Abstract

Aim: In this study, we aimed to investigate the effect of vacuum-assisted closure therapy on venous stasis wound healing in patients with chronic venous leg ulcers.

Methods: Vacuum-assisted closure therapy was applied on a total of 14 venous leg ulcers. All patients had post-thrombotic syndrome. Quantitative wound culture samples were obtained before the procedure and local wound assessments were performed. The primary outcome measures included wound healing as assessed by a local wound examination during each dressing change and the rate and velocity of ulcer reduction. Wound healing was defined as the complete closure of the ulcer, while rapid wound healing was defined as a ≥ 30% reduction in the ulcer size by week four.

Results: No surgical debridement or surgical corrective procedure was applied in any patient. The mean length of hospital stay was 32.3 days. The mean number of vacuum-assisted closure therapies for each case was 17.8 and the mean time to dressing change was 72.3 hours. Multidrug-resistant *Pseudomonas aeruginosa* and methicillin-resistant *Staphylococcus aureus* were detected in three and four patients, respectively. Wound culture results became negative after a mean duration of vacuum-assisted closure therapy of 12.1 days. None of the patients needed antibiotic therapy until the procedure was completed. Compared to baseline, the mean ulcer reduction rates were 46.4% for the first six applications and 72.8% for the subsequent applications.

Conclusion: Our study results suggest that vacuum-assisted closure therapy promotes rapid wound healing in patients with severe post-thrombotic syndrome with venous stasis leg ulcers, and reduces the need for antibiotics by reducing the biological burden.

Keywords: vacuum-assisted closure therapy, venous ulcer, post-thrombotic syndrome, wound infection, rapid wound healing.

The primary aims of wound healing are to restore the tissue architecture and integrity of the skin, achieve aesthetically favourable and sustainable results, relieve pain, improve patient comfort and quality of life, and prevent undesirable complications in a cost-effective manner.¹

Chronic leg ulcers affect nearly 1% of the adult population.² The prevalence of active leg ulcers has been estimated to be 15%.³ Venous disease accounts for 80% of all chronic leg ulcers.¹ Venous hypertension has been proposed to be the main underlying pathophysiological mechanism of reflux and/or obstruction of the venous system.¹ Standard treatment includes surgical debridement, wound dressing with topical antimicrobials, compression therapy, pharmacological treatment and corrective procedures. Venous ulcers are the most common ulcers of the lower limbs however, the rate of complete healing is very low despite the application of standard treatment.⁴

Post-thrombotic syndrome (PTS), an important chronic consequence of deep-vein thrombosis (DVT), is an important condition for which patients frequently seek medical advice. PTS occurs in 20 to 50% of patients with DVT. PTS may present with clinical manifestations ranging from mild clinical symptoms to more severe manifestations, such as chronic leg pain, persistent oedema, and leg ulcers that limit activity and the ability to work.³

In five to 10% of patients, DVT will progress to severe PTS, which can include venous leg ulcers. A multidisciplinary approach should be used for venous ulcer management, which includes compression therapy, skin care and topical dressings. When conservative therapy fails, surgical or endovascular procedures may be recommended to treat large reflux vessels in selected patients. However, ulcers may be resistant to all treatment and frequently recur.

Chronic wounds never become sterile and are mostly colonised with micro-organisms of the normal flora of intact skin. Chronic wounds are commonplace for bacteria and these bacteria do not always cause infections *per se*. The level of bacterial burden can be described as one of the following four conditions: (1) contamination, (2) colonisation, (3) critical colonisation, and (4) infection.⁴
Bacterial colonisation is present in approximately 80 to 100% of leg ulcers. Several studies have demonstrated that increased bacterial bioburden prolongs the wound-healing process. Bacterial diversity and density are critical factors for prolonged healing processes. In the presence of infected ulcers, bacteria invade living dermal and subdermal tissues, and the clinical manifestations include worsening pain, warmth, swelling and erythema of the skin around the ulcer, which hamper wound healing. Wound infection prolongs the duration of response to chronic ulcers and delays collagen synthesis, slowing the epithelialisation process and secreting proteases.

