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Conservative Management of Chronic Venous Insufficiency

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Abstract

Chronic venous insufficiency (CVI) is a common medical condition with major socioeconomic impact. Prevalence in adult population is estimated to be 5–30%. Its pathology is based on venous hypertension on the lower extremities. This results in edema, subcutaneous fibrosis, pigmentation, chronic pain and ulceration. The severity of chronic venous disease is closely related to the magnitude of venous hypertension. Treatment options for CVI include medical, interventional, surgical and physical treatments. The initial management of CVI involves conservative approaches to reduce symptoms and prevent development of secondary complications and progression of disease. It includes the elevation of the limb together with the supportive methods, pharmacotherapy and structured exercise program, as well as complex decongestive physiotherapy (CDP), intermittent pneumatic compression (IPC), compression stocking (CS), patient education and kinesio tape. CVI is a chronic disease that needs a lifelong care. In this review, we present a discussion about pathophysiology and risk factors for CVI development and conservative treatment options.

Keywords: chronic venous insufficiency, conservative treatment, compression, exercise

1. Introduction

Chronic venous insufficiency (CVI) is described as the inadequacy of the venous system to return blood to the heart [1]. It is common condition which affects many people around the world [2] and denotes some symptoms ranging from minor reticular veins to serious skin changes such as lipodermatosclerosis or venous leg ulcers [1, 3].

CVI is commonly seen in lower limbs. The prevalence of CVI varies in the populations, but it is reported that the prevalence of CVI ranges between 25 and 40% and 10 and 20% in women and

men, respectively [4–6]. Because of its clinical characteristics, it restricts individual's daily activities and has great impact on physical and psychosocial status, as well as socioeconomic status [7].

There are many treatment options such as medical, pharmacological and conservative methods used in CVI. Among these, because of being invasive and causing less adverse effects than other methods, conservative approaches are commonly used to treat CVI [8]. On account of this, we aimed to give information about the conservative approaches used in the treatment of CVI in this chapter. However, due to the different structure of the venous system, to understand the effectiveness of the treatment and how it works, it is necessary to comprehend well the anatomy and pathophysiology of the venous system.

2. Anatomy of the venous system

The venous blood flow in the lower extremity is located at three positions, which are superficial, deep and perforating veins [9].

More than 80% of the blood flow in the lower extremity occurs through the deep veins. The deep veins accompany the arteries and run deep to the muscles and fascia [10, 11]. It includes the posterior tibial vein, anterior tibial vein, femoral vein, profunda femoris vein and common femoral vein in the leg. It also includes the popliteal vein and femoral vein in the thigh, and the external iliac vein, internal iliac vein, common iliac vein and inferior vena cava in the pelvis [12].

The major axial veins are accompanied by matching the venae comitantes in the calf. These veins that accompany the posterior tibial and peroneal veins are particularly large (up to 10 mm in diameter in male) and participate in the muscle pump mechanism for the return of blood from the legs to the heart. In addition, the medial and lateral heads of the gastrocnemius and soleus muscles include vein pairs in these muscles (the calf) that accompany the major arteries. These usually have a large diameter and participate in the calf muscle pump. The calf veins drain into the popliteal vein. Once the popliteal vein has entered the thigh, it is known as the femoral vein. The deep femoral veins which open into the femoral veins derived from the common femoral vein in the inguinal triangle drain the thigh muscles. This drains into the great saphenous vein and is the major point of the venous return in the lower extremity [12].

The superficial compartment lies between the dermis and the deep fascia [12]. Duplex scanning has resulted in recognition of a saphenous subcompartment and saphenous fascia. The vena saphena magna and vena saphena parva are intermediate superficial veins. The VSM is the longest vein in the human body [10, 11].

These veins connect the superficial venous system to the deep venous system. There are approximately 150 perforating veins in each lower extremity and a few of them have clinical significance. The medial calf perforators are very important at this point [12].

The perforating veins are divided into direct and indirect perforating veins. While direct perforating veins provide a connection between the superficial and deep axial veins, indirect perforating veins provide blood drainage from the superficial venous system and join other veins in the muscles [13].

Bicuspid valves in superficial veins provide one-way venous blood flow towards the heart. The frequency of valves is particularly higher below the knee. Valves are large (median 7–10, range 4–13) and placed closer in the small saphenous vein. The frequency of valves increases from proximal to distal in the deep veins. Deep leg veins, peroneal veins and posterior and anterior tibial veins contain plenty of valves which are arranged at approximately 2 cm intervals. However, the common femoral vein usually has one valve. In addition, it must be emphasized that there is only one valve (and sometimes, there is no valve in 37% of the reported cases) in external iliac and common femoral veins, which are proximal to the saphenofemoral junction. The common iliac and vena cava veins have no valves [14].

3. Physiology and hemodynamics of the venous circulation

The primary function of the venous circulation is to return blood to the heart. Effective venous return requires the interaction of a central pump, a pressure gradient, a peripheral venous pump and competent venous valves [14].

Pressures on the vessels are determined by different mechanism. While pressure in arterial vessels is generated mainly by muscular contractions of the heart, the gravity is the main factor that determines the pressure in the venous system. When the body is in a horizontal resting position, pressures in the veins of the lower limb, in the chest, abdomen and arm are similar. On the other hand, when the body goes to the upright position, significant changes occur in the values of these pressures, especially in the venous system. In presence of the upright position, there is an accumulation of approximately 500 ml of blood in the lower extremities. Venous valves play a very important role in the return of blood to the heart from the lower extremities, especially in this position. Direct observation of human venous valves has been made possible by specialized ultrasound techniques [14].

One of the important tasks of the venous valves is to be released during exercise. Supporting the antegrade flow of the venous from the superficial veins to the deep during exercise is the essential part of this important task. Muscle activity through exercise creates volume and pressure changes in the venous system, especially in the calf. While there is no apparent flow in the resting position; with the beginning of the heel strike, the venous plexus under the heel and the plantar surface of the foot (Bejar's plexus) begin to empty from the foot and ankle towards the proximal end of the calf. Then, through calf contraction the blood within the deep veins on the calf is transported into the deep veins of the thigh [14].

