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Venous leg ulcers: a literature review

Abstract

The purpose of the literature review is to give the reader a general understanding of Venous Leg Ulcers. Understanding the history, pathophysiology, presentation, and treatment of venous leg ulcers is important in the approach and management of venous leg ulcers. Venous leg ulcers have existed since the 1200's and it is found that early intervention with combined treatments is more efficient in treating venous leg ulcers while also lowering the recurrence rate of the venous leg ulcers.

Keywords: Venous leg ulcer, chronic venous insufficiency, lower limb, venous pressure

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Safiyyah Manjra
Department of Medicine and Surgery, Faculty of Medical Sciences, The University of West Indies Cave Hill Campus, Medicine and Surgery, West Indies

Correspondence: Safiyyah Manrja, The University of The West Indies Cave Hill Campus, Bridgetown, Barbados, West Indies, Tel, +1 (246) 240-3300, Email safiyyah.jra@gmail.com

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Abbreviations: DVT, deep vein thrombosis; CVD, chronic venous disease; VLUs, venous leg ulcers

History

The clinical correlation between leg ulceration and varicose veins dates back to the time of Hippocrates.[1] In 1271, the first vignette on venous ulceration was documented, where a cobbler named Raoul developed deep vein thrombosis (DVT) which lead to unilateral oedema of his ankle. Raoul after developing a leg ulcer and in the 1200's was told to visit the grave site present in France of King Louis IX, Raoul gathered the dust found on the tomb of King Louis IX and placed it onto his opened venous ulcer which then healed. Raoul was reported to continue to have lived 11 years after placing dust from the tomb onto his venous ulcer.[2] The 1500 followed, where in 1509 Henry VIII, during his 38 years of ruling resulted in tyranny, temperament due to his 'sorre legge' the changes in his behaviour resulted in pressures in political sense and the cause of ulceration was thought to be the reason behind his cruelty in life, adherent personality and political rulings. Henry VIII love for sports, one day resulted in him injuring his left foot while he was playing a game of tennis, the injury resulted in a swollen foot which resorted him to wearing slippers and that same year, he was said to have a 'sorre legge' and was documented with an ulcer on the thigh. Treatment of the ulcer was done by Thomas Vicary and the ulcer was healed. By the age of 44, Henry VIII was stated to be obese, a fall from his horse in 1536 resulted in neurological injuries as well as fractured long bones. Although the wounds to his leg had healed, the recurrence of ulceration was unmanageable back in the 1536-1538. However, by this time Henry VIII wounds were bilateral, purulent, and deep, transient healing between abscesses and fistulous communications resulted in sepsis, fever and had lost urge to speak. Physicians at the time attempted to keep the wounds open to allow drainage and "lacing the ulcers with red hot pokers". However healing of the recurrent ulcers were not complete continuing to March 1541, where Henry VIII again succumbed to fever, and one of the legs left opened in attempt to save his life, treatment was administered and the fever had gone however the ulcer remained, this time Henry VIII had little exercise and refused to alter his diet further leading to obesity, and six years following Henry VIII had another episode of fever and by this time the ulcer had worsen having a strong stench near the ending of 1546 where Henry VIII was reported to being carried around in a chair as he was morbidly obese and unable to walk due to his painful legs. Henry VIII had died in January 1547 after having his last episode of fever and ulcerations of his leg resulting in rapid decline of his life.[3] In the 1700's Wiseman also made the connection between leg ulcerations and varicose veins. Gay and Spender working on a study in the 1900's found that ulcerations of the lower limb can be caused by deep veins in the leg. Further studies were done to the late 1900's which correlated findings stating that DVT frequently results in venous ulcerations and communicating veins such as the perforators of the leg, saphenous veins and posterior tibial veins can also be related to venous ulcerations.[1]

