



Double Focal Compression Bandaging in Vascular Ulcers: Balancing Efficacy and Cardiovascular Effects

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Abstract

The treatment of vascular ulcers, both venous and arterial, primarily relies on compression therapy, which not only supports wound healing but also improves venous circulation by reducing venous hypertension. However, many patients prematurely discontinue compression therapy once the ulcer has healed, perceiving wound closure as the endpoint of treatment. This early cessation increases the risk of ulcer recurrence, as the underlying pathology remains untreated. While compression has traditionally been contraindicated in arterial ulcers, recent studies suggest that its controlled application can facilitate healing, provided arterial perfusion is adequate (ankle-brachial index ≥ 0.6). These findings open new therapeutic avenues that require careful, long-term follow-up.

This principle is exemplified by a patient with valvular heart disease who, following cardiac surgery in 2015, developed a vascular ulcer four years later. The ulcer was successfully treated using double focal compression bandaging, achieving complete healing within six months. Rather than discontinuing compression therapy, the patient transitioned to the indefinite use of compression stockings, ensuring ongoing vascular support and reducing the risk of recurrence. This case highlights the fundamental role of patient education and adherence strategies in maintaining the benefits of compression therapy over time.

Emerging evidence suggests that, in patients with chronic venous insufficiency, sustained compression may increase cardiac preload, leading to chamber distension and stimulating the secretion of natriuretic peptides as a compensatory mechanism. Further research is necessary to fully understand the cardiovascular impact of prolonged compression therapy and its long-term effects.

Keywords: Vascular Ulcers; Double Focal Compression Bandaging; Wound Healing; Cardiovascular Benefits

Introduction

At the completion of ulcer treatment using the double focal compression technique, patients are advised: "The most challenging aspect of healing your ulcer is that you may stop bandaging your leg". This statement emphasizes the necessity of continued compression therapy through high-compression stockings, as the underlying vascular disease remains present. Compression improves venous return, reduces fatigue, and enhances circulation. Patients often report that walking with compression stockings requires significantly less effort compared to walking without them, illustrating the benefits of sustained compression therapy.

For years, compression therapy has been the primary treatment for patients with vascular ulcers. Some of these patients also present with heart disease, raising concerns that compression might exacerbate symptoms by compromising cardiovascular function. To address this, patients were systematically monitored for signs of cardiac decompensation, such as worsening dyspnoea

or peripheral oedema. If such symptoms emerged, compression therapy was adjusted or temporarily discontinued to prevent additional cardiovascular strain.

Contrary to initial concerns, clinical observations showed that compression therapy did not exacerbate symptoms. Instead, it appeared to enhance cardiac function and overall quality of life. These findings align with emerging evidence supporting cardiovascular benefits of compression therapy, as demonstrated in recent studies [1,2,3].

As stated in the NYHA classification, leg compression is contraindicated in patients with class IV heart failure, defined as: 'Patients whose heart disease prevents them from engaging in any physical activity without discomfort. Symptoms of heart failure or angina may appear even at rest and worsen with minimal exertion' [4].

The clinical improvement observed in patients extended beyond ulcer healing, suggesting an additional underlying mecha-

nism. A possible explanation lies in the pathophysiology of natriuretic peptides. In patients with chronic venous insufficiency or controlled cardiac function, compressive therapy may increase cardiac preload, leading to chamber distension and stimulating the physiological secretion of natriuretic peptides. These peptides play a compensatory role in volume regulation [2], which may partially explain the observed clinical benefits.

A reduction in cardiac diameter was observed in chest X-rays taken after the initiation of compressive therapy, compared to previous images (Figure 1). While this finding suggests a potential effect of compression therapy, other contributing factors cannot be excluded. Serial imaging over time may provide further insights into the hemodynamic impact of prolonged compression.

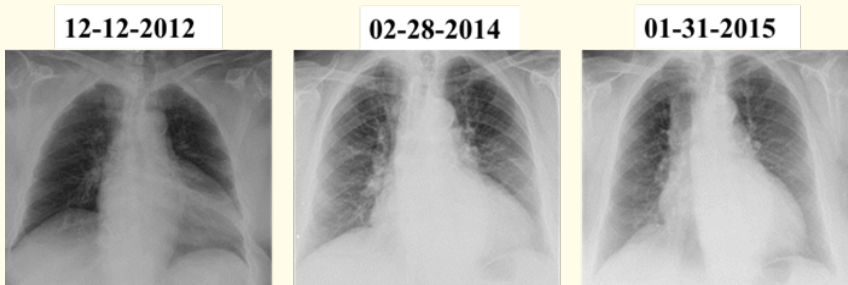


Figure 1: Cardiac patient treated with double focal compression; cardiac size progressively decreases during therapy.