In the upright position, the arterial blood pressure is approximately 100 mmHg and the venous pressure is 8 mmHg. The arteriovenous pressure gradient is 92 mmHg. However, this situation changes in the upright position. After 15 min of immobilization, circulating blood volume decreases up to 20% and the velocity of the blood flow decreases. In addition, effective ultrafiltration pressure exceeds effective resorption pressure due to passive hyperemia occurring in blood capillaries. As a result, the flow moves from the blood vessel system to the interstitium in the lower extremities. This situation changes during walking. The venous blood pressure falls up to 25 mmHg in the leg due to the venous leg pump which consists of the calf muscles, joints, skin, fascia and venous valves. The contraction of the calf muscles moves the blood from

the subfascial veins into the heart. The venous blood pressure of 25 mmHg is considerably lower than 100 mmHg but higher than 8 mmHg. This is called as physiological ambulatory venous hypertension. However, it can be tolerated by veins under normal conditions [15].

4. Pathophysiology

Clinical manifestations of CVI occur primarily due to ambulatory venous hypertension in the upright position or during ambulation. These result in clinical sign of edema, subcutaneous fibrosis, pigmentation, chronic pain and ulceration. The severity of chronic venous disease is closely related to the magnitude of venous hypertension. Patients with ambulatory venous pressures less than 40 mmHg have had a lower incidence of venous ulceration compared to patients with ambulatory venous hypertension greater than 80 mmHg [16].

The indications of ambulatory venous pressure are complex. Determinants of ambulatory venous pressure include venous reflux and obstruction as well as calf muscle pump function [17]. Studies have shown that calf muscle pump dysfunction occurs early in the development of CVI and it does not worsen with skin changes and ulceration [18].

In the normal case, the lower extremity muscle pumps and valves limit the accumulation of blood in the lower extremity veins. Failure of the lower extremity muscle pump is associated with peripheral venous insufficiency due to outflow obstruction, musculofascial weakness, loss of joint motion or valvular impairment. However, an effective peripheral pump becomes a compensator for some degrees of reflux and obstruction and prevents symptoms of CVI [13].

When the system operates normally, the ambulatory venous pressure in the superficial system is maintained between 20 and 30 mmHg. The superficial venous pressure may increase up to 60–90 mmHg in cases of venous occlusion, venous valve insufficiency or inappropriate muscle contraction. As mentioned above, this level causes venous hypertension. Thus, it shows that anatomic, physiologic and histological changes associated with chronic venous insufficiency begin. Chronic venous hypertension leads to some histological changes in the capillary bed, which is known as venous hypertensive microangiopathy. These changes include the folding and expansion of the capillary bed and increased endothelial surface [19].

Valvular failure is associated with a rapid recovery time after muscle contraction. If deep venous valves are insufficient, blood simply oscillates within the deep veins and there is no reduction in pressure. Chronic venous hypertension or persistent pressure results in pathological effects in the deep subcutaneous tissues such as edema, pigmentation, fibrosis and ulceration [13].

5. Risk factors for CVI

Risk factors for development of CVI include advanced age, female sex, heredity and trauma to the extremities. Lower limb injuries are a risk factor in women. Age is linked at all levels of the disease in both genders. Older age, Hispanic white ethnicity and family history are risk factors for

CVI. For visible disease, CVI is about twice as common in women than in men. It was reported in the Edinburgh study that CVI was observed two-fold more in men than women [20].

Pregnancy is a critical period for the lower extremity venous system [21], and more than 30% of varicose veins develop only in this period [22, 23]. The reason is considered to be increase in the venous pressure due to the increased blood volume. Moreover, the compression of the iliac veins is an important factor in the later stages of pregnancy. Venous function is undoubtedly influenced by hormonal changes. In particular, progesterone which is released by the corpus luteum stabilizes the uterus by causing relaxation of smooth muscle fibers. This directly affects venous function. Although progesterone affects varicosities in pregnancy at first sight, estrogen also has profound effects [24].

Previous pregnancy, less oral contraceptive use, obesity and mobility at work in women, and height and straining at stool in men may be implicated in the development of reflux [25]. HRT duration or parity is positively associated with all levels of functional disease seen in women. Coughlin et al. [26] reported that multiparity was related with varicose veins in pregnant women.

6. Symptoms for CVI

Symptoms and signs of CVI include hyperpigmentation, stasis dermatitis, pain, cramping, chronic edema and venous ulcers. Approximately 3–11% of the adult population has skin changes and edema due to CVI [27]. Edema was about 50% more common in men than women [28]. In the Edinburgh study it was reported that there were more edema in women; however, the CVI was much more common in men [29].

Edema begins in the perimalleolar region and ascends up the leg. Sense of discomfort in legs is often referred to as weight or pain after standing for a long time and it is relieved by leg elevation. Cutaneous changes include skin hyperpigmentation with hemosiderin deposition and eczematous dermatitis. This fibrotic process produces lipodermatosclerosis and there are risks of cellulite, leg ulcers and delayed wound healing. In addition, chronic venous insufficiency contributes to the development of lymphedema [6].

7. Prevalence

The prevalence and gender distribution are very variable in venous disorders. Although recent studies have not reported prevalence differences related to age in men and women, most studies demonstrate a higher prevalence in women than men [27].

The prevalence of CVI is lower in African and Asian or Australian aboriginal populations. Changes in lifestyle and eating habits in the industrialized countries (especially a low-fiber diet causing constipation) and abdominal pressure are considered as one of the main causes of this phenomenon [30].

8. Classification

The Chronic Venous Disorders (CVD) Guideline was released for the Clinical, Etiologic, Anatomic and Pathophysiologic (CEAP) classification in 1994 by American Venous Forum, which is an international ad-hoc committee, and was endorsed by the Society for Vascular Surgery. It was incorporated into “Reporting Standards in Venous Disease” in 1995. Nowadays, clinical papers released on CVD use the whole or some parts of CEAP classification to determine features of disease [31].

The purpose of the CEAP classification is to provide an objective classification system that is valid and reliable throughout the world. This classification identifies the clinical symptoms (C), etiologic factors (E), anatomical features (A) and underlying pathophysiological event (P) [32, 33]. Detailed information is given in **Table 1**.

The clinical classification has seven categories (between 0 and 6) and is further categorized by the presence or absence of symptoms. The etiologic classification is based on congenital, primary and secondary causes of venous dysfunction [34]. The anatomic classification describes the superficial, deep and perforating venous systems with multiple venous segments that may be involved. The pathophysiologic classification describes the underlying mechanism resulting in CVI, including reflux, venous obstruction or both [6].

