The role of compression therapy in the treatment of venous leg ulcers

Paulina Mościcka^{1,2,A–D,F}, Maria T. Szewczyk^{1,2, A–C,E,F}, Justyna Cwajda-Białasik^{1,2,A–C,E,F}, Arkadiusz Jawień^{3,A–C,E,F}

¹ Department of Surgical Nursing and Treatment of Chronic Wounds, Department of Health Sciences, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

² Department of Vascular Surgery and Angiology, Antoni Jurasz University Hospital No. 1 in Bydgoszcz, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

³ Department of Vascular Surgery and Angiology, Antoni Jurasz University Hospital No. 1, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation;

D – writing the article; E – critical revision of the article; F – final approval of the article

Advances in Clinical and Experimental Medicine, ISSN 1899–5276 (print), ISSN 2451–2680 (online)

Adv Clin Exp Med. 2019;28(6):847–852

Address for correspondence

Paulina Mościcka
E-mail: moscicka76@op.pl

Funding sources

None declared

Conflict of interest

None declared

Received on July 2, 2016

Reviewed on August 14, 2016

Accepted on October 20, 2017

Published online on August 7, 2018

Abstract

Epidemiological data regarding venous leg ulcers, specifically low healing rates and frequent recurrences (occurring in 20–70% of the cases), seems surprising regarding recent progress in the management of this complication. The aim of this review is to present the current state of knowledge on venous leg ulcer management, especially compression therapy. Treatment of venous ulcers should be comprehensive and well-organized, based on modern standards and up-to-date, and should involve elaborated treatment strategies. A thorough diagnostic process followed by adequate treatment may result in marked improvement of the outcomes, with up to 67% healing rate at 12 weeks and up to 81% within 24 weeks. Continuation of therapeutic activities after the ulceration has been healed is reflected by a marked decrease in the recurrence rates, down to 16% whenever the patient is actively involved in the therapeutic process. Furthermore, early diagnosis and appropriate causal treatment may prevent progression of the illness.

Key words: venous ulcers, compression therapy, recurrent ulcers

Cite as

Mościcka P, Szewczyk MT, Cwajda-Białasik J, Jawień A.
The role of compression therapy in the treatment of venous leg ulcers. *Adv Clin Exp Med.* 2019;28(6):847–852.
doi:10.17219/acem/78768

DOI

10.17219/acem/78768

Copyright

Copyright by Author(s)

This is an article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

Introduction

Crural venous ulcers constitute a serious health, social and economic problem. A multicenter study on the epidemiology of venous diseases carried out in Poland demonstrated that 1.26% of patients suffer from venous ulceration and another more than 2% have recovered from this condition.¹ Approximately 80% of lower extremity ulceration cases are a consequence of chronic venous insufficiency (CVI).^{2,3} Healing of venous ulcers is complex and lasts from a few weeks to several years. Treatment should include multidirectional and interdisciplinary activities aimed at decreasing/reduction of venous hypertension.^{4,5} Compression therapy is the gold standard in the treatment of crural venous ulcers, as it reduces vein diameter and stimulates venous drainage.^{6,7} In line with current recommendations, continuation of causal treatment and sustaining physiological blood pressure values are recommended after the ulcers have healed completely.^{3,7} Conservative treatment, without compression therapy and venous hypertension control, is associated with high risk for recurrent ulceration.^{8,9} Epidemiological data implies that recurrence may occur in 26–70% of the patients.^{10–12}

Improper venous outflow results from a dysfunction of 1 or, more often, a few causative mechanisms of venous circulation: valve efficiency, vein patency, vessel wall tension, and muscle pump performance.^{13,14} Valve insufficiency causes the blood to recede in a peripheral direction after a momentary movement upwards during muscle performance. This takes place during the diastolic phase, when valve closure is the only mechanism preventing the blood from flowing backwards. The presence of non-adapted valves is the cause of primary deep vein failure. On the other hand, past thrombotic process is most frequently responsible for the secondary damage of valves.^{4,15}