We present the case of an 85-year-old patient with a history of chronic heart disease, including hypertensive cardiomyopathy and atrial fibrillation, who developed a pretibial vascular ulcer on her right leg. The wound had shown a poor healing response despite prior treatments, including conventional wound dressings and topical therapies, persisting for one month without significant improvement. Given her underlying cardiac condition and the chronic nature of the ulcer, there was concern that prolonged inflammation and venous stasis could further compromise local tissue perfusion and hinder wound healing.

Her ankle-brachial index was 1.1, indicating preserved arterial perfusion, which allowed for the safe implementation of compression therapy. Due to the lesion’s chronicity and her multiple comorbidities, the double focal compression bandaging technique was chosen. This method was preferred over standard compres-

sion therapy as it provides targeted pressure distribution, enhancing local perfusion while minimizing excessive circulatory load and the potential risk of cardiac decompensation.

During the initial weeks of treatment, daily monitoring was conducted to assess wound progression and identify any early complications. Surveillance focused on evaluating the healing trajectory, ensuring adequate tissue perfusion, and detecting any adverse effects related to compression therapy. Special attention was given to early signs of cardiac decompensation, with systematic assessment of hemodynamic parameters, including peripheral oedema, dyspnoea, variations in blood pressure, and indications of local ischemic changes. These measures were implemented to promptly adjust therapy if any hemodynamic instability was detected, ensuring patient safety and optimizing treatment outcomes (Figure 2).



Figure 2: Clinical evolution of the ulcerative lesion over time.

Following a traumatic injury, the wound showed early signs of ulceration, but the prompt application of compression bandaging successfully prevented its progression. The wound healed effectively, and her relatives were trained by the medical staff on how to properly apply the bandaging technique to ensure continued care. From then on, they applied the bandage daily, removing it at night and reapplying it in the morning. It is worth noting that the patient did not tolerate strong compression stockings, making bandaging the preferred option.

In July 2016, during a cardiology consultation, her medical report described hypertensive heart disease with preserved left ventricular ejection fraction, chronic atrial fibrillation with contra-indication for anticoagulation, moderate-to-severe mitral regurgitation, moderate tricuspid regurgitation, pulmonary hypertension,

and secondary heart failure. Despite this complex cardiovascular profile, she remained stable over the next six years, without episodes of decompensated heart failure that required hospitalization. Her quality of life significantly improved, particularly in terms of mobility and relief of lower limb oedema, with no changes in her treatment other than the introduction of compression bandaging. She passed away in 2018 at the age of 91 due to a fall down the stairs, which was unrelated to her cardiovascular condition.

Illustrative case

An 85-year-old patient with hypertension, obesity, dyslipidaemia, and osteoarthritis was diagnosed with ischemic cardiomyopathy, left bundle branch block, and moderate aortic stenosis. She reported fatigue and exertional dyspnoea. Her heart failure was classified as NYHA class II–III (Figure 3).

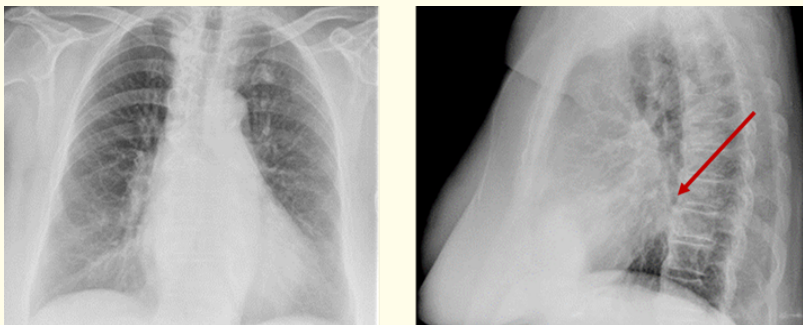


Figure 3: Preoperative chest X-ray before aortic valve replacement showing cardiomegaly.