C0: This refers to early the disease stages and there is usually not any visible or palpable sign of venous disease. Despite the fact that sometimes no clinical evidence is found by the physicians during the examination; clinically CVI is manifested by the presence of some symptoms such as aching legs, heaviness, sensation of burning and nocturnal cramps. Depending on the

Clinical	Etiologic	Anatomic	Pathophysiologic
C ₀ : There is no sign of venous disease	Ec: Congenital	As: Superficial veins	Pr: Reflux
C ₁ : Telangiectases or reticular veins	Ep: Primary	Ap: Perforator veins	Po: Obstruction
C ₂ : Varicose veins	Es: Secondary (post-thrombotic, post-traumatic)	Ad: Deep veins	Pr,o: Reflux and obstruction
C ₃ : Edema	En: There is no cause determined	An: There is no venous location identified	Pn: There is no venous pathophysiology determined
C ₄ : Pigmentation or eczema on the skin			
C _{4b} : Lipodermatosclerosis or atrophie blanche			
C ₅ : Healed venous ulcer			
C ₆ : Active venous ulcer			

Table 1. CEAP classification system.

severity of the symptoms, there may not be a significant change in the patient's daily life or it may cause significant limitations on the patients.

C1: Telangiectasia (dilated intradermal venules up to a size of about 1 mm) and reticular varicose veins (dilated, nonpalpable and subdermal veins up to a size of about 4 mm). These signs which are associated with increased venous pressure and chronic venous insufficiency occur due to the capillary disorder.

C2: There are several dilated and simultaneously elongated varicose veins. Varicose veins according to etiologic origins are classified as primary (idiopathic), secondary (caused by the post-thrombotic syndrome) and other secondary varicose veins of unclear etiology.

C3: Swelling is one of the most frequent signs of CVI. It is characterized by the accumulation of proteins and water in subcutaneous tissues. While in lying position, the swellings tend to disappear; in long-standing position, swelling increases and causes significant limitations in the daily life of the patients.

C4: As CVI progresses, changes on the skin begin to be observed. On this stage pigmentation, eczema and lipodermatosclerosis are common findings.

C5: Venous ulcers, which are defined as a loss of skin tissue, may develop on the skin in the following periods. Skin changes with healed ulcer are seen in this stage. Only healed varicose ulcers are included within the class 5.

C6: This class is defined by the presence of an active varicose ulcer [35].

Qualification	Absent: 0	Mild: 1	Moderate: 2	Severe: 3
Sense of discomfort, Pain, aching, fatigue heaviness	No	Sometimes, it does not reflect in daily activities	Daily, it does not affect daily activities	Daily, it restricts most of activity
Varicose veins	No	Few and scattered	Confined to calf or thigh	Involve calf and thigh
Venous edema	No	Restricted foot and ankle	Spreads above ankle but don't pass the knee	Extends to knee or above the knee
Pigmentation of the skin	No or focal	Restricted in perimalleolar area	Diffuse over lower third of calf	Diffuse more than lower third of calf
Inflammation	No	Restricted in perimalleolar area	Diffuse over lower third of calf	Diffuse more than lower third of calf
Skin induration	No	Restricted in perimalleolar area	Diffuse over lower third of calf	Diffuse more than lower third of calf
Number of active ulcers	0	1	2	>2
Duration of ulcer	Absent	<3 months	Between 3 months and 1 year	Unhealing >1 year
Size of ulcer	Absent	Diameter <2 cm	Diameter 2–6 cm	Diameter >6 cm
Compression therapy	Not used	Intermittent	Most days	Every day, mostly

Table 2. Venous clinical severity scoring.

To complement the CEAP classification and further define the severity of CVI, the venous clinical severity score (VCSS) was developed (**Table 2**) [36, 37]. The revised VCSS provides clarification of the terms and better definition of the descriptors and has further clinical applicability [38].

The VCSS consists of 10 attributes with four grades (absent, mild, moderate and severe). It has been shown to be useful in evaluation of the response to treatment in CVI. To evaluate severity of disease and treatment outcomes in CVI, it is recommended to make use of validated disease-specific quality-of-life questionnaires in clinical evaluations [39].

9. Treatment of CVI

The initial management of CVI involves conservative approaches to reduce symptoms and prevent development of secondary complications and progression of disease. If conservative approaches fail or provide an unsatisfactory response, further treatment methods should be considered on the basis of anatomic and pathophysiologic features [40]. The treatment options of CVI ranges from simple compression stockings to very complicated venous reconstructions, and the most important step includes patient education to obtain better outcomes after treatment [5].

9.1. Conservative treatment

Conservative approaches aim mainly to restore the altered physiological functions of the venous system. It includes the elevation of the limb together with the supportive methods, pharmacotherapy and structured exercise program, as well as complex decongestive physiotherapy (CDP), intermittent pneumatic compression (IPC), compression stocking, patient training [41–44] and kinesio tape [45].

In order to overcome the effects of the gravity, the elevation of the limb in venous diseases has been applied for centuries. The venous pressure in the tissues around the ankle falls to approximately zero when the feet are at/above the heart level (i.e. toes-above-nose position). When the patient is in this position, all the signs and symptoms, which are directly associated with venous hypertension (i.e. ulcers, eczema, swelling, etc.), resolve without the need for any other treatment, particularly, in lower severity of disease [44].

Training the patient about the changes in lifestyle is important side of the total care. The control of weight, care of the limbs and optimum exercises to keep the calf muscles and ankle joint supple are among the strategies [44].

Compression is advised for the purpose of decreasing ambulatory venous hypertension in patients who have CVI. Changes in lifestyle including weight loss, exercise and elevation of the legs during the day whenever possible are also advised for such patients [46].

9.1.1. Complex decongestive physiotherapy

Since CVI includes combined formats of lymphedema, compression therapy (CT) is preferred as treatment method especially for advanced stages of CVI. CDP is a treatment program

consisting of two phases. First phase is the phase in which the edema is decreased and lasts for 4 or more weeks and consists of four components as follows: (1) manual lymph drainage (MLD), (2) skin care, (3) compression bandage and (4) therapeutical exercises. The second phase is the one in which the decreased volume is protected with skin care, compression stocking and exercises. The precautions and patient training are emphasized for the extremity care for this program. The participation and harmony of the patient within the program are important for successful results [47].