Another potential cause of disorders in venous outflow is the closure of deep vein lumen, which results in a complete and/or partial obstruction in the deep venous system. The only way of venous return are superficial veins that become dilated and overloaded due to an enhanced perfusion. Venous dilation may result in valve insufficiency, development of varicose veins in the superficial system and reverse flow in perforating veins. At the same time, deep vein thrombosis results in recanalization which leads to the damage and dysfunction of the vascular wall and valves.¹⁵ The changes of the vascular wall lead to dilation of the vein, which, as a result, causes valve cusps to move away from each other. This results in venous reflux and venous hypertension, and eventually leads to varicose vein development.¹³

Disorders in venous blood outflow may be also associated with muscle pump dysfunction. The most likely reason of the latter is muscle atrophy (for example, due to arthritis or physical inactivity), which reduces the compression force necessary to overcome gravity, stimulate the flow and prevent long-term blood retention.¹⁶

The occurrence of 1 and often a few of the dysfunctions leads to reflux and retention of blood in 1, 2 or 3 venous systems and, as a consequence, to the pathological increase in hydrostatic pressure to more than 90 mm Hg, i.e., so-called venous hypertension. This is particularly evident in the area of the distal perforating veins that connect these 2 systems. As a result, the most severe skin lesions develop in the area of the medial malleolus.^{14,17}

Clinical manifestation of chronic venous insufficiency

Clinical manifestation, along with the degree of development of chronic venous disorders (CVD), more advanced changes in the form of CVI and ulceration, is presented by CEAP classification (C – clinical, E – etiology, A – anatomic, P – pathophysiologic). The clinical picture of CVI comprises the occurrence of varicose veins, edema, atrophic skin lesions, and subjective afflictions such as pain, muscle cramps, paresthesia, and itchiness, in the advanced form of venous ulcers. It depends on the kind of pathology occurring in the venous system, the pace of the changes, hemodynamic disorders, duration of the illness, and also on subjective sensations reported by the patient.¹⁸

A varicose vein is defined as a vein which has changed its shape, course or elongation. In the initial stage, only cosmetic effects are visible in the form of telangiectasia – the dilation of intradermal veins to 1 mm, and in reticular veins – as an intradermal dilations to 4 mm, which can be a sign of the early stage of venous insufficiency. These effects can occur individually or create clusters in the form of “a sprawling shrub”. They can be located in any place, e.g., on the side of a thigh, in the popliteal fossa or in the area of the medial malleolus, where their radial composition is referred to as corona phlebectatica.^{4,18–20} Larger varicose veins emerge most frequently (90%) from inflows or trunks of the great saphenous vein; cases of varicose veins of small saphenous vein are less visible (5%).^{18,19}

Edema occurs in about 50% of patients with CVI. It is a clinical sign of an increased volume of extravascular extracellular fluid, the presence of which can be confirmed if, after pressing the skin with a thumb for 10 s, an imprint is visible.²¹ In the initial phase, edema recedes after a night's sleep, but increases during daytime proportionately to physical exertion and the time spent in a sitting or standing position or under the influence of high temperature.²² After a couple of years, protrusions appear in the area of the edema.²² Atrophic skin lesions are, apart from edema and developing varicose veins, the earliest signs of venous hypertension. The characteristic area where they occur is located above the ankles, most frequently on the shin medial surface.^{4,23,24} The clinical picture of these lesions is diverse: from skin discoloration and inflammation to hard-to-heal ulcers. The 1st symptom of skin lesions is intensified hemosiderosis.^{3,18,24} Color changes, the

so-called skin lesions, are accompanied by changes in the thickness of the skin and subcutaneous tissue. The skin becomes thin, less elastic, hard, and dry. Such skin condition on the shins is defined as lipodermatosclerosis.^{18,24} In the case of regular skin discolorations, areas of microcirculatory vessel atrophy can occur, called Milian's white atrophy (atrophie blanche), which are characterized by the presence of white, very delicate and thin skin with visible peripheral vessels.³ In the next stage, fibrosis causes accretion of the fibrous ring above the ankles, and the skin becomes more vulnerable to infections and local allergic reactions.^{2,18}

In about 1% of the patients, ulceration appears within the area of the skin lesions. It can emerge due to superficial, deep and perforating vein insufficiency, any 2 or all of them simultaneously.^{3,18,25} Similarly to skin lesions, ulceration is most frequently located in the area of the medial malleolus, i.e., in the area which is the most vulnerable to the influence of venous hypertension.^{3,26} Ulceration can emerge as a result of, e.g., injury, skin irritation or scratching of the skin due to itchiness.

