Healing of an arterial leg ulcer by compression bandaging: a case report

Abstract: This case report demonstrates that arterial ulcers may heal with compression therapy. The patient was a 72-year-old woman with elevated blood pressure and type II diabetes mellitus, who underwent angiography because of a painful, non-healing ulcer over the shin. The angiography showed multisegment arterial occlusive disease, not amenable to surgical revascularisation. After 4 months of wearing inelastic bandages, with increased pressure over the ulcer area and hypertension and diabetes under control, the patient was pain-free and the ulcer had healed. The ankle-brachial pressure index (ABPI) increased from 0.54 at the start of treatment to 0.70.

In patients with arterial leg ulcers, properly applied compression therapy using increased pressure over the wound may lead to an improvement of the arterial flow and to complete healing.

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Common belief is that arterial ulcers are frequently put into the same category as ‘critical ischaemia’. The Trans-Atlantic Inter-Society Consensus (TASC) II group gave the following definition:¹

‘The term critical limb ischemia should be used for all patients with chronic ischemic rest pain, ulcers or gangrene attributable to objectively proven arterial occlusive disease.’

In a more recent consensus paper on critical limb ischaemia (CLI), more precise definition is given.² Recommendation 1 for CLI, in patients with and without diabetes, is defined by either of the following two criteria:

- Persistently recurring ischaemic rest pain requiring regular adequate analgesia for more than two weeks with an ankle systolic pressure <50 mmHg and/or toe systolic pressure <30 mmHg
- Ulceration or gangrene of the foot or toes, with an ankle systolic pressure <50 mmHg or toe systolic pressure <30 mmHg.

These criteria are a contraindication for sustained compression therapy in accordance with international guidelines, which recommend arterial revascularisation in such cases.³ However, sticking to these criteria, leg ulcers (LUs), without any ischaemic skin changes distal from the ankle, do not meet the criteria of critical ischaemia. An acknowledged, clear definition of ‘arterial ulcers’ is lacking. We consider LUs complicated, but not caused by concomitant arterial occlusive disease. Most are of venous origin (so-called ‘mixed arterial-venous leg ulcers’).⁴ They may also be caused by microvascular pathology as such as Martorells ulcer,⁵ in whom additional occlusions of large arteries may occur. Several studies have shown that conservative therapy or skin grafting may be successful without arterial revascularisation.⁶⁷

According to a recent consensus document between 5–30% of LUs are arterial in origin, including patients with occlusions in the large arteries and small vessel disease, as in patients with diabetes and/or arterial hypertension.⁸ In this document referral for revascularisation is recommended if the ankle brachial pressure index (ABPI) <0.5. However, it is noted that modified compression using stiff material may be applied with frequent reassessment and monitoring for ischaemia and pressure damage. Even without any venous damage, the hydrostatic pressure in the upright position promoting oedema always plays a triggering role for ulceration. The beneficial effects of compression therapy that have been demonstrated in mixed LUs, are in part due to the effect of compression on the venous pathology.⁷ The following case report shows that ulcer healing may be achieved also in arterial LUs without venous insufficiency.

Case report

A 72-year-old woman presented with a painful ulcer over the left shin to the office of CS on August 11 2015. The ulcer, which had been present for three months, probably after minor trauma, was accompanied by pain, both day and night.
The patient had type II diabetes, hypertension, asthma and coxarthrosis, and a body mass index of 22 kg/m². She had never smoked. Angiography had been performed one month before because of rest pain in the contralateral leg. This had shown an occlusion of the femoral artery on both sides. She was admitted to the local hospital where chronic ischaemia grade IV of the left lower extremity was diagnosed. ABPI was 0.42. It was decided not to neovascularise the left leg because of the poor arterial situation, which had additional distal arterial occlusions. The patient was dismissed from the hospital after two weeks and was advised to cover the wound with non-adhering local dressings and to avoid any kind of compression.

At the first visit to CS, a sharply demarcated, fairly circular ulcer over the proximal shin was recorded with the largest diameter of 4 cm and was around 5 mm deep (Fig 1). It contained some black, necrotic slough at the ulcer base. The periwound skin showed signs of inflammation, reddening and oedema, but no signs of chronic venous insufficiency, such as lipodermatosclerosis or pigmentation. The ulcer was extremely painful so that the patient avoided walking and spent her day in the sitting position. A new angiography of the left leg showed a long occlusion of the superficial femoral artery and a poor filling of the distal arteries in the lower leg (Fig 2). At this stage the clinical picture was interpreted as an arterial occlusive disease stage III. Her left leg was cooler than the right with very weak peripheral pulses that could be palpated. The systolic ankle pressure was 100 mmHg, systolic arm pressure 185 mmHg, and the ABPI 0.54.

There were no varicose veins visible and no refluxes were detected over superficial or deep veins using a Doppler probe (Duplex would have been preferred but is not available in the office of the GP who treated the patient). Swab cultures were taken and revealed a growth of *Pseudomonas aeruginosa*.

**Drug therapy**

Hypertension was treated with the AT1 receptor blocker olmesartan 40 mg daily. Diabetes was controlled by diet and metformin. The statin rosuvastatin was given to control dyslipidaemia, clopidogrel as an antiplatelet drug. Paracetamol was given as a pain-killer during the first two months.

**Compression therapy**

After cleaning the wound bed, with physiological saline, ‘double focal compression bandaging technique’ was applied, using non-adhering dressings on the wound, covered by 10x10cm gauze pads, which are...
fixed by a latex-free cohesive bandage (Cpk Fix - Farmaban, 8x20cm). A second short-stretch bandage (Tex-San/Tecson, 10x5 m) was wrapped over and the patient asked to walk for up to 10 minutes and to come back when there was an increase in the level of pain, which never happened. During the first days of treatment, the pressure of both bandages was kept rather low and the patient asked to remove the outer bandage during night. She was seen in the doctor’s office every day and the skin status under the compression carefully checked and documented by daily photographs (Fig 3).