The patient was admitted on February 10, 2015, with acute decompensated heart failure, manifesting as worsening dyspnoea and volume overload. Further assessment revealed disease pro-

gression with severe mitral regurgitation and aortic stenosis (Figure 4). After stabilization with optimized medical therapy, she underwent valve replacement surgery on October 5, 2015, without immediate postoperative complications.



Figure 4: Decompensated Heart Failure.

On May 6, 2019, she presented to our clinic with a persistent wound on her left leg following a traumatic injury. Despite multiple prior treatments, the wound remained unhealed. Until this consultation, she had not received double focal compression therapy.

Given her complex cardiovascular history, including ischemic cardiomyopathy and moderate aortic stenosis, a thorough evaluation was performed, including peripheral perfusion assessment. An ankle-brachial index of 0.96 indicated preserved arterial circulation, allowing for the safe implementation of compression ther-

apy. Due to her chronic wound and absence of infection, double focal compression bandaging was initiated. This technique was preferred over standard compression therapy for its targeted pressure distribution, enhancing local perfusion while minimizing circulatory overload and the risk of cardiac decompensation.

During the first few weeks, daily follow-ups were scheduled to monitor wound progression and detect early signs of cardiac instability. Vital signs, including peripheral oedema, dyspnoea, and blood pressure, were systematically assessed to ensure hemodynamic stability. Special attention was given to potential ischemic

changes, with prompt therapy adjustments if cardiovascular complications arose. The patient tolerated the compression bandage well, reporting significant wound healing and improved overall comfort.

After five months of consistent therapy, the ulcer had fully healed (Figure 5) without the need for antimicrobial agents [5]. To prevent recurrence, long-term compression therapy was recommended, given her complex cardiovascular history and the need for continued vascular support. Regular follow-ups confirmed her clinical stability, with no ulcer recurrence or signs of cardiac decompensation.



Figure 5: Complete wound healing was achieved after five months of treatment with the double focal compression technique.

This case further supports the feasibility of compression therapy in patients with complex cardiovascular conditions, as shown in previous studies [3]. While compression bandaging is well established for vascular ulcers—promoting wound healing by reducing oedema and improving venous return—its systemic hemodynamic effects remain insufficiently explored and require further investigation. Although research on its use in heart failure patients is still limited, recent studies have begun assessing its safety and efficacy, showing encouraging preliminary findings. This growing interest highlights the need for rigorous clinical trials to better define its role in this population [6-8].

When a wound presents with delayed healing, I always assess for an underlying vascular disorder (venous, arterial, or mixed) or a neoplastic process.

It is important to consider other factors that may delay wound healing, such as undiagnosed vascular conditions, infections, or metabolic imbalances. In this case, the diagnosis of venous ulcer was established based on the identification of clinical signs and symptoms, such as oedema, hyperpigmentation, and manifestations of chronic venous insufficiency, including lipo-dermatosclerosis (Figure 6).

Despite the patient’s cardiac condition, the oedema was unlikely to be due to heart failure, as it was unilateral and exhibited features more indicative of venous stasis than cardiac-related fluid retention. Performing a differential diagnosis of the oedema is necessary for an appropriate therapeutic approach. The presence of a biological valve prosthesis reflected her complex cardiovascular history.

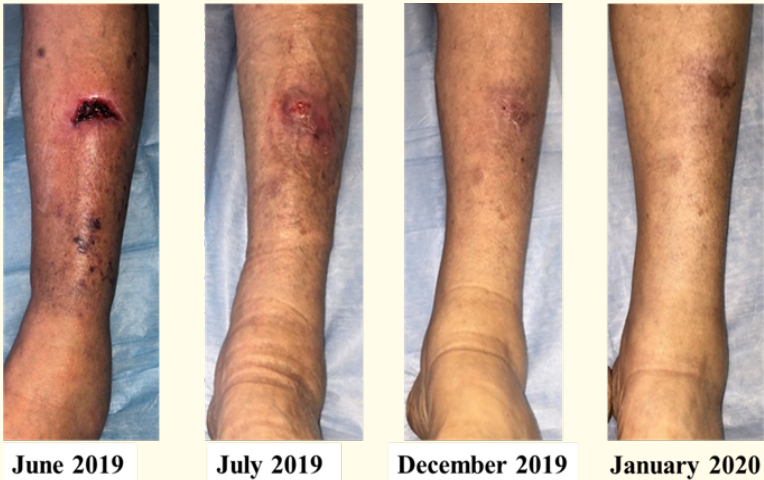


Figure 6: Leg appearance before treatment and after ulcer healing.