The clinical symptoms of CVI are diverse and to a large extent depend on the subjective attitude of the patient, stage of disease development, environmental and cultural conditions, as well as the patient's personality, and her/his psychosocial needs. Among the reported symptoms, there are: discomfort caused by the cosmetic defect, sensation of heaviness and/or fullness, and distension of lower limbs (especially in a sitting position). Skin itchiness, prickling, paresthesia, a burning sensation and muscle cramps are also reported by the patients. In the advanced form of the disease, symptoms are even more acute and, additionally, severe pain occurs, especially in the case of venous ulceration.^{2,4,18,27} People with a long history of ulceration or with frequently recurrent ulceration suffer from various forms of depression of diverse intensity.^{4,27–30}

Venous ulcers: Clinical aspects

Venous ulceration is the most frequent cause of chronic wounds located within the lower limbs. Active and healed ulcerations occur in 0.3 up to ca. 3% of the adult population. Ulcers occur twice as often in women (especially in their 40s) as in men, who are affected by the problem 10 years later, on average. The highest morbidity occurs between the age of 50 and 80.¹ According to the CEAP classification, venous ulceration is defined as a deficiency of full-thickness skin, usually in the area of the ankles, which has no tendency of idiopathic healing and is maintained by venous disorders.³¹ It is most frequently (74%) located in the area of the medial malleolus, but it can also occupy other surfaces.⁴ The small, shallow wound can reach even enormous size, encircling the entire shin. Ulceration is usually of oval shape with a flat bottom covered with necrotic tissue, fibrin clusters and, if there is an infection, also with pus.^{2,4} If a proper healing process takes place, the

ulceration has flat irregular edges, and in wounds which have been present for years, a thick, shaft-shaped edge may be present.^{2,32} The skin surrounding the ulceration also looks different, as it gets thicker and dry.³³

Venous ulceration treatment, apart from invasive treatment which is introduced with patients with no contraindications to a surgical procedure, includes numerous elements of conservative therapy. The basic aim of this treatment is the improvement of conditions of the local circulation, and reintroduction of proper nutrition and oxygenation of tissues.^{34–36} It requires undertaking actions connected with compression therapy, i.e., limitation of the influence of venous hypertension, limitation of the processes of inflammation and wound infections, as well as many comprehensive actions, such as venous system diagnostics (Duplex scan, ankle-brachial index), causative therapy – compression therapy, topical treatment, physiotherapeutic procedures (sequential pneumatic massage/manual massage – edema reduction), physical activity, prophylaxis, limb care and hygiene, educating the patient, using analgesic, phlebotropic and rheologically active drugs, weight reduction, supplementing of deficiencies, and high-protein diet.^{4,36,37}

Compression therapy in the treatment of venous leg ulcers

Compression therapy is the gold standard in the treatment of venous ulcers. It involves application of gradual, external and layer pressure with the highest pressure in the area of ankle and the lowest pressure under the knee, using special bandages or ready-to-use layered compression bandage systems. Proper systematic application of compression therapy reverses the pathological changes of the venous system, i.e., “venous hypertension”, which are the cause of ulceration, and improves ulceration healing conditions.^{3,7,36,38} The narrowing of the vessel lumen leads to a decrease of the volume of venous blood in a limb and speeds up its flow. It also reduces the painfulness of ulceration and edema. There is also an improvement in the activity of the calf's muscle pump, condition of the skin and subcutaneous tissue.^{39,40} Among the methods used in compression therapy in venous ulceration treatments, we can distinguish: short-stretch bandages, elastic bandages, compression stockings and other knitwear products, as well as devices generating dynamic compression/intermittent pneumatic compression.^{3,4} The most suitable compression material in the treatment of active ulceration are short-stretch bandages put on in a 2- or multi-layer system.^{7,41} However, in the case of small-area ulceration, ready-to-use compression products are the most suitable solution. The opinion of experts on the matter of compression therapy is unequivocal and says that in venous ulceration treatment, the recommended pressure in the ankle area is 40 mm Hg and 17–20 mm Hg under the knee.^{3,7,36}