Wound culture is the mainstay of treatment for chronic wounds. The most common culprits of chronic leg ulcers are *Staphylococcus aureus* (*S aureus*), *Pseudomonas aeruginosa* (*P aeruginosa*), *Escherichia coli* (*E coli*), *Proteus mirabilis* (*P mirabilis*), and *Enterobacter cloacae* (*E cloacae*). In recent years, however, the spectrum of micro-organisms most frequently isolated has expanded. A few years ago, researchers paid particular interest to gram-positive micro-organisms such as *S aureus*. However, gram-negative bacteria are more frequently being isolated worldwide and have become more clinically significant. Although *S aureus* is the most common pathogen in leg ulcers, recent studies have demonstrated higher rates of anaerobic and gram-negative bacilli in clinically infected leg ulcers than in those without infection. The majority of bacteria are resistant to a variety of antibiotics, such as doxycycline and penicillin, and more than half of cases have multidrug resistance (MDR).

Vacuum-assisted closure (VAC) therapy is used for chronic wounds to stimulate rapid wound healing. The therapy consists of an open-cell polyurethane sponge sealed by an adhesive drape 400 to 600 microns in size and 0.703 to 0.0228 kgf/cm² compression with an open-cell structured network. An open-cell, soft connection port is placed, and the other end of the port is mounted on the device, producing negative pressure (Fig. 1). The VAC system provides sub-atmospheric pressure for physical contraction by removing toxins, excessive interstitial fluid and cell debris from the wound bed.

Macrostrain is the visible change that occurs when negative pressure contracts the foam that is in direct contact with the wound bed, drawing the wound edges together (Fig. 2). Due to the macrostrain effect, wound edges are diminished, and exudate and infectious material are removed, leading to reduced bacterial bioburden and tissue oedema. The VAC drape is an adhesive layer, ensuring a closed, moist, wound-healing environment and preventing external contamination. In recent years, VAC therapy using local negative pressure has been used as a non-invasive adjunct in the field of plastic and reconstructive surgery. The clinical efficacy of VAC therapy has been shown in the treatment of venous leg ulcers.

In our study on the healing of infected chronic venous stasis leg ulcers in patients with severe PTS after DVT, we aimed to investigate the effect of VAC treatment, to elucidate its mechanism of action and to determine whether it could be used for these patients with limited treatment options.

**Methods**

Patients with chronic venous stasis ulcers who were admitted to the cardiovascular surgery clinic between January 2016 and January 2019 were included in this study. PTS Villata–Prandoni severity rating scoring was performed for all patients. Twelve patients with a Villata–Prandoni score > 14 (mean age: 50.2 years; range 38–71 years) were considered to have severe PTS. VAC treatment was applied to the 12 patients with severe PTS and 14 venous stasis ulcers. Written informed consent was obtained from each patient.

The inclusion criteria were as follows: previous diagnosis of DVT, severe PTS, chronic venous insufficiency, a venous ulcer that did not heal despite venous correction procedures, and clinical signs and symptoms of infection with micro-organism growth in the pre-procedural wound culture. The exclusion criteria were as follows: acute DVT, peripheral arterial insufficiency [ankle–brachial index (ABI) < 0.8], anti-aggregant and anticoagulant therapy, visible vascular structures, untreated osteomyelitis, necrotic wounds with severe scarring, and decision to undergo venous correction and compression therapy first for ulcer healing.

The study protocol was approved by the ethics committee of the Republic of Turkey, Ministry of Health, Turkish Medicines and Medical Devices Agency, Research & Development Department (65355237-604.01.02-E.134, Decree No. 26). The study was conducted in accordance with the principles of the Declaration of Helsinki.
Demographic and clinical characteristics of the patients were recorded. Before the procedure, quantitative wound-culture samples were collected from all ulcers, and systemic antibiotic therapy was initiated according to the isolated strains. The wounds were washed with isotonic saline before the placement of the VAC sponge and necrotic tissue was removed. No local antiseptic agents were used for dressing changes. A VAC device (Smith & Nephew RENASYS EZ MAX NPWT device, Switzerland AG, Baar, Switzerland) was utilised at 125 mmHg continuous negative pressure for the first six consecutive applications and at 80 to 125 mmHg (mean: 100 mmHg) intermittent negative pressure for the subsequent applications.19

The wound was examined for signs of infection during every dressing change. The length and width of the wound were measured by the classical method and the results were recorded in cm.20 Wound healing, formation of granulation tissue and epithelialisation were evaluated and captured by photography. No surgical debridement or surgical corrective procedure was applied in any patient. None of the patients received venotonic agents during the hospitalisation period.

