditionally treated with a local application of silver nitrate or potent fluorinated topical steroids (e.g. clobetasol 0.05%). Both Flamazine (1% silver sulphadiazine) + topical metronidazole (0.75%) can be used short term in malodorous ulcers, the former when *Pseudomonas* is present and the latter in anaerobic infections.

OVERGRANULATION



OTHER DRUGS

There is some evidence of benefit with pentoxifylline⁹ in healing chronic venous ulcers. No such benefit has been demonstrated for stanozolol,¹⁰ aspirin or zinc. Diuretics can be useful as an adjunct but are not a substitute for adequate compression.

The most pessimistic data regards present recurrence rates which can be as high as 70% at one year, rising almost to 100% at five years if the deep veins are abnormal. Well fitted compression hosiery post healing and the advent of “healed ulcer” clinics or “leg clubs” will hopefully continue to impact on these depressing statistics as will a greater understanding of skin barrier function by nursing staff and carers particularly in relation to its age related compromise. The application of open toe class II compression hosiery changed every three months in the Cardiff study reduced recurrence to 11%!⁸ A well informed patient who has been involved at all stages in their treatment is more likely to comply with post healing regimes.

Malignant changes are a recognised rare complication of chronic wounds (so called Marjolin's ulcer). If there is clinical suspicion, a punch biopsy for the suspicious area can easily be obtained.

MARJOLIN'S ULCER



CRITERIA FOR REFERRAL

An appropriate referral must be considered for the following categories:

- 1). Venous ulcers failing to progress at three months or healed by 12 months.
- 2). All ulcers of an arterial or mixed aetiology for assessment for reconstructive surgical/radiological procedures.
- 3). Failure to adequately control underlying co-morbidities.
- 4). Suspected or confirmed malignant change.
- 5). Selective venous ulcers post healing, especially in younger patients – 50% of patients have deep vein reflux almost invariably consequent on previous DVTs which are often undiagnosed.
- 6). Suspected contact sensitivity for consideration of patch testing.

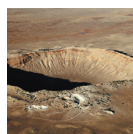
Rarely in medicine is there a perception that improved patient care can save money. Effective management of chronic leg ulceration is one such example!

TEACHING POINTS

- 1). A leg ulcer is not a diagnosis but a manifestation of the underlying disease process.
- 2). Easy access to accurate Doppler assessment should be available to all involved in leg ulcer care.
- 3). Dressings don't heal wounds.
- 4). Leg ulcers are a high risk site for contact sensitivity reactions.
- 5). The healing of an ulcer is **not** the clinical endpoint – appropriate referral for assessment and intervention should be considered and systems for maintaining ulcer healing further developed.

REFERENCES

- 1). Phillips CJ *et al.* Cost of managing patients with venous leg ulcers. *Int Wound J.* 2020;17:1074-1082
- 2). Guest JF *et al.* Cohort study evaluating the burden of wounds to the UK's National Health Service in 2017/2018: update from 2012/2013. *BMJ Open* 2020;10:e045253
- 3). Fletcher, Cullum, Sheldon; A systematic review of compression treatment for venous leg ulcers, *British Medical Journal* Vol 315 6/9/97.
- 4). Blair SD *et al.* Do Dressings influence the healing of chronic venous ulcers? *Phlebology* 1988; 3: 129-34.
- 5). O'Meara S, Cullum N, Nelson EA, Dumville JC. Compression for venous ulcers. *Cochrane Database of Systemic Reviews* 2012 Issue 11.
- 6). Callam MJ, Dale JJ, Ruckley CV, *et al*; Hazards of compression treatment of the leg, *British Medical Journal* 1987; 295: 610, 1382.
- 7). Moffatt CJ; Compression bandaging/The state of the art, *The Journal of Wound Care* Vol 1 No 1 1992.
- 8). Unpublished trial.
- 9). Iglesias C, Claxton K *et al.* (2001) The value of clinical trials of Trental in the treatment of chronic venous ulcers, *Medical Decision Making* 21(6), p.531.
- 10). Layer GT, Stacey MC, Burnard KG (1986) Stanazolol + treatment of venous ulceration, *Phlebology* 1: 197-230.



APPENDIX 1

LAPLACE'S LAW

How graduated compression works

Some of the concepts that affect the pressure exerted by bandages are explained by

a Law of physics – Laplace's Law which states
$$P = \frac{T \times N \times \text{constant}}{C \times W}$$

P = Sub bandage pressure

Pressure exerted by bandage.

T = Tension

Bandage tension depends on the elasticity of the bandage i.e. how much stretch is applied on application.

N = Number of layers

The more layers applied, the higher the sub bandage pressure, as in the multi-layer system.