Effective and safe compression therapy depends on the correct application, which involves the proper theoretical and practical preparation of specialist nurses. Compression therapy should not be performed by unqualified people.

Local treatment of venous leg ulcers

Venous ulcer care involves optimization of the micro-environment of the wound's background and includes only actions reflecting the physiological course of healing. The TIME strategy to a great extent contributes to the stimulation of natural healing mechanisms and includes the following elements of ulceration debridement: T – tissue debridement, I – infection and inflammation control, M – moisture balance, and E – epidermization stimulation.^{3,7}

In accordance with the TIME strategy recommended by scientific societies, wound debridement involves all the procedures performed within the wound connected with deterging, as well as eliminating the exudate, damage and contamination occurring within the wound bed (e.g., foreign bodies, dirt, sand, or splinters), bacteria and other germs, tissues that are necrotic and have limited blood supply, and pus.^{42–44} There are various methods of ulceration debridement, e.g., surgical, enzymatic, autolytic, mechanical, the use of biosurgical methods, negative pressure, and ultrasound.^{3,7,44}

Preventing recurrence of venous leg ulcers

The healing of the ulceration does not mean the pathology in the venous circulation system is cured. The main risk factor of ulceration recurrence is the continuous influence of venous hypertension, which is most frequently caused by CVI and post-thrombotic syndrome. Recurrence risk factors are most often divided into local and general.^{5,10,40}

Local factors include: 1. no compression therapy after the healing of the ulceration – insufficient knowledge and neglect on the part of medical staff; 2. lack of cooperation of the patient during the compression therapy, e.g., a low level of knowledge and motivation of the patient, confidence in the ineffectiveness of compression in the prevention of recurrence, treating the healing as a complete recovery, confidence about other causes of recurrence, difficulties in putting on and the inconvenience connected with wearing compression stockings, and lack of cooperation on the part of the patient's family/caregiver; 3. lack of systematic application of compression therapy; 4. decrease in tolerability of compression therapy (improper degree of compression), and 5. orthopedic disorders: numbness of ankle joint, improperly fitted shoes, etc.

Systemic factors influencing the risk of ulceration recurrence include obesity, arterial hypertension, improper

nutrition (including protein-energy deficit), lack of proper exercise stimulating ankle joint mobility, increasing venous return, smoking, alcoholism, post-thrombotic syndrome, and others.^{8–10,33,45–47}

After the healing of ulceration, the monitoring of the patient and the constant maintenance of low pressure values are required. That is why compression therapy is recommended after the healing of ulceration.⁴⁸ Educating the patients and their family is an important part of the therapy and prophylaxis. It is the kind of action that aims at changing the role of the patient from a passive task performer to a partner, who will consciously and actively take part in the healing process. Customized and systematic education enables the patient to understand the core of the problem, which is a condition for good cooperation between the patients and the therapeutic team. This can be the basis of the effective healing process.^{39,49–51}