9.1.1.1. *Manual lymphatic drainage*

Manual lymphatic drainage (MLD) is defined as a special manual technique stimulating the superficial lymphatic vessels in order to remove the excessive interstitial fluid [48] and to increase the lymph flow [49]. It has been used for more than 50 years as a type of conservative treatment method in CVI for the purpose of removing extremity edema [50]. In the origin of the technique, there is a soft massage for the purpose of stimulating lymphatic vessels and propel fluid through the channels. Since 20% (or more) patients, who have CVI, also have a lymphedema component, manual lymphatic drainage might have a significant role in a compression therapy program intended for CVI [51]. There have been four MLD techniques well known in the literature, up to now. These include the Foldi, Vodder, Casley-Smith and Leduc techniques [46].

In the study conducted by Foldi et al., it was reported that the MLD was inadequate without using compression therapy [52]. Similarly, Ochalek suggested that the use of compression therapy with MLD was necessary to sustain the effects of MLD [53]. In another study conducted by Bakar et al., 62 patients who had chronic venous disease were included and all the patients treated with CDP. According to the results of the study, CDP application decreased the volume and the percentage-volume of CVI-related edema at a significant level and decreased the intensity of the pain, which was caused by this condition, in the elderly [54].

While it is reported in a meta-analysis study conducted by Karki et al. that MLD was not significantly effective in sustaining decreased edema when used alone [55], Szewczyk et al. reported that MLD was effective in decreasing the edema in the lower limbs of the patients with CVD [56].

9.1.1.2. *Skin care*

Since CVI is a progressive disease, it disrupts the skin integrity. In healthy skin, the hydro-lipid mixture consisting of water and lipid in the epidermis cover and protect the skin from external effects. The lipid component in it decreases the vaporization of the water and ensures the flexibility of the skin. Acidic products (pH 4.5–5.7) and microbial CVI may damage the skin integrity at further stages especially in internal malleoli area. There will be cracks and, in the end, inclination to infection occurs. In order to decrease the risk of infection and the disruption on the skin integrity, it is important to keep the skin moist in the affected area. For this reason, natural products that have balanced contents and that are similar to dermal lipids must be used. Normal skin moisture factors and the lipids that form barriers are important to keep the skin in elastic and slippery form. The applications that are healing and protective are important

for both phases of the CDT. In Phase 1, the focus is on healing and care of the damaged skin. In Phase 2, the sustaining of the skin care is important [47].

9.1.1.3. Compression

The limb compression descriptions are to be found in *Corpus Hippocraticum* (450–350 BC) and it is known to be a major milestone in venous insufficiency treatment [51]. Compression treatment is an important part of the two phases of the CDP. During Phase 1, it is necessary to use the compression bandages for 23 hours a day. The purpose of the bandages is to have a certain form of the edema, and decrease the volume in the extremity. During Phase 2, compression is ensured with the compression stockings produced considering the size of the person [47].

The constriction of the leg veins is an essential mechanism of Compression Therapy (CT). In the supine position, a reduction in vein diameter accelerates blood flow velocity and also helps to prevent deep vein thrombosis. In the upright position, external compression is supposed to counteract the hydrostatic pressure. Due to the effect of gravity, external high pressures are required to maintain the same effect in standing position. It can be achieved with high pressure bandages to reduce ambulatory venous hypertension in patients with CVI [57].

The external pressure of approximately 30–40 mmHg is required to constrict the leg veins in the upright position. Complete occlusion of the leg veins occurs at a pressure of 20–25 mmHg in the supine position, at a pressure of 50–60 mmHg in the sitting position and at a pressure of approximately 70 mmHg in the standing position [57].

The treatment aims to correct the long-term complications of CVI at the possible highest level. CT increases pressure on the skin and on the underlying structures to react with the gravity force when it is applied in an external manner to the leg, which may help to relieve the symptoms in the lower limbs by affecting the venous and lymphatic systems to improve the removal of the fluid (i.e. the blood and the lymph) from the relevant limb [51].

The aims of CT may be summarized as follows:

1. Decrease the swollen limb to its minimum size as fast as possible.
2. Maintain the limb at its possible smallest size by using the simplest methods that are possible.
3. Allow the patient to participate in the care of the limb in an active manner by avoiding the factors that aggravate the edema.
4. Train the patient on how to modify his/her own therapy method in case of a problem with edema [51].

In order to treat or prevent the adverse effects, the topical CT provides help. Limb compression changes the tissue pressure gradient, which, in return, reduces the formation of the edema and increases the resorption of the edema; reduces the vein caliber and increases venous flow velocity; reduces orthostatic reflux, residual volume and ambulatory venous pressure (partly by re-recruiting venous valves and by reducing reflux in the perforating vessels) and improves the muscle pump effectiveness [51].

There are many methods in the field of providing CT. The four-layer elastic bandaging (4LB) and the short stretch bandaging (SSB) are the most widely used techniques. The introduction of the graded compression stocking in varying degrees of pressures at the ankle area is a major advance in CT. The purpose of these systems is to provide graduated compression to the lower limb for the purpose of improving venous return and to reduce the edema [51]. Different ambulatory compression techniques and devices cover the compression stockings (CS), paste gauze boots (i.e. the Unna boot), multilayer elastic wraps, dressings, elastic and non-elastic bandages and non-elastic garments. Pneumatic compression devices are also used in patients with refractory edema and venous ulcers [58].

9.1.2. Compression stocking

Compression stocking (CS) was first developed in the 1950s by Conrad JOBST. CS is available in various forms, force and size. It provides a pressure decreasing towards proximal from the distal and it has become one of the key treatments and standard procedure in the management of venous lymphatic disorders. Compression pressure ranges and the definition of these classes vary among countries. For this reason, using the pressure ranges as mmHg will be beneficial for universal understanding. CS is classified into four classes according to their pressure values (**Table 3**). The pressures of the CS used in CVI depend on the clinical severity; for CEAP second and third classes, 20–30 mmHg and for CEAP fourth to sixth classes, 40–50 mmHg are used. The most-frequently used size is the one that stretched up to the knee, because the patient participation is more and the symptom healing is adequate. The stockings that reach up to the thigh and groin may be compulsory for the patients that have edema on the knee; however, the use of such stockings is difficult. It must be changed every 6 months [5]. CS must be worn in the stages when the veins are at the emptiest stage. The CS that is worn in the early morning when the patient gets up is the most effective one. The patient must walk in the stockings during the day [40].