Introduction

Venous ulcers are defined as skin lesions which are opened and are situated in a particular part of the body that suffers from venous hypertension.[4] Venous ulcers are known to cause significant morbidity and studies showed that 1% of the world's population is affected by venous ulcers.[5] Venous ulcers are ubiquitous worldwide as a large proportion of the population is stated to suffer with lower-extremity ulcers, these ulcers are often chronic ulcers and treatment costs several billion dollars worldwide.[6] In the United Kingdom it was reported that £400 million per year is spent on treatment for at least 1% of the population.[7] The importance of effectively treating these venous ulcers will ensure that funds are efficiently being used and patient's lifestyles are not being deteriorated due to the cost of the treatment. Venous ulcers can present as recurrent cases and the open ulcers can last from weeks to years.[8] Studies have shown that without ongoing treatment healed venous leg ulcers do reoccur.[9] The prognosis for the healing ulcer decreases if the ulcer is present for more than 3 months and the length of the ulcer is greater than 10cm, the patient has existing lower limb disease, is elderly and has a body mass index greater than normal.[4] Majority of the venous ulcers are present in the leg and are caused by changes in blood flow in the leg, such as occlusion of the vein, incompetence of the valves of the vein resulting in high pressure present in these veins of the leg.[10] To date, the recommended treatment is treating the underlying cause of venous hypertension usually by surgical intervention and using compression therapy.[7] The pressure in the veins will be removed or repaired using surgical intervention and the compression therapy allows for further reduction in the pressure of the veins, allowing for prevention of recurrence and treatment of the venous ulcer.[10]

Epidemiology

Although there is variation seen among different studies with respect to the epidemiology of venous ulcers, the overall consensus that 1% of the population has a history of a venous ulcer.[6] Studies

showed that venous ulcers are common in the lower extremities. Studies done by Reichenberg and Davis showed that ulcers which lasted for more than 4 weeks were seen more men older than 65 years of age than in men younger than 65 years of age, hence it was shown that as age increase the prevalence of ulcers lasting greater than 4 weeks also increased. Among women similar findings were found as women increased in age so did the prevalence of ulcers, however in the women studies the age demarcation was at 45 years. Recurrent ulcers were also identified as 47% of all ulcers studied by Reichenberg and Davis. Ulcers which remain for more than 6 weeks are termed chronic ulcers and overall 58% to 70% of all ulcers are stated to be chronic ulcers.[6] In 1991, a study was done using a survey of the Lothian and Forth Valley Health Districts, both having a population over one million persons, the prevalence of chronic leg and foot ulcers was noted to be 1.48 per 1000 of the population. 600 patients were clinically evaluated and a total of 76% of legs were diagnosed with venous disease.[11] To conclude, venous ulcers are estimated to be more commonly found in women than in comparison to men.[6] A study done in the United States showed most first ulcers present before the age of 60 years and are prominently found in women than in men, however overall venous ulcers are more prevalent between the ages of 60 and 80 years.[5] However, as the men increase age, findings support that ulcers are more commonly found in men than women.[6]

Pathophysiology

Venous ulceration occurs through a series of cellular and physiological events caused by venous hypertension.[12] Two main mechanisms responsible for the formation of venous ulcers are venous reflux and obstruction.[4] Within the venous system, the main venous vessels carry blood of low resistance, hence allowing large volumes of blood to be transported with low energy use. The vein wall allows for the venous capacitance, where by as the volume increases, the vein widens and the pressure is not increased but is compensated by the widening of the vein to accommodate the volume of blood. The direction of flow is also of importance as it allows arterial inflow and cardiac output, facilitating the blood to return, and enter the venous system. The blood moves from the lower extremity veins proximally in two ways, when the negative pressures in the chest causes a gradient that initiates the flow of blood from the lower extremities to the proximal areas and when the calf muscle pumps the blood from the lower extremities to the proximal area as the ankle bends, the back flow of the blood is prevented by the valves in the vein. Alteration in the conductivity, the capacity or the direction of venous bloods leads to valve incompetence and insufficient pumping of the calf muscle pump.[12]