Wound healing was defined as the complete closure of the ulcer, while rapid wound healing was defined as a ≥30% reduction in the ulcer size by week four.21 Primary outcome measures included wound healing as assessed by local wound and infection examination results and wound surface area measurements at every dressing change;22 and the rate and velocity of ulcer reduction.

Statistical analysis

Statistical data were evaluated using Windows-compatible Statistical Package for Social Sciences 13.0 (SPSS 13.0) (SPSS Inc, Chicago, IL). Descriptive data are expressed as medians (interquartile ranges) or as numbers and frequencies.

Significant differences between mean values were analysed using the Mann–Whitney U-test, the Wilcoxon signed ranks test and the Kruskal–Wallis test, and subgroups with significant differences from each other were analysed using the Bonferroni corrected Mann–Whitney U-test. The chi-squared test was used to evaluate the relationships between categorical variables. In all analyses, p < 0.05 was considered significant.

Results

The mean number of VAC therapies for each case was 17.8 (range 12.4–26.8) and the mean time between dressing changes was 72.3 hours (range 61.1–83.4). The mean length of hospital stay was 32.3 days (range 24.2–38.6). The wound surfaces of all ulcers were measured before VAC therapy, and the mean length and width were found to be 9.2 cm (range 6.1–12.6) and 8.1 cm (range 3–14.2), respectively. The mean pre-procedural wound surface area was 68.2 cm² (range 22.5–154.6). Pre-procedural local wound and infection examination results and wound surface area measurements are summarised in Table 1.

Compared to baseline, the mean reduction rates of ulcers were 46.4% for the first six applications and 72.8% for the subsequent applications (Fig 3). In addition, all ulcers were colonised by a variety of bacteria at baseline (Figs 4–7). MDR P aeruginosa and methicillin-resistant Staphylococcus aureus (MRSA) were detected in three and four patients, respectively (Table 1).

Before VAC therapy, the mean dosage of antibiotic therapy was three times daily (range 1–8) for each patient. Quantitative wound culture results became negative after a mean duration of

### Table 1. Pre-procedural wound assessments and wound surface measurements (n = 14)

<table>
<thead>
<tr>
<th>No.</th>
<th>Location</th>
<th>Size and decollation (cm²)</th>
<th>Bacterial spp.</th>
<th>Wound edges/surroundings</th>
<th>Wound bed surface</th>
<th>Exudate/drainage</th>
<th>Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right medial malleolus</td>
<td>90.92</td>
<td>MSSA</td>
<td>Regular/indurated</td>
<td>Fibrinous</td>
<td>Fibrinous/moderate</td>
<td>Transient</td>
</tr>
<tr>
<td>2</td>
<td>Right lateral malleolus</td>
<td>45.15</td>
<td>Pseudomonas aeruginosa</td>
<td>Regular/erythematous</td>
<td>Fibrinous</td>
<td>Purulent/moderate</td>
<td>Persistent</td>
</tr>
<tr>
<td>3</td>
<td>Mid-anterior of right leg</td>
<td>78.98</td>
<td>Pseudomonas aeruginosa</td>
<td>Irregular/indurated</td>
<td>Fibrinous</td>
<td>Purulent/severe</td>
<td>Persistent</td>
</tr>
<tr>
<td>4</td>
<td>Left medial malleolus</td>
<td>49.51</td>
<td>MRSA</td>
<td>Irregular/indurated</td>
<td>Fibrinous</td>
<td>Serous/moderate</td>
<td>Persistent</td>
</tr>
<tr>
<td>5</td>
<td>Right lateral leg</td>
<td>53.05</td>
<td>Beta-haemolytic streptococci</td>
<td>Irregular/indurated</td>
<td>Fibrinous</td>
<td>Fibrinous/severe</td>
<td>Transient</td>
</tr>
<tr>
<td>6</td>
<td>Medial malleolus</td>
<td>83.62</td>
<td>MRSA</td>
<td>Regular/indurated</td>
<td>Fibrinous</td>
<td>Serous/moderate</td>
<td>Transient</td>
</tr>
<tr>
<td>7</td>
<td>Anterior leg, inferior</td>
<td>55.81</td>
<td>Actinomyces israeli</td>
<td>Irregular/increase temperature</td>
<td>Fibrinous</td>
<td>Purulent/moderate</td>
<td>Persistent</td>
</tr>
<tr>
<td>8</td>
<td>Anterior leg, middle</td>
<td>29.63</td>
<td>Eubacterium rectale</td>
<td>Irregular/erythematous</td>
<td>Fibrinous</td>
<td>Purulent/severe</td>
<td>Persistent</td>
</tr>
<tr>
<td>9</td>
<td>Anterior leg, middle</td>
<td>22.52</td>
<td>MRSA</td>
<td>Irregular/erythematous</td>
<td>Fibrinous</td>
<td>Fibrinous/moderate</td>
<td>Transient</td>
</tr>
<tr>
<td>10</td>
<td>Lateral leg, inferior</td>
<td>37.96</td>
<td>Escherichia coli</td>
<td>Regular/indurated</td>
<td>Fibrinous</td>
<td>Serous/moderate</td>
<td>Transient</td>
</tr>
<tr>
<td>11</td>
<td>Medial malleolus</td>
<td>66.04</td>
<td>Escherichia coli</td>
<td>Regular/macerated</td>
<td>Fibrinous</td>
<td>Serous/moderate</td>
<td>Transient</td>
</tr>
<tr>
<td>12</td>
<td>Anterior and posterior leg</td>
<td>154.56</td>
<td>Pseudomonas aeruginosa</td>
<td>Irregular/increase temperature</td>
<td>Fibrinous</td>
<td>Purulent/moderate</td>
<td>Persistent</td>
</tr>
<tr>
<td>13</td>
<td>Medial, middle</td>
<td>145.88</td>
<td>MRSA</td>
<td>Irregular/erythematous</td>
<td>Fibrinous</td>
<td>Fibrinous/moderate</td>
<td>Persistent</td>
</tr>
<tr>
<td>14</td>
<td>Medial, inferior</td>
<td>41.17</td>
<td>Propionibacterium acnes</td>
<td>Irregular/erythematous</td>
<td>Fibrinous</td>
<td>Purulent/moderate</td>
<td>Persistent</td>
</tr>
</tbody>
</table>