Conclusions

Compression therapy is the gold standard in the treatment of venous ulcers and results in the highest healing rates. It involves employing external and layered pressure, using special bandages, ready-to-use layered compression bandage systems, and, in the case of small ulceration which does not weep, compression stockings.^{3,7,36,52,53} A systematic literature review shows that every form of properly employed compression favorably influences the process of venous ulceration healing, and it is difficult to determine which method is the most effective.^{25,34,54} In the research carried out by Szewczyk among 112 people suffering from venous ulcers, the healing dynamics of ulceration treated with 2- and 4-layer compression system was comparable.⁴ In another study conducted in an ulceration treatment clinic on a group of 46 patients with venous ulcers, the authors demonstrated a similar effectiveness of the 2- and 4-layer system as well as compression stockings.⁵⁵ In yet another study, a group of 134 patients with wounds of venous etiology was randomly divided into 2 groups.⁴¹ In one group, individually selected compression stockings were used, and in the other group, short-stretch bandages were applied to compare their effectiveness. In the group where stocking were used, the rate of healing and the time of healing were higher. On the other hand, another study, which included 200 patients with ulceration of venous etiology, compared the effectiveness of 4-layer compression therapy and elastic bandages.⁵⁶ The final analysis demonstrated that 4-layer compression was more effective. Customized compression and pressure degree significantly improve the conditions existing in venous circulation and microcirculation, if applied constantly.

In the majority of cases of venous ulcers, conservative treatment in line with the standards brings good results if the care is based on systematic interdisciplinary actions. If the care is occasional, the ulceration lasts longer and the

rate of recurrence is high. The most important predictors include the duration of ulceration and its initial area. It has been proved that ulcerations characterized by short duration (<6 months) and small initial area (<5 cm²) have the highest (95%) chance of healing within a period shorter than 24 weeks. It takes much more time for developed and extensive ulcers to heal.^{4,32,57,58}

The care of a patient with CVI does not end with the healing of the ulceration. In order to maintain skin continuity, actions are required to be undertaken by both the patient and the therapeutic team. No or insufficient participation of one of the parties may lead to another ulceration. Epidemiological studies show high rates of recurrence which are about 26–70%.^{12,32,57,58} According to the study data, about 26% of ulcers recur within the 1st 12 months after the therapy is over.²⁶ Regularly controlled and constantly monitored patients can significantly extend the period of remission, or even avoid ulceration recurrence.⁵⁹ It is thought in the scientific community that the most effective method of prevention is the continuation of compression therapy. Although there are still no high-quality studies comparing recurrence rates depending on application/no application of compression, experts unequivocally emphasize the high effectiveness of compression therapy in both the treatment of ulcers and the prevention of recurrence.^{3,7,60–62}

To summarize, systematic application of properly customized compression therapy in the form of special bandages or ready-to-use compression products remains the gold standard in the conservative treatment of venous leg ulcers. Compression therapy is recommended in order to prevent recurrence as well as emergence of recurring ulceration in the case of a healed ulceration.^{3,7}