Valve incompetence is common in perforator veins and great saphenous vein, and is present in 80% - 100% of all recurring ulcers with increase in the rate of the healing time and 25% - 50% respectively. However, combination of superficial and perforator venous incompetence is seen 21% - 40% of patients with venous leg ulcers. Axial reflux is commonly present in persons who have chronic venous disease (CVD), this type of reflux occurs from groin to the ankle. Studies showed depending on whether the reflux is axial or segmental the severity of CVD increases, if deep vein reflux or segmental reflux becomes axial the CVD becomes more severe. The competence of venous valves differs with respect to its associated function, in venous valves, the movement of blood from the intramuscular veins is observed, in perforating veins the one-way flow of blood from deep to superficial veins is possible and if the valves are incompetent there is backflow of blood as it returns to the intramuscular vein and sinusoids interfering with the arterial flow and muscle pump. Valve incompetence is of great importance as it can pose as a major threat to formation and recurrence of venous ulcers.[12]

Obstruction in the proximal veins is seen commonly in patients who lave VLUs and their treatment is seen to be less effective than if persons have had distal vein obstruction. Relief of venous obstruction has shown more effective results in treatment hence the importance of alleviating venous obstruction in patients with VLUs. However, invasive methods are needed to detect the presence of venous obstruction as non-invasive techniques do not identify the venous obstruction. When blood flows in the veins it has a low velocity, low pressure, and high volume, this increases the sensitivity to slight variations in the changes in pressure gradients and expansion of the wall of the vein. However, if there is sclerosis of the walls of the vein, or obstruction in the wall or lumen of the vein, this can present the vein from expanding as blood flows through, hence decreasing the venous capacity, and increasing the resistance to the outflow of blood leading to increasing in pooling of the blood and lack of circulation further contributing to the development of VLUs. To determine if there is an obstruction, ascending phlebography is known to be the standard method of imaging, intravascular ultrasound is more efficient in determining the anatomic location and how far the stenosis has occurred. A study was done on patients who had venous ulcers to see the degree of obstruction, and 40% of patients who had DVT were shown to have greater than 80% of obstruction present in iliac veins. If duplex ultrasound showed signs of obstruction, it is that of outflow obstruction, and evidence showed that obstruction was mainly found in post thrombotic veins and therefore non-invasive procedures are not as effective.[12]

The calf muscle pump allows for venous blood to flow from lower limbs to the heart and blood is also able to move in both horizontal and vertical directions. A pressure gradient is created between the thigh and veins in the lower legs and causes bidirectional flow within the perforators present in the calf, which in turn distributes the pressure evenly between the deep veins and superficial veins present in the lower leg. The pressure gradient that is formed within the lower limb is known as ambulatory pressure gradient and it is to activate the calf muscle pump, however if the veins are incompetent the ambulatory pressure causes venous reflux and causes venous hypertension of the lower limb.[13] In patients if the calf muscle pump is not effective that could be the main result of venous hypertension, at times patients present with immobile ankle joint and ineffective calf muscle pump hence the venous pressure is persistent as well as the ambulatory venous pressure elevated hence leading to venous hypertension. In patients who suffer with venous ulcers they are also noted to have reduction in movement at the ankle joint this also impairs calf muscle function and causes increase in venous pressure.[12]

Factors predisposing persons to the formation of venous ulcers include, previous occurrence in family history, female gender, increase number of pregnancies, increase levels of estrogen, living sedentary lifestyle and obesity.[4] These factors also show that there is a genetic component involved in the development of these venous ulcers. Venous ulceration and genetics are linked in that there are many genetic mutations which are responsible for the formation of venous ulcers. A genetic mutation in the F13A1 gene can cause a decrease in Factor XIII, because the F13A1 gene encodes for the Factor XIII and is responsible for growth and migration of fibroblast which is responsible for wound healing. A decrease in Factor XIII is seen in large ulcers and shows a delay in healing and likewise an increase in Factor XIII due to high concentrations in F13A1 showed better healing of the ulcer. The coloration of the dermis around the surrounding venous ulcer changes when iron is deposited from