9.1.3. Intermittence pneumatic compression (IPC)

Wide consensus exists in the literature that compression is a necessary part of all treatments for CVI and venous ulcers. Compression is usually provided by stockings. Most of the elderly patients have reduced strength and skill and therefore it makes these stockings difficult to wear. Compression can also be provided with the Unna's boot or different types of bandages [27].

Extremity compression can be achieved with pneumatic compression tools. Pneumatic compression devices consist of an inflatable garment for the arm or leg and an electrical pneumatic pump that fills the garment with compressed air. The boot is intermittently inflated and deflated and creates a pumping effect [27]. The inflation and deflation cycles mimic the muscle pump. This is one of the key mechanisms to provide proper venous and lymphatic flow [59].

Compression class	1	2	3	4
Compression degree	Mild	Moderate	Strong	Very strong
Pressure (mmHg)	18–21	23–32	34–46	49<

Table 3. Compression classes.

First generation pneumatic compression devices consisted of an inflatable single compartment pressure chamber that applied a non-segmented uniform and sustained level of compression to the entire extremity. Later, multi-segment compression devices were developed in order to increase the effectiveness of the devices. Thanks to these devices, technically, the pressure gradient is created between distal and proximal parts of the limb. The pressure of distal chamber is higher than in the proximal chamber and this enables a sequential mechanism of distal to proximal application of pressure. Pneumatic compression devices have further evolved in recent years and allow digital programming to imitate MLD techniques and promote fluid clearance at the proximal trunk and extremities [60] (**Table 4**).

Pneumatic compression device	Distinctive features
Single chamber non-programmable pumps	<ul style="list-style-type: none"> • The single sleeve inflates to apply pressure on the leg • No manual control on pressure distribution • No pressure gradient • Nowadays it is not an optimal method for lymphedema treatment
Multi-chamber (segmented) non-programmable pumps	<ul style="list-style-type: none"> • Approximately three or four chambers swell completely from the distal to the proximal segment and then descend • Perhaps a limited pressure programming option can be found but not independently adjustable • Structurally, each chamber is designed to reach the same pressure gradient, with support from the extremity shape • Pumps can use one or both of the same legs or arms
Multi-chamber programmable pumps	<ul style="list-style-type: none"> • The pressure gradient is set to be higher in the distal segment and lower in the proximal segment • It produces three different pressure zones, and some pumps allow the divider to set the pressure • Manual programmable and pressure-adjustable for the desired zone • Adjustable from 4 to 36 chambers
Advanced pneumatic compression systems	<ul style="list-style-type: none"> • Digitally programmable • With proper application to adjoining trunk segments, it can provide a uniform distribution of edema by relaxing the proximal and distal limb • Garbage and proximal chambers can open lymphatic pathways • Only 1 and 2.5 compartments can actively simulate manual lymphatic drainage by providing a pressure increase and progression from distal to proximal

Table 4. Pneumatic compression devices.

The most basic definition of the IPC is that it consists in the application of a force on an edema in order to evacuate its components as much as possible towards the physiological ways of drainage (venous—lymphatics—interstitium) [61].

IPC has physiological effects such as increase in capillary perfusion, increase in tissue oxygenation, increase in the fibrinolytic potential of endothelial cells, increased blood-flow in the deep veins, decrease in stasis, decrease in venous hypertension and decrease in interstitial edema [62–64].

There are some contraindications to pneumatic compression [65] (**Figure 1**). Despite the fact that there is no serious complication of pneumatic compression, It has been reported that there are some complications in the literature [27, 66, 67] (**Figure 2**).

In the study of Caprini, it was reported that pneumatic compression applied 4 hours per day provided significant improvement in symptoms [68]. Another study examined the efficacy of pneumatic compression in those with CVI and it was concluded that a single 30-min session per day of pneumatic compression decreased edema, increased venous blood-flow and improved symptoms [69].

Figure 1. Contraindications of IPC.

Figure 2. Complications of IPC.

In the literature, it is common in CVI that high pressure is more effective compared to low pressure, multi-layer bandages are more effective compared to single-layer bandages and compression application is more effective than no compression application [63].

9.1.4. Exercise

The calf muscle pump is the basic mechanism in the return of blood from the lower extremity to the heart and also it is supported by the foot pump, the thigh pump and the respiratory pump. The ankle joint is the main component of the calf muscle pump. Therefore, dorsiflexion and plantar flexion of the ankle is the basis for effective function of the calf muscle pump [70, 71]. It is known that limited ankle mobility increases the severity of edema formation and venous reflux in patients with CVI. CVI causes ankle immobility due to storage of fibrotic tissue. Because of immobility, the calf muscle pump cannot be activated and venous blood does not return to the heart [72]. Presence of any abnormality in pump functions of calf muscle plays a significant role in the development of CVI. In patients with CVI, progressive exercise program have been used to rehabilitate the muscle pump function and improve symptoms [6]. A randomized controlled study designated individuals with advance venous disease (CEAP class C4–C6) to structured calf muscle exercise or routine daily activities. To assess venous hemodynamics duplex ultrasound and APG were used, and muscle strength was assessed with a dynamometer. After 6 months, parameters of pump function of calf muscles normalized in patients receiving customized exercise program for calf muscles. However, there was no change in the

amount of reflux or severity scores. Although it was found that severity scores of reflux was not changing statistically, it appears that calf muscle pump function could be established by structured exercise in CVI that may prove effective as supplemental therapy to medical and surgical treatment in advanced disease [73].

Research over the past 10–12 years points to ankle joint movement as the key biomechanical element in a functioning calf pump. When artificially restricting the movement of the ankle joint in healthy volunteers, it is demonstrated that a significant decrease occurred in the efficiency of the pump to affect a decrease in venous pressure during exercise [74]. Calf muscle strength may also affect the efficiency of venous return [75]. An improvement in calf muscle strength correlates with improved venous return [76] and loss of muscle strength is seen in patients with long-lasting venous ulcer [75]. Taheri et al. stated that there are three types of atrophy in biopsies of the gastrocnemius muscle in patients with vein insufficiency. These were disuse, denervation and ischemia, which play a role in muscle destruction [77]. How much calf muscle volume contributes to venous insufficiency is unclear [75].

Back et al. stated that a normal walking motion is required for activation of the calf muscular pump and this requires 90 degrees of dorsiflexion [78]. It has been found that exercise program twice a week increased the angle of dorsiflexion and plantar flexion in those with CVI [79].