References

- Jawień A, Grzela T, Ochwat A. Prevalence of chronic venous insufficiency in men and women in Poland: Multicentre cross-sectional study in 40,095 patients. *Phlebology*. 2003;18(3):110–122.
- Negus D, Coleridge Smith P. Diagnostyka różnicowa owrzodzeń podudzi. In: Negus D, Coleridge Smith P, Bergan J, eds. *Owrzodzenia Podudzi – Diagnostyka i Leczenie*. Bielsko Biala, Poland: Alfa Medica Press; 2006:90–105.
- Szewczyk M, Jawień A, Andruszkiewicz A, et al. Zalecenia specjalistycznej opieki nad chorym z owrzodzeniem żylnym goleni. *Pielęg Chir Angiol*. 2007;3:95–138.
- Szewczyk MT. Ocena Dynamiki Procesu Gojenia Owrrzodzeń Żylnych z Zastosowaniem Kompresjoterapii i Holistycznego Modelu Opieki Pielęgniarskiej. Bydgoszcz, Poland: Wydawnictwo Naukowe Uniwersytetu Mikołaja Kopernika; 2006.
- Szewczyk MT. Wybrane aspekty opieki nad chorym z owrzodzeniem żylnym. In: Jawień A, Szewczyk MT, eds. *Kliniczne i Pielęgniarskie Aspekty Opieki nad Chorym z Owrrzodzeniem Żylnym Goleni*. Poznań, Poland: Wydawnictwo Medyczne Termedia; 2008:125–131.
- Szewczyk MT, Jawień A. Kompresjoterapia jako podstawowy element leczenia owrrzodzeń żylnych. *Leczenie Ran*. 2004;1:1–4.
- Jawień A, Szewczyk M, Kaszuba A, et al. Wytyczne grupy ekspertów w sprawie gojenia owrrzodzeń żylnych goleni. *Leczenie Ran*. 2011;8: 59–80.
- Jawień A, Szewczyk MT. Profilaktyka trzyczorzędowa w zapobieganiu nawrotowym owrrzodzeniom żylnym goleni. *Prz Flebol*. 2005;13(6): 223–268.
- Szewczyk MT, Cwajda-Bialasik J, Jawień A. Prevention of recurrent venous ulceration. *Postepy Dermatol Alergol*. 2012;29(4):308–312.
- Gohel MS, Taylor M, Earnshaw JJ, Heather BP, Poskitt KR, Whyman MR. Risk factors for delayed healing and recurrence of chronic venous leg ulcers: An analysis of 1324 legs. *Eur J Vasc Endovasc Surg*. 2005;29(1): 74–77.
- Nelson EA, Harper DR, Prescott RJ, Gibson B, Brown D, Ruckley CV. Prevention of recurrence of venous ulceration: Randomized controlled trial of class 2 and class 3 elastic compression. *J Vasc Surg*. 2006;44(4):803–808.
- Finlayson K, Edwards H, Courtney M. Factors associated with recurrence of venous leg ulcers: A survey and retrospective chart review. *Int J Nurs Stud*. 2009;46(8):1071–1078.
- Migdalski A, Ciecierski M, Jawień A. Fizjologia i patofizjologia odpływu żylnego. *Przew Lek*. 2004;8(7):33–35.
- Szotkiewicz A, Jawień A. Patogeneza owrrzodzeń żylnych goleni. In: Jawień A, Szewczyk MT, eds. *Owrzodzenia Żylne Goleni*. Warszawa, Poland: Wydawnictwo Medyczne "Twoje Zdrowie"; 2005:11–19.
- Zapalski S. Anatomia i fizjologia układu żylnego kończyn dolnych. In: Zapalski S, ed. *Niewydolność Układu Żylnego Kończyn Dolnych*. Gdańsk, Poland: Via Medica; 1999:27–34.
- Gravereaux EC, Donaldson MC. Niewydolność żylna. In: Adamiec R, ed. *Choroby Naczyni. Podręcznik Towarzyszący do Braunwald's Heart Disease*. Lublin, Poland: Wydawnictwo Czelej; 2008:908–918.