the breakdown of hemoglobin present in the red blood cells.[12] The hemochromatosis gene takes up the iron when it is released from the breakdown of hemoglobin in the red blood cells however in persons with VLUs there is a defect in the hemochromatosis HFE gene and hence iron is not absorbed as effectively as it continues to accumulate beneath the dermis of the skin.[13] With respect to non-healing ulcers, studies also showed that a defect in the fibroblast growth factor 2 also is responsible for the non-healing ulcers.[12] Specific genetic conditions which predispose persons to having venous insufficiency are Klippel-Trenaunay syndrome and Ehlers-Danlos syndrome. Other genes such as FOXC2 and MMPs have also shown to be associated with VLUs as an increase in MMPs and cytokines are seen in patients with VLUs which cause effects on the vein wall, venous valve, endothelium and the glycocalyx causing further deterioration of the dermis and development of the ulcer. Specifically, the MMPs are seen in large amounts leading to an over expression of proteinase activity and also poor healing associated with the VLUs.[13] Another study also showed an inflammatory response occurs where there is leukocyte aggregation, rupture of the endothelial cells, and aggregation of platelets surmounting to inflammatory response being mediated which causes the release of proteolytic enzymes together contributing to increase in permeability of the cells leading to intracellular edema and encouraging the development of these venous ulcers.[2] The pathophysiology plays an important role in understanding the formation of VLUs. Although there is no specific process yet identified indicating the exact pathophysiology of the venous leg ulcers.[7] Ongoing studies have demonstrated that the pathophysiology of venous leg ulcers includes an amalgamation of factors including genetics, cellular, inflammatory and structural anomalies.

Presentation

Patients who have venous leg ulcers often present with pain or dull aching in the lower limbs also associated with swelling of the lower limb and the pain worsens on standing and reduces when the lower limb is raised.[7] However, the venous leg ulcers are often differentiated depending on the clinical history, presentation and physical examination done on the patient and various findings would enable differentiation of the type of ulcer present in the lower extremity.[4] Venous ulcers are often present over bone like projections of the body for example over the medial malleolus, which is actually called Gaiter area and is a common spot for venous ulcers.[2] The venous ulcers can appear as small or circumferential, the ulcers have an irregular shape, are shallow, and granulation tissue or fibrous tissue with sloughy tissues is usually found in the base of the ulcer.[2,8] The venous ulcers do not reach deep to the muscle layer, muscle fascia or bone and the boarders are well defined.[4,5] The venous ulcers appear to have red granulation or yellow fiborous tissue or at times can show necrosis and appear black. Surrounding the venous ulcer the tissue can be of brown colour or be hyperpigmented, the hyperpigmentation can be a cause of hemosiderin deposition. There can also be signs of lipodermatosclerosis, which can present in the acute form or chronic form. Over time fibers begin to form from the subcutaneous tissue and also the dermis, because of the venous insufficiency and it also leads to induration of the skin. Over time the fibrosis can cause the shape of the leg to alter where the proximal area swells, becomes edematous and the distal portion of the leg narrows in because of the lack of subcutaneous fat and lack of fibrosis in that area giving it the classic inverted wine bottle appearance. In the acute stage, there is inflammation often associated with pain, redness, and warmth of the skin present in the area of the ulcer, because of this appearance it can be confused with "cellulitis, inflammatory morphoea or panniculitis".[5] However, in distinguishing the venous ulcers certain signs are sought, such as; patients are eczematous, become sensitive to topical application of medicines, lower extremity oedema, varicose veins, corona phlebectatica, atrophie blanche and lipodermatosclerosis are frequently present in such patients with venous ulcers.[4,5]