MRSA: methicillin-resistant Staphylococcus aureus; MSSA: methicillin-sensitive Staphylococcus aureus.
Fig. 4. Infected venous leg ulcer and complete wound healing after VAC therapy.

Fig. 5. Pre-procedural image of venous leg ulcer and granulation formation and epithelialisation after VAC therapy.

Fig. 6. Granulation tissue and epithelialisation phase after VAC therapy.

Fig. 7. Pre-procedural image of venous leg ulcer on medial malleolus and complete wound healing after VAC therapy.
VAC of 12.1 days (range 5–21) (mean number of VAC sessions 4.2). None of the patients needed antibiotic therapy until the VAC therapy was completed.

The mean duration of leg ulcers was 7.2 months. Of all the ulcers, recurrence was observed in one case twice and in another three times. Five of the ulcers were newly diagnosed.

Discussion

In this study, we examined the effect of VAC therapy on venous stasis wound healing in patients with chronic venous leg ulcers. Our study results showed that VAC therapy was an effective and rapid method of treating venous leg ulcers and had an accelerated wound-healing effect.

Negative pressure was first used for wound healing in 1993 by Fleischmann et al. Approximately 70% of chronic leg ulcers are caused by venous diseases. The underlying aetiology of venous insufficiency is venous hypertension. However, its pathogenesis, ranging from valve regurgitation to ulceration, remains unclear.

Clinical symptoms of venous insufficiency include oedema, lipodermatosclerosis, hyperpigmentation, hyperkeratosis and atrophic blanche (white atrophy). At a microvascular level, microlymphangiopathy, dilatations of the larger lymph vessels, dilatation and extension of capillaries, occlusion of the capillaries by microthrombi or white cells, decline in the number of functional capillaries, increase in the capillary passage, leakage of plasma proteins and red blood cells into the interstitium and accumulation of iron into the interstitium and siderophages, accumulation of fibrins, and inward growth of the fibroblasts along the fibrin fibres can occur. Most studies consider that haemodynamic alterations occurring at the microvascular level explicitly indicate venous ulceration. In our study, chronic venous insufficiency was the main aetiology of all ulcers.