After the ulcer healed, she began using strong compression stockings during the day, removing them at night, a habit she continues to follow, which demonstrates her adherence to the treatment.

A key concern when applying compression therapy in a patient with heart disease is the potential risk of cardiac decompensation.

To mitigate this risk, a daily clinical follow-up during the initial weeks of treatment is essential.

Over the years, no signs or symptoms of cardiac decompensation have been observed, and her overall quality of life has significantly improved (Figure 7). She maintained regular cardiology follow-ups, which allowed for close monitoring of her cardiovascular status.

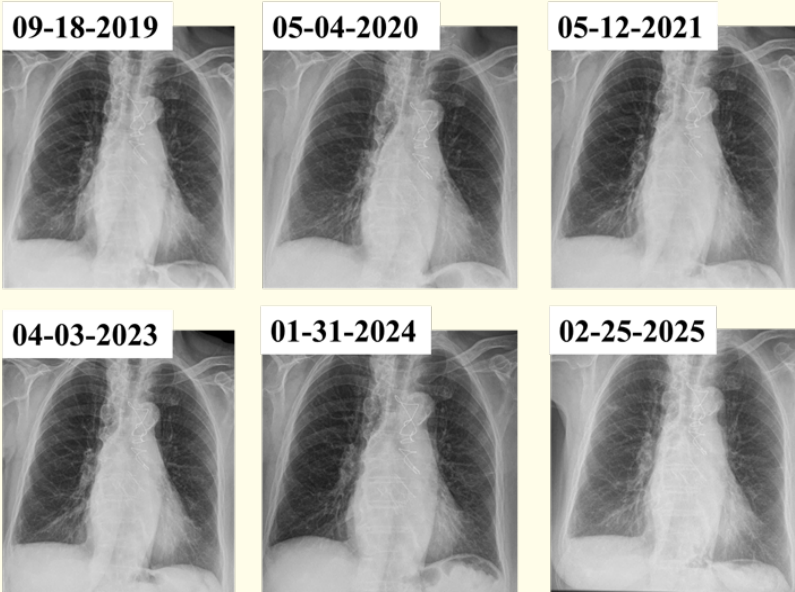


Figure 7: Throughout the duration of compression therapy, no evidence of cardiac decompensation was detected.

Moreover, studies indicate that compression therapy, when tailored to the patient’s functional status, can reduce hospitalization time and the need for diuretic medication in patients with heart failure [8]. This comprehensive and individualized approach has proven to be a valuable complement to standard care for patients with coexisting cardiovascular disease and chronic wounds.

On March 3, 2025, the patient attended a scheduled follow-up consultation. During the clinical examination, a small pretibial

hematoma was detected on her left leg, likely caused by a minor, unnoticed trauma, posing a risk to the healing process. Since she had been advised to seek medical attention for any leg injury, immediate intervention was undertaken. Her compression stocking was temporarily removed, and focal compression bandaging was applied to the affected area, remaining in place for two weeks to prevent ulcer formation. The lesion resolved within that period, after which the patient resumed the use of strong compression stockings (Figure 8).



Since compression therapy began in 2019, significant improvements have been observed in both skin texture and vascular appearance compared to the pre-treatment condition. This progress not only supports the efficacy of the therapy but also underscores the importance of consistent adherence for sustained clinical benefits. Regular compression therapy, combined with periodic follow-ups, has been essential in preserving vascular health and positively influencing cardiac function.

Discussion and Conclusion

In this article, the cardiovascular benefits of compression therapy in patients with uncompensated heart failure are discussed. Both patients continued with compression therapy: one with bandaging, due to intolerance to compression stockings, and the other with strong compression stockings, in accordance with clinical recommendations.

Both received detailed training focused on the early recognition of signs and symptoms of cardiac decompensation, with clear instructions to immediately discontinue compression therapy and

seek medical attention should such signs appear. This did not occur. Long-term follow-up and clinical outcomes support the safety of this technique.

1st/ In the first case, the patient passed away due to an accidental fall; however, she did not experience any episodes of cardiac decompensation during the period in which she received compression bandaging, which was applied by her family members —previously trained in the technique by us— due to her intolerance to strong compression stockings. Moreover, she experienced a notable improvement in her quality of life.