Treatment

There are various ways of treating venous ulcers and conservative management, mechanical management and medications are used to treat wounds along with surgical intervention. The initial goal of the treatment is to heal the ulcer followed by reducing the swelling and preventing the ulcer from appearing again. To encourage wound healing, debridement of the wound is the first step, removing the necrotic tissue is done by sharp, enzymatic, larval or autolytic removal.[4] Through these processes at regular timings the venous leg ulcers can result in efficient management needed to heal.[2] Studies have also shown that debridement can further reduce wound healing size, in comparison to wounds which were not treated with debridement had larger wound sizes. In enzymatic debridement, collagenase can remove unwanted tissue, larval therapy uses maggots, and it also allows for disinfection and causes healing to occur, prevents the formation of biofilm because of the bacteria and eradication, however the downside to larval therapy is that it can result in pain. Autolytic debridement is initiated by placing stocking or dressings which retain the moisture and can be used as an adjunct along with other types of debridement.[4] In conclusion debridement is through three simple steps, cleaning the base of the wound, removing any biofilms made by the bacteria which contaminated the wound and reduce the total effects of the burden of the venous leg ulcer.[2]

Compression therapy

Compression therapy is the mainstay for the treatment of venous leg ulcers and chronic venous ulcers.[2] Compression therapy is effective in terms of reducing the rate of healing to the time of complete healing in that the rate is reduced as well and the reduction of the appearance of the wound on the surface is reduced within a faster rate, which indicates compression can initiate faster healing of the wound.[9] Compression therapy also prevents recurrence of the venous ulcer and hence can be used not only for initial treatment but also for continuation of treatment.[4] Studies done on compression therapy showed that multicomponent compression therapy is more effective than single component compression therapy, medical compression bandages are more efficient in healing of the wound in comparison to short stretch or non-medical compression bandages, if two component compression was used alongside elastic components, within one year an increase in healing rates were observed, with three component systems if elastic component was added alongside the healing rates improved within three months and four layer compression systems showed the same efficacy as Unna boots after the use of six months.[9] Compression therapy will narrow the Venous diameter and reflux improving the function of the calf muscle pump.[2] By preventing venous hypertension and encouraging venous return to the heart allows for improvement of the venous pump function and lymphatic drainage.[5] Edema is reduced by the increase in local hydrostatic pressure and reducing venous pressure in the superficial veins preventing fluids and macromolecules to be released into circulation, aiding to arterial flow and oxygenation which allows for healing and fibrinolysis to occur.[2,5]

Leg Elevation

Elevating the leg allows for venous drainage by increasing the deep venous flow as it increases return of blood to the heart, and reduces venous pressure, swelling at the ankle and encourages

cutaneous microcirculation.[4,13] In leg elevation the leg is raised above the level of the heart for 30 minutes and done for three or four times daily. However, following a strict routine for elevating the leg can be cumbersome for all patients to follow but can result in faster healing time.[8] A study done also showed that elevating the leg for one hour a day using venous compression for 6 days a week using compression socks can reduce ulcer recurrence.[4]

Dressings

Dressings are applied beneath compression bandages to cover the wound, the dressing prevents the compression bandages to stick to the base of the ulcer, facilitating drainage and allowing for a moist environment while preventing infections by carrying bacteriostatic properties.[2] Dressings which are available include foams, hydrogels, hydrocolloids, foams, hydrogels, pastes, and simple non adherent dressings.[8] On choosing a dressing consideration should be given to the location of the wound, size of the wound, depth and moisture balance needed, if there is an infection, the patient's allergies, comfort of the dressing, changes needed, cost required and availability of the dressing.[4] If patients present with concomitant stasis eczema and puritis, the use of topical corticosteroids is required, and care should be taken not to prolong the course of corticosteroids as it can result in skin atrophy, acne, folliculitis, and if absorbed into the systemic circulation can results in adrenal insufficiency.[2] Topical antiseptics were used which aid in the healing of ulcers, amongst cadexomer iodine, povidone iodine, peroxide-based preparation, and honey-based preparations as well as silver used to treat venous ulcers, studies showed that use of cadexomer iodine improved the healing of venous ulcers however evidence for the other antiseptics are lacking in studies.