9.1.5. *Kinesio tape*

Kinesiology taping (Kinesio Tape) which was developed by Dr. Kenzo Kase in 1996 is a technique that is used to restore muscle function, to increase vascular and lymphatic circulation, to relieve pain or to correct the impaired joint alignment. Kinesio Tape is an elastic, latex-free, adhesive and waterproof tape. Moreover, it is an application that may remain in the skin for 3–5 days. Skin adhesive tape is thicker and more elastic than conventional tape and also it has the ability to stretch up to 120–140% of its normal length. Although there is no definite evidence for its proprioceptive effect, it is thought to act by means of cutaneous mechanoreceptors [80].

Skin-taping increases the circulation in the region by creating convulsions that cause the dermis to rise up. Kinesio Tape, which is frequently used in edema, hematoma and wound healing, has been used as an alternative to compression therapy in patients with venous insufficiency in recent years. Even if it is frequently used, Kinesio Tape has not been proven to have a positive effect on venous insufficiency. In some studies, it has been shown that there was an increase in lymphatic circulation and venous return of individuals who underwent fan technique without strain [81].

10. Conclusion

Chronic venous insufficiency (CVI) is a common disorder. As a result of various pathologies that develop at the lower extremity venous system, venous pressure increases, affecting

the whole lower extremity. This could result in valvular insufficiency at superficial or axial deep veins and perforator veins, venous obstruction or a combination of these. These factors worsen with calf muscle pump dysfunction. These mechanisms partially provide grounds for the development of venous hypertension during standing. Patient may not have any symptoms, or they can complain from swelling at legs, discomfort with pain, anxiety, night cramps or pain occurring when standing. There are many treatment options such as medical, pharmacological and conservative methods used in CVI. Among these, conservative approaches because of being invasive with less adverse effects than other methods are commonly used to treat CVI. Conservative treatment of patients with CVI relies on the use of compression therapy, which is considered the “gold standard,” provided by means of elastic stockings, bandages and pneumatic compression devices. Patient education is essential. CVI is a chronic disease that needs a life-long care. So patients with CVI should encourage to maintain a normal body weight, exercise daily (a walking program is especially good) and wear compression garment throughout the day.

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References

- [1] De Aguiar E, Pinto L, Figueiredo M, Savino N. Diretrizes sobre Diagnóstico, Prevenção e Tratamento da Sociedade Brasileira de Angiologia e Cirurgia Vascular (SBACV). Úlcera de Insuficiência Venosa Crônica. *J Vasc Br.* 2005;4(Supl 2):S195–200.
- [2] Kaplan RM, Criqui MH, Denenberg JO, Bergan J, Fronck A. Quality of life in patients with chronic venous disease: San Diego population study. *Journal of Vascular Surgery.* 2003;37(5):1047–53.
- [3] Marston W. Evaluation and treatment of leg ulcers associated with chronic venous insufficiency. *Clinics in Plastic Surgery.* 2007;34(4):717–30.
- [4] Khan A, Chaudhri R, Ashraf M, Mazaffar M, Zawar-ul-Imam S, Tanveer M. Prevalence and presentation of chronic venous disease in Pakistan: a multicentre study. *Phlebology.* 2013;28(2):74–9.
- [5] Eberhardt RT, Raffetto JD. Chronic venous insufficiency. *Circulation.* 2005;111(18):2398–409.
- [6] Eberhardt RT, Raffetto JD. Chronic venous insufficiency. *Circulation.* 2014;130(4):333–46.

- [7] Paul JC, Pieper B, Templin TN. Itch: association with chronic venous disease, pain and quality of life. *Journal of Wound, Ostomy, and Continence Nursing: Official Publication of the Wound, Ostomy and Continence Nurses Society/WOCN*. 2011;38(1):46.
- [8] Castro A, Santos M, ALS CA, Teixeira A. Diagnóstico e tratamento da doença venosa crônica. *Jornal Vascular Brasileiro*. 2005;4(2):185–94.
- [9] Sistem YMAEV. Klinik Anatomisi. İÜ Cerrahpaşa Tıp Fakültesi Sürekli Tıp Eğitimi Etkinlikleri Sempozyum Dizisi. 2007;56:9–17.
- [10] Drake R, Vogl W, Mitchell A. *Grey's Anatomy*. Elsevier/Churchill Livingstone: Philadelphia. 2005.
- [11] Bergan JJ, Bunke N. *The Vein Book*. Oxford University Press: New York. 2014.
- [12] Menon RR. Lower limb venous anatomy. In: Vaidyanathan S, Menon RR, Jacob P, John B, editors. *Chronic Venous Disorders of the Lower Limbs*. Springer: New Delhi, 2015. pp. 9–16.
- [13] Meissner MH, Moneta G, Burnand K, Gloviczki P, Lohr JM, Lurie F, et al. The hemodynamics and diagnosis of venous disease. *Journal of Vascular Surgery*. 2007;46(6):S4–S24.
- [14] Ellis H, Logan BM, Dixon AK. *Handbook of venous disorders*. Annals of The Royal College of Surgeons of England. 2010;92(2):179.
- [15] Földi E, Földi M. Chronic venous insufficiency and venous-lymphostatic insufficiency. *Földi's Textbook of Lymphology*, 2nd ed. Elsevier: Munich, Germany. 2006:434–47.
- [16] Tran NT, Meissner MH. The epidemiology, pathophysiology, and natural history of chronic venous disease. *Seminars in Vascular Surgery*. 2002;15(1):5–12.
- [17] Nicolaides A. Investigation of chronic venous insufficiency a consensus statement. *Circulation*. 2000;102(20):e126–e63.
- [18] Welkie JF, Comerota AJ, Katz ML, Aldridge SC, Kerr RP, White JV. Hemodynamic deterioration in chronic venous disease. *Journal of Vascular Surgery*. 1992;16(5):733–40.
- [19] Pappas P, Durán W, Hobson R. Pathology and cellular physiology of chronic venous insufficiency. *Handbook of Venous Disorders*, 2nd ed. Arnold: New York. 2001:58–67.
- [20] Criqui MH, Denenberg JO, Langer RD, Kaplan RM, Fronek A. Epidemiology of chronic peripheral venous disease. In: Bergan JJ, editor. *The Vein Book*. Elsevier: USA. 2007. pp 27–37.
- [21] Zicot M. Venous diseases and pregnancy. *Revue medicale de Liege*. 1999;54(5):424–8.
- [22] Stvrtinová V, Kolesar J, Wimmer G. Prevalence of varicose veins of the lower limbs in the women working at a department store. *International Angiology*. 1990;10(1):2–5.
- [23] Preziosi P, Galan P, Aissa M, Hercberg S, Boccalon H. Prevalence of venous insufficiency in French adults of the SUVIMAX cohort. *International Angiology*. 1999;18(2):171.