Clinical studies have reported low rates of wound healing with standard treatment of venous leg ulcers. General medical management is the cornerstone of standard treatment. This is followed by clinical examination and the maintenance of adequate oxygenation and perfusion. An intervention is planned, if required. For the underlying pathology, venous surgery, endovenous laser ablation, radiofrequency ablation, sclerotherapy, or subfascial endoscopic perforator surgery may be applied. In certain cases, compression therapy is useful with local wound care.

In a retrospective, multicentre study, 68 patients with venous leg ulcers were treated with serial debridement, and complete wound closure was achieved in 32 (47%) patients by week 12. In the remaining centres, 298 patients were treated and complete healing was achieved in 88 (30%) patients by week 12. In our study, none of the patients received surgical debridement. The mean reduction rate of ulcers was 46.4% by week four, indicating a greater reduction in a shorter time compared to the data in the literature.

The most frequently isolated micro-organisms in infected venous leg ulcers are Pseudomonas aeruginosa, Staphylococcus aureus and haemolytic streptococci. Surgical debridement followed by wound dressing with topical antibiotics and antimicrobials is recommended for the management of bacterial bioburden. In a Cochrane Review of 25 published studies, there was a statistically significant result in favour of cadexomer iodine compared to standard care in the frequency of complete healing at six weeks. In addition, there was some evidence to show a reduction in the bacterial bioburden in patients with infected venous leg ulcers. In another study, increased blood flow reduced interstitial tissue oedema and eliminated harmful bacterial enzymes, promoting the wound-healing process and quality.

In our study, all ulcers were infected at baseline. Consistent with the literature, MDR P. aeruginosa and MRSA were isolated in three and four patients, respectively. In addition, current consensus guidelines recommend antibiotic treatment for a minimum of two weeks unless persistent evidence of wound infection is present. In our study, the mean duration of antibiotic therapy was 4.5 days (mean: three to 12 days) for the first six applications.

Quantitative wound culture results became negative after a mean VAC duration of 12.1 days and none of the patients needed antibiotic therapy until the end of the VAC therapy. The wound culture became negative five days after the initiation of VAC therapy (two sessions) in three patients. In our study population, antibiotics reduced the bacterial colonisation and bioburden in the wound site and removed the need for surgical debridement. Additionally, no local antiseptic agents were used for dressing changes.

During the third dressing change with a sterile adhesive bandage, the new formation of granulation tissue and surroundings was evident. No bacterial growth was detected in subsequent VAC applications until the end of the therapy. Our application seems to be consistent with the literature, suggesting the use of antibiotics for a minimum of two weeks. Although the wounds were closed with dressings, VAC therapy was completed without the need for surgical debridement and further antibiotic therapy in all cases.

In a study analysing 679 swabs of 285 patients with venous leg ulcers, 76.1% gram-positive and 58.2% gram-negative bacteria were isolated. In our study, gram-negative bacteria were found to be associated with more pain and were isolated in eight ulcers. These patients reported a higher pain severity, consistent with the literature.

High-pressure (30–40 mmHg) compression stockings are recommended for venous leg ulcers. In a Cochrane Review of 19 databases and two randomised, controlled trials, the authors investigated the effect of compression stockings in preventing the recurrence of venous ulcers. Half of the patients were unable to wear compression stockings on their own, with a 30 to 65% non-compliance rate. In addition, compression therapy was found to be more effective than no compression therapy, high-pressure compression stockings were more effective than low-pressure stockings, and multi-layer compression bandages were more effective than single-layer bandages. In over 400 patients with ulcer healing, the continuous use of compression stockings reduced the recurrence rate after three to five years of follow up.

In a randomised, controlled study, the healing and recurrence rates after treatment with compression with or without surgery were evaluated in patients with leg ulcers. Venous duplex imaging of ulcerated or recently healed legs in 500 patients from three centres was performed, and the recurrence rate was found to be significantly lower at four years in the combination-therapy group than in the compression-alone group (24 vs 52%, respectively). However, up to 20% of leg ulcers showed no healing by 50 weeks after compression therapy.
In another study, the likelihoods of wound healing were only 22% for large ulcers and 71% for smaller ulcers with long-term compression therapy. In our study, none of the patients was instructed to wear compression stockings during VAC therapy. However, given that compression therapy reduces the ulcer recurrence rate, all patients in our institute were recommended to wear high-pressure (30-40 mmHg) compression stockings continuously throughout the day at the end of the treatment.