Medications

Pentoxifylline inhibits platelet aggregation and therefore acts as a hemorrhagic agent, it lessens blood viscosity and improves microcirculation and oxygenation.[2,8] Studies have shown that pentoxifylline when used 400 mg three times daily, is effective when used with compression therapy and improves healing of the venous ulcer.[8,4] Pentoxifylline can also be used alone for patients who are intolerant to compression therapy, as studies showed when used alone it also improves healing of the venous ulcer.[4] However due to the side effects of pentoxifylline it often is not preferred by patients as it causes, gastrointestinal discomfort, reduce appetite, diarrhea, nausea and vomiting, prolongs bleeding time and also can cause headaches.[2,4]

Aspirin, when used 300 mg daily and in adjunct with compression therapy has found to increase the healing time of the ulcer and reduce ulcer size as well as recurrent rate than if compression therapy was used alone.[2,8] If there are no contraindications to the use of aspirin then it is used in adjunct with compression therapy, it is not routinely used to treat venous leg ulcers however it is used in patients who have peripheral arterial disease as well as venous leg ulcers.[2]

Antibiotics are used if there are bacterial infections associated with the venous leg ulcer, a Cochrane review of 22 randomized control trials of systemic and topical antibiotics used for treating venous leg ulcers showed that there is no certainty that routine use of antibiotics improves healing rates of the venous leg ulcer.[8] However, if venous leg ulcers do present with infection where bacteria can infect the tissues causing signs such as release of exudate, pain, swelling, delay in healing, erythema, leukocytosis, fever and chills, then venous leg ulcer infection can be treated with antibiotics.[4] Oral antibiotics are the preferred used of antibiotics and treatment should not prolong more than 2 weeks, unless the infection is still present.[4]

Surgical therapies are used to reduce venous reflux, reduce the healing time and prevent the recurrence of the ulcer.[8] A study done where 500 patients from three hospitals were to undergo treatment of venous leg ulcers using compression therapy with superficial venous surgery.[2] The studied showed a marked reduction in recurrence of the venous leg ulcer found in patients who had undergone surgical intervention with the compression therapy to treat the venous leg ulcer as compared to patients who had just undergone monotherapy using compression.[2] Endovenous thermal ablation is another recommended form of therapy for saphenous vein incompetence as it shows reduced convalescence, reduced pain, decrease morbidity and endovenous thermal ablation is advised rather than doing open surgery.[2]. The first line of treatment of venous leg ulcers are endothermal ablation of saphenous veins and perforators rather than the use of open surgeries.[2] However, complications which can arise from endovenous ablation is pain and deep vein thrombosis.[4] There is strong evidence that superficial venous surgeries depress the recurrence rate of venous leg ulcers by 12 months compared to if compression therapy was used alone and the healing rate of ulcers were 88% effective with 13% recurrence rate over 10 months as shown by studies.[8]

To conclude a more recent study conducted also showed that early intervention does prove to have lower recurrence rate, shorter healing time, the timespan between the treatment of ulcer and recurrence of the ulcer is longer if treatment begins during early intervention and early endovenous ablation with compression therapy leads to a shorter healing time of the venous leg ulcers than if compression therapy was used alone, which demonstrated effectiveness of the endovenous ablation of superficial venous reflux.[14]

Conclusion

Venous leg ulcers have existed since the 1200s, there is importance and emphasis placed on understanding the background of venous leg ulcers as this would lead to a better understanding and implementation on innovation of appropriate management and treatment of venous leg ulcers. It can be noted that the management and treatment of venous leg ulcers is dependent on the presentation of the venous leg ulcers. With combined management and treatment along with early intervention of the venous leg ulcers do a better outcome in treating venous leg ulcers is noted.

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None

Conflicts of interest

I declare there is no conflict of interest.

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