- [24] Townsend Jr CM, Beauchamp RD, Evers BM, Mattox KL. Sabiston Textbook of Surgery. Elsevier Health Sciences: Philadelphia. 2012.
- [25] Gourgou S, Dedieu F, Sancho-Garnier H. Lower limb venous insufficiency and tobacco smoking: a case-control study. *American Journal of Epidemiology*. 2002;155(11):1007–15.
- [26] Coughlin L, Gandy R, Rosser S, De Cossart L. Factors associated with varicose veins in pregnant women. *Phlebology*. 2001;16(4):167–9.
- [27] Berliner E, Ozbilgin B, Zarin DA. A systematic review of pneumatic compression for treatment of chronic venous insufficiency and venous ulcers. *Journal of Vascular Surgery*. 2003;37(3):539–44.
- [28] Criqui MH, Jamosmos M, Fronek A, Denenberg JO, Langer RD, Bergan J, et al. Chronic venous disease in an ethnically diverse population the San Diego population study. *American Journal of Epidemiology*. 2003;158(5):448–56.
- [29] Evans C, Fowkes F, Ruckley C, Lee A. Prevalence of varicose veins and chronic venous insufficiency in men and women in the general population: Edinburgh Vein Study. *Journal of Epidemiology and Community Health*. 1999;53(3):149–53.
- [30] Carpentier P, Priollet P. Epidemiology of chronic venous insufficiency. *Presse Medicale* (Paris, France: 1983). 1994;23(5):197–201.
- [31] Eklöf B, Rutherford RB, Bergan JJ, Carpentier PH, Gloviczki P, Kistner RL, et al. Revision of the CEAP classification for chronic venous disorders: consensus statement. *Journal of Vascular Surgery*. 2004;40(6):1248–52.
- [32] Padberg FT. CEAP classification for chronic venous disease. *Disease-A-Month*. 2005; 51(2):176–82.
- [33] Rabe E, Pannier F. Clinical, aetiological, anatomical and pathological classification (CEAP): gold standard and limits. *Phlebology*. 2012;27(suppl 1):114–8.
- [34] Lacroix P, Aboyans V, Preux P, Houles M, Laskar M. Epidemiology of venous insufficiency in an occupational population. *International Angiology*. 2003;22(2):172.
- [35] Staffa R. Chronic venous insufficiency epidemiology. Prevalence. 2002;60(70):55–7.
- [36] Neglén P, Thrasher TL, Raju S. Venous outflow obstruction: an underestimated contributor to chronic venous disease. *Journal of Vascular Surgery*. 2003;38(5):879–85.
- [37] Araki CT, Back TL, Padberg FT, Thompson PN, Jamil Z, Lee BC, et al. The significance of calf muscle pump function in venous ulceration. *Journal of Vascular Surgery*. 1994;20(6):872–9.
- [38] Vasquez MA, Rabe E, McLafferty RB, Shortell CK, Marston WA, Gillespie D, et al. Revision of the venous clinical severity score: venous outcomes consensus statement: special communication of the American Venous Forum Ad Hoc Outcomes Working Group. *Journal of Vascular Surgery*. 2010;52(5):1387–96.

- [39] Labropoulos N, Leon M, Nicolaidis AN, Giannoukas AD, Volteas N, Chan P. Superficial venous insufficiency: correlation of anatomic extent of reflux with clinical symptoms and signs. *Journal of Vascular Surgery*. 1994;20(6):953–8.
- [40] Periferik TKDCD. Arter ve Ven Hastalıkları Tedavi Kılavuzu. Ankara: Öncü Basımevi. 2008:97–123.
- [41] Weiss RA, Munavalli G, editors. Endovenous ablation of truncal veins. *Seminars in Cutaneous Medicine and Surgery*: WB Saunders: Philadelphia, USA. 2005.
- [42] Mariani F. Compression. Consensus Document Based on Scientific Evidence and Clinical Experiences. Minerva Medica: Torino. 2009.
- [43] Bakar Y. Mastektomi sonrası gelişen lenfödemde fizyoterapinin etkinliği Ankara: Hacettepe University: Ankara. 2005.
- [44] Vaidyanathan S, Menon RR, Jacob P, John B. Chronic venous disorders of the lower limbs. *A Surgical Approach*: Springer; 2014.
- [45] Aguilar-Ferrándiz ME, Castro-Sánchez AM, Matarán-Peñarrocha GA, García-Muro F, Serge T, Moreno-Lorenzo C. Effects of kinesio taping on venous symptoms, bioelectrical activity of the gastrocnemius muscle, range of ankle motion, and quality of life in post-menopausal women with chronic venous insufficiency: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation*. 2013;94(12):2315–28.
- [46] dos Santos Crisóstomo RS, Candeias MS, Ribeiro AMM, Martins CdLB, Armada-da-Silva PA. Manual lymphatic drainage in chronic venous disease: a duplex ultrasound study. *Phlebology*. 2014;29(10):667–676.
- [47] Asmussen P, Strossenreuther R. Compression therapy. *Földi's Textbook of Lymphology* San Biotext: Francisco. 2006:563–627.
- [48] Leduc O, Bourgeois P, Leduc A. Manual lymphatic drainage: scintigraphic demonstration of its efficacy on colloidal protein reabsorption. *Progress in Lymphology*. 1988;11:551–4.
- [49] Tan I-C, Maus EA, Rasmussen JC, Marshall MV, Adams KE, Fife CE, et al. Assessment of lymphatic contractile function after manual lymphatic drainage using near-infrared fluorescence imaging. *Archives of Physical Medicine and Rehabilitation*. 2011;92(5):756–64.
- [50] Steins A, Junger M. Physical therapy in patients with chronic venous insufficiency. *Phlebologie-Stuttgart*. 2000;29(2):48.
- [51] Felty CL, Rooke TW. Compression therapy for chronic venous insufficiency. *Seminars in vascular surgery*: Elsevier. 2005;18(1):36–40.
- [52] Földi E. The treatment of lymphedema. *Cancer*. 1998;83(S12B):2833–4.
- [53] Ochalek K, Grądalski T. The use of manual lymph drainage in vascular diseases. *Acta Angiol*. 2011;17(3):189–98.