C = Limb circumference

Only variable sub bandage pressure is inversely proportional to the circumference of the leg. Therefore it is important to measure the ankle circumference – just above the malleolus (2cm). The ankle circumference will determine the regime of bandaging according to manufacturers instructions.

W = Width of bandage

The narrower the bandage width the more compression applied. More layers are applied with narrower width. Generally a 10 cm bandage is used.



Stock photo. Posed by models.

From young itchy eczema to elderly varicose eczema

Dermol® Cream

Gentle antimicrobial leave-on emollient and soap substitute

Rich hydrating emollient cream

Cosmetically acceptable¹, gentle, creamy formulation that easily absorbs into the skin.

High oil content (20%) and humectant to hydrate, protect and soothe vulnerable skin.

Soap substitute

A gentle, non-ionic cleanser makes **Dermol Cream** suitable for use as a soap substitute.

Formulated for sensitive skin



Antimicrobials

Staphylococcus aureus colonisation can trigger an itchy inflammatory reaction.

Dermol Cream has proven activity against *Staph aureus* including MRSA², FRSA², Mupirocin-resistant *Staph aureus*³; as well as, *Pseudomonas aeruginosa*⁴, *Streptococcus pyogenes*⁵ and *Malassezia furfur*⁶ when tested *in vitro*.

Patient-preferred

Preferred by patients to their previously used emollients for relief of itching, relief of dryness and cosmetic acceptability.¹

Prescribe Dermol Cream to knock out *Staph* and soothe very dry, itchy skin conditions

Dermol® Cream

Benzalkonium chloride 0.1% w/w, chlorhexidine dihydrochloride 0.1% w/w, liquid paraffin 10% w/w, isopropyl myristate 10% w/w.

Uses: An antimicrobial emollient cream for the management of dry and pruritic skin conditions, especially eczema and dermatitis, and for use as a soap substitute.

Directions: Adults, children and the elderly: Apply direct to the dry skin or use as a soap substitute.

Contra-indications, warnings, side effects etc: Please refer to SPC for full details before prescribing. Do not use if sensitive (especially generalised allergic reaction) to any of the ingredients or if there is a possible history of allergic reaction to a chlorhexidine compound. In the unlikely event of a reaction stop treatment.

Local skin reactions are very rare (<1/10,000 based on spontaneous reporting). Reactions have been observed occasionally when used excessively as a leave-on application in the anogenital area. Keep away from the eyes.

Instruct patients not to smoke or go near naked flames. Fabric (clothing, bedding, dressings etc) that has been in contact with this product burns more easily and is a potential fire hazard. Washing clothing and bedding may reduce product build-up but not totally remove it.

Package quantities, NHS prices and MA numbers: 100g tube £2.86, 500g pump dispenser £6.63, PL00173/0171.

Legal category: [P]

MA holder: Dermal Laboratories, Tatmore Place, Gosmore, Hitchin, Herts, SG4 7QR, UK.

Date of preparation: May 2019.

'Dermol' is a registered trademark.

Adverse events should be reported. Reporting forms and information can be found at yellowcard.mhra.gov.uk. Adverse events should also be reported to Dermal.

References: 1. Data on File. Dermal Laboratories. January 2005; 2. Gallagher J. *et al*. Routine infection control using a proprietary range of combined antiseptic emollients and soap substitutes – their effectiveness against MRSA and FRSA. Poster presented at 18th EADV Congress, October 2009, Berlin, Germany; 3. Gallagher J. and Roshier P. Topical antiseptic products – Antimicrobial activity

against mupirocin resistant *Staphylococcus aureus*. Data presented at the 16th Annual Maui Derm for Dermatologists, January 2020, Maui, USA; 4. Gallagher J. and Roshier P. Infected wounds – *in vitro* activity of topical antiseptic products against *P. aeruginosa*. Poster presented at the 23rd EADV Congress, October 2014, Amsterdam, The Netherlands; 5. Gallagher J. and Roshier P. Evaluation of the bactericidal activity of two antiseptic emollient formulations against *Streptococcus pyogenes*. Poster presented at the 73rd Annual Meeting of the American Academy of Dermatology, March 2015, San Francisco, USA; 6. Gallagher J. & Roshier P. *In vitro* antimicrobial activity of two topical antiseptic products against *Malassezia furfur*. Poster presented at the 10th EADV Spring Symposium, May 2013, Cracow, Poland.

MRSA, Meticillin-resistant *Staph aureus*;
FRSA, Fusidic acid-resistant *Staph aureus*.

 **DERMAL™**
TOPICAL INNOVATION
www.dermal.co.uk