Previous studies have demonstrated that venous leg ulcers affect 0.76% of males and 1.42% of females in adults aged between 65 and 95 years. In our study, all patients were males. Furthermore, possible risk factors for venous leg ulcers include advanced age, female gender, familial history of leg ulcers, white race, obesity, history of DVT or phlebitis, previous severe traumatic leg injury, chronic lower-limb oedema, sedentary lifestyle, and a job requiring prolonged standing. Although venous leg ulcers more frequently affect elderly individuals, the first diagnosis was made at the age of 40 years in 22% and at the age of 30 years in 13% of patients.

In their study, Taylor et al. found advanced age and male gender to be significant predictors of poor outcomes. Similarly, in our study, the oldest case was 79 years old, in whom diabetic foot and venous leg ulcers had recurred three times within the previous 18 months. In another study, bacterial colonisation was associated with delayed wound healing in patients with venous leg ulcers, and beta-haemolytic streptococci and anaerobic bacteria were the main culprits of delayed healing. Consistent with these findings, beta-haemolytic streptococci were isolated in one of our patients with the longest healing time. This case was also the oldest patient, with diabetic foot ulcer. He received 21 sessions of VAC and antibiotic therapy twice daily for 15 days. Nonetheless, we consider that the aetiology of delayed wound healing is multifactorial in venous leg ulcers.

A previous history of leg trauma is considered a poor prognostic factor for leg ulcers. In our study, one patient had a history of leg trauma and the ulceration did not respond to previous standard treatment, with the longest ulceration time (12 months). In addition, arterial pathologies were detected in 25% of venous leg ulcers. In our study, one patient with peripheral arterial insufficiency (ABI < 0.8) was excluded, to obtain more reliable results. VAC therapy is associated with an increased risk for thromboembolism and is contra-indicated in such patients. In our study, we also excluded two patients with a history of DVT and one patient with acute DVT.

Documentation of the characteristics of the wound bed and the wound site is of utmost importance to evaluate the healing process and the treatment response. Wound assessment requires an accurate and reproducible measurement. It is also critical for evaluation of the response rates. In their study, Kantor et al. examined the relationship between planimetric wound area and simple wound measurements and found that the correlation between simple values and planimetric area was significantly decreased for wounds > 40 cm².

In our study, we used simple measurements, and the baseline ulcer area was found to be 68.2 cm² (mean baseline length 9.2 cm, mean baseline width 8.1 cm). Additionally, wounds with < 30% healing within the four-week treatment period were classified as hard-to-heal wounds by week 12; such patients should be re-examined for accurate diagnosis and treatment.

In our study, the mean wound surface area was measured as 29.4 cm² at the end of the six VAC applications by week three, indicating a 42.7% reduction. The reduction velocity was the highest within the first two weeks of VAC therapy, which can be attributed to the necrotic tissue-reducing and tissue oedema-reducing effects of VAC. Until week four, the mean time to reduction was slower, however, formation of the granulation tissue increased due to the new tissue formation in the wound bed induced by VAC during this period. The epithelialisation phase covering the wound surface continued in week four.

In a similar study showing the efficacy of VAC therapy, a total of 60 patients with chronic leg ulcers were equally randomised to either VAC therapy or conventional wound care. Among the patients receiving VAC therapy, split-thickness skin grafting was performed for wound-bed preparation. In these patients, the median preparation time was reduced by 58%, and the overall complete healing time was reduced by 35.6%, compared to the conventional wound-care group. In addition, VAC therapy reduced the treatment-related cost by 28.8% and the total nursing time by 39.9%. In our study, we achieved a 42.7% reduction in ulcer size at three weeks with VAC therapy, indicating rapid wound healing.

The main limitation of this study is the use of a classical wound-measurement technique, which might have underestimated the accurate length and width over time, depending on the change in the wound shape. Therefore, further studies should use maximum vertical length and diameter (diameter and tissue measurement) measurements for the wound surface. Additionally, although it is not feasible in daily clinical practice, three-dimensional images can be obtained using sophisticated systems such as stereophotogrammetry.

Conclusion

VAC therapy is an effective and safe option for the treatment of complicated and hard-to-heal wounds and venous leg ulcers. In our study, VAC therapy enhanced wound healing, reduced the bacterial bioburden and the need for antibiotic therapy, and promoted the regression of infection. Based on these study findings, we suggest that VAC therapy is an effective and suitable tool for the treatment of venous leg ulcers.

References