- [54] Bakar Y, Öztürk A, Calisal MA, Ertürk K, Daglar B. Complete decongestive physiotherapy for older people with chronic venous insufficiency. *Topics in Geriatric Rehabilitation*. 2010;26(2):164–70.
- [55] Kärki A, Anttila H, Tasmuth T, Rautakorpi U-M. Lymphoedema therapy in breast cancer patients—a systematic review on effectiveness and a survey of current practices and costs in Finland. *Acta Oncologica*. 2009;48(6):850–9.
- [56] Szewczyk T. The importance of massage in edema of lower limbs in patients with chronic venous insufficiency. *Valetudinaria Postępy Medycyny Klinicznej i Wojskowej*. 2002;7:50–3.
- [57] Partsch B, Partsch H. Calf compression pressure required to achieve venous closure from supine to standing positions. *Journal of Vascular Surgery*. 2005;42(4):734–8.
- [58] Molski P, Kruczyński J, Molski A, Molski S. Manual lymphatic drainage improves the quality of life in patients with chronic venous disease: a randomized controlled trial. *Archives of Medical Sciences*. 2013;9(3):452–8.
- [59] Szolnoky G, Dobozy A, Kemény L. Towards an effective management of chronic lymphedema. *Clinics in Dermatology*. 2014;32(5):685–91.
- [60] Feldman J, Stout N, Wanchai A, Stewart B, Cormier J, Armer J. Intermittent pneumatic compression therapy: a systematic review. *Lymphology*. 2012;45(1):13.
- [61] Belgrado J-P, Bourgeois P, Röh N, Moraine J. Intermittent pneumatic compression in the treatment of lymphedema: current state of knowledge. *European Journal of Lymphology Related Problems*. 2007;17(50):4–10.
- [62] Kumar S, Walker MA. The effects of intermittent pneumatic compression on the arterial and venous system of the lower limb: a review. *Journal of Tissue Viability*. 2002;12(2):58–66.
- [63] Partsch H. Indications for compression therapy in venous and lymphatic disease consensus based on experimental data and scientific evidence under the auspices of the IUP. *International Angiology*. 2008;27(3):193.
- [64] Comerota AJ. Intermittent pneumatic compression: physiologic and clinical basis to improve management of venous leg ulcers. *Journal of Vascular Surgery*. 2011;53(4):1121–9.
- [65] Brennan MJ, Miller LT. Overview of treatment options and review of the current role and use of compression garments, intermittent pumps, and exercise in the management of lymphedema. *Cancer*. 1998;83(S12B):2821–7.
- [66] Werbel G, Shybut G. Acute compartment syndrome caused by a malfunctioning pneumatic-compression boot. A case report. *Journal of Bone and Joint Surgery. American volume*. 1986;68(9):1445–6.
- [67] Strup S, Gudziak M, Mulholland S, Gomella L. The effect of intermittent pneumatic compression devices on intraoperative blood loss during radical prostatectomy and radical cystectomy. *The Journal of Urology*. 1993;150(4):1176–8.

- [68] Arcelus JL, Caprini JA, Sehgal LR, Reyna JJ. Home use of impulse compression of the foot and compression stockings in the treatment of chronic venous insufficiency. *Journal of Vascular Surgery*. 2001;34(5):805–11.
- [69] Güçük A, Peker Ö, Bircan Ç, Yiğit M, Şenocak Ö, Gülbahar S et al. The effectiveness of intermittent pneumatic compression in patients with chronic venous insufficiency. *Ege Fiziksel Tıp Rehabilitasyon Dergisi*, 2001; 7(3–4): 117–122.
- [70] van Uden CJ, van der Vleuten CJ, Kooloos JG, Haenen J, Wollersheim H. Gait and calf muscle endurance in patients with chronic venous insufficiency. *Clinical Rehabilitation*. 2005;19(3):339–44.
- [71] Miller BF, Gruben KG, Morgan BJ. Circulatory responses to voluntary and electrically induced muscle contractions in humans. *Physical Therapy*. 2000;80(1):53–60.
- [72] Kunimoto B, Cooling M, Gulliver W, Houghton P, Orsted H, Sibbald RG. Best practices for the prevention and treatment of venous leg ulcers. *Ostomy Wound Management*. 2001;47(2):34–51.
- [73] Padberg FT, Johnston MV, Sisto SA. Structured exercise improves calf muscle pump function in chronic venous insufficiency: a randomized trial. *Journal of Vascular Surgery*. 2004;39(1):79–87.
- [74] Kügler C, Strunk M, Rudofsky G. Venous pressure dynamics of the healthy human leg. *Journal of Vascular Research*. 2001;38(1):20–9.
- [75] Davies J, Bull R, Farrelly I, Wakelin M. Improving the calf pump using home-based exercises for patients with chronic venous disease. *Wounds UK*. 2008;4(3):48–53.
- [76] Kan YM, Delis KT. Hemodynamic effects of supervised calf muscle exercise in patients with venous leg ulceration: a prospective controlled study. *Archives of Surgery*. 2001;136(12):1364–9.
- [77] Taheri SA, Heffner R, Williams J, Lazar L, Elias S. Muscle changes in venous insufficiency. *Archives of Surgery*. 1984;119(8):929–31.
- [78] Back TL, Padberg FT, Araki CT, Thompson PN, Hobson RW. Limited range of motion is a significant factor in venous ulceration. *Journal of Vascular Surgery*. 1995;22(5):519–23.
- [79] Yang D, Vandongen Y, Stacey M. Effect of exercise on calf muscle pump function in patients with chronic venous disease. *British Journal of Surgery*. 1999;86(3):338–41.
- [80] Valentim da Silva RM, Cavalcanti RL, de França Rêgo LM, Leite Nunes PF, Meyer PF. Effects of Kinesio Taping® in treatment of Cellulite: randomized controlled blind trial. *Manual Therapy, Posturology & Rehabilitation Journal*. 2014;12:175.
- [81] Murray H, Husk L. Effect of kinesio taping on proprioception in the ankle. *The Journal of Orthopaedic and Sports Physical Therapy*. 2001; 31(1): 689-26.