- Szewczyk MT, Jawień A. Wybrane aspekty zachowawczego leczenia owrrzodzeń żylnych. Część I: Kompresjoterapia. *Postepy Dermatol Alergol*. 2005;3:133–140.
- Ciecierski M. Obraz kliniczny przewlekłej niewydolności żylniej. In: Jawień A, Szewczyk MT, eds. *Owrzodzenia Żylne Goleni*. Warszawa, Poland: Wydawnictwo Medyczne "Twoje Zdrowie"; 2005:51–57.
- Jawień A, Ciecierski M, Piotrowicz R. Przewlekła niewydolność żylna. *Przew Lek*. 2000;1(3):7–75.
- Chi YW, Raffetto JD. Venous leg ulceration pathophysiology and evidence-based treatment. *Vasc Med*. 2015;20(2):168–181.
- Ciecierski M, Grzela T, Miszewski M. Obrzęki kończyn dolnych. In: Jawień A, Szewczyk MT, eds. *Kompresjoterapia*. Poznań, Poland: Wydawnictwo Medyczne Termedia; 2009:29–68.
- Wiktor M. Patofizjologia. In: Chęciński P, ed. *Obrzęk chłonny*. Poznań, Poland: Wydawnictwo Medyczne Termedia; 2010:27–41.
- Skórski M. Przewlekła niewydolność żylna. In: Pasierski T, Gaciong Z, Torbicki A, Szmidt J, eds. *Angiologia*. Warszawa, Poland: Wydawnictwo Lekarskie PZWL; 2004:362–368.
- Szewczyk MT, Jawień A, Cwajda J. Zaburzenia integralności skóry u chorych z przewlekłą niewydolnością żylną. *Postepy Dermatol Alergol*. 2005;3:141–147.
- Danielsson G, Eklof B, Grandinetti A, Lurie F, Kistner RL. Deep axial reflux, an important contributor to skin changes or ulcer in chronic venous disease. *J Vasc Surg*. 2003;38(6):1336–1341.
- Nelson EA, Bell-Syer SE. Compression for preventing recurrence of venous ulcers. *Cochrane Database Syst Rev*. 2012;15(8):CD002303.
- Vandenberg EG, Hopman WM, Carley ME, Kuhnke JL, Harrison MB. Leg ulcer nursing care in the community: A prospective cohort study of the symptom of pain. *BMC Nurs*. 2013;12:1472–6955.
- Szewczyk M, Jawień A, Hildebrandt Z. Problemy chorych z przewlekłą niewydolnością żylną. Psychospołeczne konsekwencje choroby – część II. *Prz Flebol*. 2005;13(4):99–107.
- Szewczyk MT, Jawień A, Hildebrandt Z. Subiektywne postrzeganie stanu zdrowia chorych z przewlekłą niewydolnością żylną – część I. *Prz Flebol*. 2005;13(4):175–181.
- Szewczyk MT, Rogala J, Cwajda J, Cierzniańska K, Jawień A. Psychological and social problems of patients with chronic venous disease. *Postepy Dermatol Alergol*. 2007;24:207–210.
- Eklof B, Rutherford RB, Bergan JJ, et al. Revision of the CEAP classification for chronic venous disorders: Consensus statement. *J Vasc Surg*. 2004;40(6):1248–1252.
- Phillips TJ, Machado F, Trout R, Porter J, Olin J, Falanga V. Prognostic indicators in venous ulcers. *J Am Acad Dermatol*. 2000;43(4):627–630.
- Heinen MM, Persoon A, van de Kerkhof P, Otero M, van Achterberg T. Ulcer-related problems and health care needs in patients with venous leg ulceration: A descriptive, cross-sectional study. *Int J Nurs Stud*. 2007;44(8):1296–1303.
- Kramer SA. Compression wraps for venous ulcer healing: A review. *J Vasc Nurs*. 1999;17(4):89–97.
- Johnson S. Compression hosiery in the prevention and treatment of venous leg ulcers. *J Tissue Viability*. 2002;12(2):72–74.