2nd/ In the second case, the 91-year-old patient, who had received a biological aortic prosthesis in 2015, has not required hospitalization for cardiac decompensation since then. After a minor trauma to her left leg on June 5, 2019, treatment with double focal compression bandaging was initiated, achieving complete ulcer closure within five months. Despite initial concerns about the potential induction of decompensation due to increased preload, no complications were observed. The patient experienced a sig-

nificant improvement in her quality of life, which was confirmed in her cardiology consultations, which also showed a reduced need for medical attention in the following years. She has strictly adhered to the daytime use of strong compression stockings, removing them only at night, a practice she continues to this day.

3rd/ Absence of infection: One of the main concerns was that infection of the ulcer could trigger a cardiac decompensation episode. No such situation occurred. Based on my previous experience, focalized pressure on the ulcer prevents infection [5]. This framework theoretically explains the rationale behind this assertion (Figure 9). The focal pressure applied through padding and external bandaging activates two pathophysiological processes: arteriogenesis and angiogenesis [9].

When an arterial vessel is obstructed, blood flow reversal increases pressure in the proximal segment, triggering the activation

of previously dormant collateral arteries. This process, known as arteriogenesis, facilitates the formation of a physiological bypass. In contrast, reduced or absent blood flow in the distal region leads to hypoxia, which serves as the primary stimulus for angiogenesis, promoting the expansion of the capillary network. The interplay between arteriogenesis and angiogenesis enhances tissue perfusion, facilitating healing and the closure of the vascular ulcer.

The absence of infection can be explained by the role of monocytes; the external pressure exerted by the padding and bandages on the ulcer increases blood flow in the intact vessels by reducing their diameter. This mechanism is consistent with the continuity equation for fluids, which states that a reduction in vessel diameter results in an increase in blood flow velocity, maintaining constant volumetric flow [10]. Similarly, we have refrained from using debridement agents, given the autolytic action of monocytes differentiated into macrophages [11].

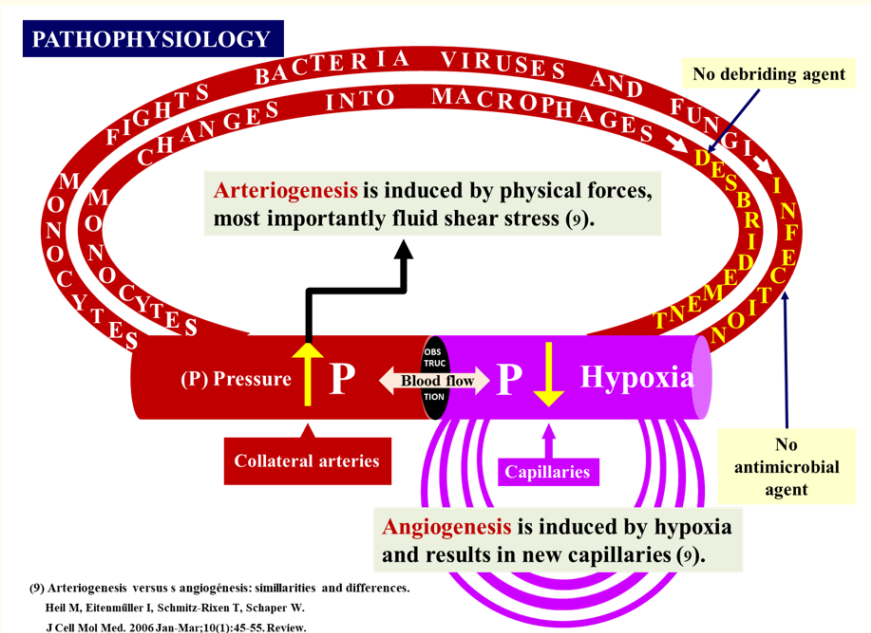


Figure 9: Schematic of the pathophysiological basis of the mechanism of action of this technique.

4th/ From a physiological perspective, the cardiovascular benefits can be attributed to the secretion of natriuretic peptides, induced by the increase in preload due to the displacement of blood volume in the oedematous limb. Consequently, it can be concluded that compression therapy is not contraindicated in patients with non-decompensated or non-advanced heart failure (NYHA IV). Although the hypothesis underlying this therapy has not been fully validated, it provides a solid pathophysiological framework that explains the observed improvements.

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