After two months the colonisation with Pseudomonas aeruginosa cleared without the need for antimicrobial therapy. The patient reported less pain and the bandages were left day and night on the leg. During this period the resting pressure measured under the pads by a Kikuhime-transducer was around 50 mmHg immediately after bandage application.

After four months the ulcer was healed and the patient was free from pain and could walk slowly without any restriction. Systolic arm pressure was 130 mmHg, ankle pressure 92 mmHg (ABPI 0.7).

After six months the ulcer was still closed (Fig 4), the patient applies her own compression bandages and has very good peripheral pulses on the left leg. Her ability to walk has improved and she feels better.

Discussion

The pathogenesis of the ulcer in the present case is quite complex. It may be assumed that in addition to the arterial occlusions in the large arteries predominantly in the lower leg, changes in the small arteries and the arterioles of the microcirculation due to both diabetes and hypertension, contributing to the local skin defect.

The typical localisation over the shin lets us assume that a minor trauma in that region had triggered the skin necrosis, patients often do not remember if they hit their legs. While such mechanisms would not cause local necrosis in other parts of the body this is different in the legs where the venous drainage against the forces of gravity is a critical factor, even in normal veins. The slow flow in the capillaries will induce adhesion of leucocytes to the damaged endothelial layer. These will penetrate together with protein-rich fluid through the capillary wall resulting in oedema formation and inflammation. As long compression does not impede the arterial inflow, the effects are quite beneficial as this is shown in the presented case.

Clear discrepancies regarding diagnosis and therapeutic consequences are shown between a vascular surgical department of a hospital and a medical practitioner experienced in ulcer treatment. In the hospital, the patient was diagnosed as chronic ischaemia grade IV of the left lower extremity’ (as the classification of Fontaine ‘stage IV’ is defined as the most advanced stage of arterial occlusive disease with gangrene and high risk of limb loss). Due to the multi-level, arterial occlusions in this diabetic patient, revascularisation was considered not promising enough and the patient was dismissed, banning any kind of compression therapy.

For the GP recommending compression, even with increased pressure over the ulcer area, this is a difficult situation needing much discussion. Furthermore, the patient will need guiding through the initial period, in which the pressure and the wearing time of the bandage has to be adjusted to the pain level. For this purpose, but also for medicolegal reasons daily visits of the patient were arranged.

In accordance with long-lasting experience with ulcer patients the leg wound started to get flat and clean, without debridement, application of any special local dressing or the use of antimicrobial...
substances and the pain improved. The positive development was demonstrated by daily photographs which also reassured the patient, as a result of which she followed the advice to walk as much as possible with increasing adherence. Ultimately the ulcer was healed after four months.

Doppler sonography showed a clear qualitative improvement of the arterial sounds of the distal pulses, which changed from a monophasic to a biphasic pattern, and an increase of the ankle pressure, reflected by an increment of the ABPI from originally 0.54 to 0.7. The increase of the ABPI indicates improvement in the collateral circulation as this was demonstrated after intermittent pneumatic compression (IPC) and supervised exercise.\textsuperscript{12}

Acute experiments, in healthy subjects\textsuperscript{13} and patients with arterial occlusive disease have demonstrated an increase of the arterial flow under a four-layer bandages and modified inelastic bandages respectively.\textsuperscript{7} This is true as long the compression pressure does not exceed the local arterial perfusion pressure. For patients with an ABPI between 0.5 and 0.8 ‘modified compression’ has been recommended, which is characterised by applying inelastic bandages with a resting pressure lower than 40 mmHg and frequent bandage changes allowing close inspection of the skin.\textsuperscript{7}

Several mechanisms of action may explain an increase of the arterial flow under compression.\textsuperscript{14–16}

- An autoregulatory response to the decrease of the transmural pressure gradient and a myogenic relaxation in the arterial wall
- A vasodilatory axon reflex response, mediated by nervous and biomechanical signals
- A reduction of arteriovenous pressure gradient by improvement of venous return, especially in combination with walking exercises.

Oedema reduction by compression will reduce the distance between the capillaries and the tissue cells thereby shortening the way the nutrients must pass to reach their target cells. Bringing the blood capillaries in closer contact with the cells will lead to an improvement of nutrition.\textsuperscript{17}

Inelastic compression together with walking creates a rhythmic massage which resembles the effects of intermittent pneumatic pumps resulting in a release of vasoactive mediators from the venular endothelial cells due to the pulsating increase of the shear stress in the microcirculatory flow.\textsuperscript{11} In contrast to elastic material, which would not be tolerated with an initial resting pressure of around 50 mmHg, there is an immediate pressure loss due to instant oedema reduction. In addition to these local compression effects, an improvement of the venous pumping function may be taken into consideration, as this was shown in sports people without venous pathology.\textsuperscript{18}

It seems unlikely that the medication which was taken before the compression therapy played a role in the reduction of oedema, inflammation and the flattening and healing of the ulcer.

**Conclusion**

Compression is indicated in arterial ulcers under the following prerequisites:

- Ankle pressure measured by Doppler ultrasound should be >50 mmHg
- ‘Modified compression’ using short-stretch material with reduced interface pressure
- Walking exercise and frequent bandage changes with careful skin checks.

Our case report demonstrates that this will lead to an improvement of arterial flow so that arterial leg ulcers may heal.

**References**