36. Vin F, Benigni JP. Compression therapy. International Consensus Document Guidelines according to scientific evidence. *Int Angiol.* 2004; 23(4):317–345.
37. O'Meara S, Cullum N, Nelson EA, Dumville JC. Compression for venous leg ulcers. *Cochrane Database Syst Rev.* 2012;14(11):CD000265.
38. Lorimer KR, Harrison MB, Graham ID, Friedberg E, Davies B. Venous leg ulcer care: How evidence-based is nursing practice? *J Wound Ostomy Continence Nurs.* 2003;30(3):132–142.
39. Moffat C, Harper P. *Leg Ulcer.* London, UK: Churchill Livingstone; 1997.
40. Heinen M, Borm G, van der Vleuten C, Evers A, Oostendorp R, van Achterberg T. The lively legs self-management programme increased physical activity and reduced wound days in leg ulcer patients: Results from a randomized controlled trial. *Int J Nurs Stud.* 2012;49(2):151–161.
41. Junger M, Wollina U, Kohnen R, Rabe E. Efficacy and tolerability of an ulcer compression stocking for therapy of chronic venous ulcer compared with a below-knee compression bandage: Results from a prospective, randomized, multicentre trial. *Curr Med Res Opin.* 2004; 20(10):1613–1623.
42. Szewczyk MT, Mościcka P, Hancke E, Cwajda J, Cierzniańska K, Jawień A. Use of TIME strategy in wound bed debridement: Venous leg ulcer. *Zakażenia.* 2006;6:86–89.
43. Gottrup F, Apelqvist J, Bjarnsholt T, et al. EWMA document: Antimicrobials and non-healing wounds. Evidence, controversies and suggestions. *J Wound Care* 2013;22(5 Suppl):S1–89.
44. Strohhal R, Dissemmond J, Jordan O'Brien J, et al. EWMA document: Debridement. An updated overview and clarification of the principle role of debridement. *J Wound Care.* 2013;22(1):5.
45. Graham ID, Harrison MB, Shafey M, Keast D. Knowledge and attitudes regarding care of leg ulcers. Survey of family physicians. *Can Fam Physician.* 2003;49:896–902.
46. Flaherty E. The views of patients living with healed venous leg ulcers. *Nurs Stand.* 2005;19(45):82–83.
47. Jawień A, Szewczyk MT. Specjalistyczna opieka medyczna. In: Jawień A, Szewczyk MT, eds. *Owrzodzenia Żyłne Goleni.* Warszawa, Poland: Wydawnictwo Medyczne "Twoje Zdrowie"; 2005:166–177.
48. Franks PJ, Oldroyd MI, Dickson D, Sharp EJ, Moffatt CJ. Risk factors for leg ulcer recurrence: A randomized trial of two types of compression stocking. *Age Ageing.* 1995;24(6):490–494.
49. Erickson CA, Lanza DJ, Karp DL, et al. Healing of venous ulcers in an ambulatory care program: The roles of chronic venous insufficiency and patient compliance. *J Vasc Surg.* 1995;22(5):629–636.
50. Edwards LM. Why patients do not comply with compression bandaging. *Br J Nurs.* 2003;12(11 Suppl):S5–6, S8, S10 passim.
51. Brooks J, Ersser SJ, Lloyd A, Ryan TJ. Nurse-led education sets out to improve patient concordance and prevent recurrence of leg ulcers. *J Wound Care.* 2004;13(3):111–116.
52. Anderson I. Compression hosiery to reduce leg ulcer recurrence. *Nurs Times.* 2013;109(6):18–20.
53. Mooij MC, Huisman LC. Chronic leg ulcer: Does a patient always get a correct diagnosis and adequate treatment? *Phlebology.* 2016;31(1 Suppl): 68–73.
54. Anderson I. Optimising concordance with compression hosiery in the community setting. *Br J Community Nurs.* 2015;20(2):67–68.
55. Szewczyk MT, Jawień A, Cierzniańska K, Cwajda-Bialasik J, Mościcka P. Comparison of the effectiveness of compression stockings and layer compression systems in venous ulceration treatment. *Arch Med Sci.* 2010;6(5):793–799.
56. Enoch S, Williams D, Price P, Harding P. Randomized clinical trial and economic analysis of four-layer compression bandaging for venous ulcers (Br J Surg 2003;90:794–798). *Br J Surg.* 2003;90(10):1307.
57. Margolis DJ, Berlin JA, Strom BL. Which venous leg ulcers will heal with limb compression bandages? *Am J Med.* 2000;109(1):15–19.
58. Szewczyk MT, Jawień A. Kompresjoterapia w leczeniu owrzodzeń żylnych. In: Jawień A, Szewczyk MT, eds. *Kompresjoterapia.* Poznań, Poland: Wydawnictwo Medyczne Termedia; 2009:82–95.
59. Kunitomo BT. Management and prevention of venous leg ulcers: A literature-guided approach. *Ostomy Wound Manage.* 2001;47(6):36–42.
60. Kunitomo B, Cooling M, Gulliver W, Houghton P, Orsted H, Sibbald RG. Best practices for the prevention and treatment of venous leg ulcers. *Ostomy Wound Manage.* 2001;47(6):34–46.
61. Barwell JR, Davies CE, Deacon J, et al. Comparison of surgery and compression with compression alone in chronic venous ulceration (ESCHAR study): Randomized controlled trial. *Lancet.* 2004;363(9424): 1854–1859.
62. Coleridge Smith PD. Diagnostyka anatomiczna – ultrasonografia duplex Doppler. In: Negus D, Coleridge Smith P, Bergan J, eds. *Owrzodzenia Podudzi – Diagnostyka i Leczenie.* Bielsko-Biała, Poland: Alfa Medica Press; 2006:117–125.