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Diagnosis and Management of *Varicose Vein*

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Abstract: ***Varicose veins disease are part from chronic venous disease, in addition toother chronic venous disease, is cutaneous venous dilatation, as telangectases and reticular vein. Approximately 23% of adults population in Uited States suffering from varicose vein. In general, women suffering from varicose veins more than man about 22 million in woman compared to 11 million in man. Risk factors f to be associated with varicose veins is age, sex, family history of varicose veins, obesity, pregnancy, phlebitis and previous leg injury. It also found such as environmental factors and behaviors associated with varicose veins such as standing for long periods and sitting position during work.***[1]

Keywords: varicose, vein, management

1. Anatomy and Physiology of Vein

The function of the peripheral venous system as a reserve to store blood and as a canal to restore blood to the heart. Blood entering the lower extremity venous system against the gravity and fluctuation from thoracooabdominal pressure return to the central circulation while standing. [2.3] Inferior extrememity venous divided into superficial venous system and the deep venous system that is linked by the perforator vein. The superficial vein system lies above the *fascial muscular* layer. The venous system consists of interconnected venous tissue, which functions as the primary container system and some superficial trunk vein that serves as a backflow canal to the deep venous system. The superficial vein of the lower limb is composed from short (or smaller) saphenous vein, which lies from the *ankle* region toward and joins the popliteal vein in the *saphenopopliteal junction* and large saphenesvein which flow from the ankle and join the *common femoral vein* in the *saphenophen- moral junction*. The venous system in the lower extremity composed from posterior tibial venous, peroneal vein and anterior tibialisvena on popliteal region, located beneath the *fasciamuscular* and the function to collect the flow out of the extremities. *Venous sinusoids* in the leg muscles combine to form *intermuscular venous plexi*. The paired popliteal veins linked to the arterial axial combine to form a large popliteal vein. Popliteal vein that passes through the *adductor canal* as a femoral vein. The femoral vein is formed from the *deep femoral* vein in the upper femur area and form the outflow of the lower extremity, *common femoral* and external iliacavein.[1,2]

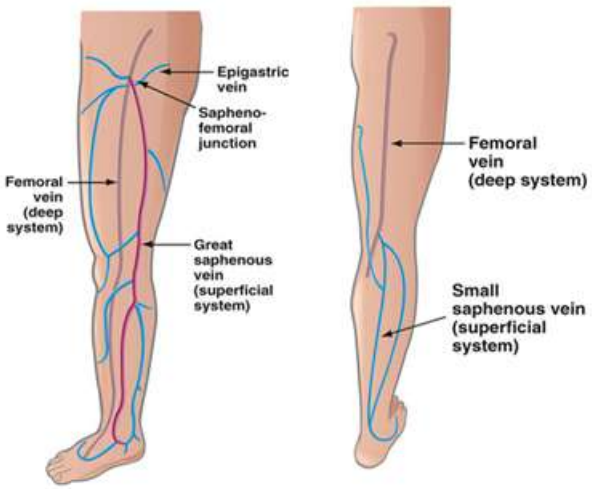

Figure 1: The deep and superficial vein system inferior extremity.[2]

The bicuspid valves are located in the deep and superficial veins and ensure the blood moves towards the *cephal*, preventing blood flow back to the feet especially when standing. The number of venous valves is greater in the proximal area than the distal feet to prevent increased pressure in the distal regions due to the influence of gravity. *Perforating veins* also consist of one-way valves to prevent blood reflux from deep veins leading to the superficial system. [3,4]

2. Risks Factor of Veinsdisfunction and Patophysiology

The risk factors associated with varicose veins are age, sex, family history of varicose veins, obesity, pregnancy, *phlebitis*, previous leg injury, long standing and sitting position at work.

Category	Risk Factor	Proposed Mechanism
Hormonal	Female gender	High estrogen state
Lifestyle	Prolonged standing and/or sitting	Venous hypertension
	Smoking	Venous endothelial injury
Acquired	Obesity	Venous hypertension
	Pregnancy	High estrogen state Venous hypertension
	Deep vein thrombosis	Deep venous obstruction Venous valvular incompetence
	Age	Venous valvular incompetence
Inherited	Family history	Venous valvular incompetence
	Tall height	Venous hypertension
	Congenital syndromes	Venous valvular incompetence Venous hypertension Deep venous obstruction

Table 1: Risk Factors occurrence varicose veins. 2

Venous pathology occurs when venous pressure is increased and reverse blood flow is impaired, this may be due to several mechanisms: venous valve incompetence (superficial veins, venous, or perforated veins) or combination with obstruction. These factors are exacerbated by muscle pump dysfunction in the lower extremities. This mechanism leads to chronic venous hypertension, fluid accumulation in the interstitial space, skin changes with hyperpigmentation, subcutaneous tissue fibrosis (lipodermatoschlerosis) and ulceration eventually.[1,2] The incompetence of the venous valve causes a *retrograde* flow of blood, however the contents of blood flow still persist from arterial *inflow*. So that venous pressure increase. The failure of the valve can be primary due to weakness of blood vessel wall or *valve leaflets* that had been there before, or secondary to trauma, *superficial phlebitis*, or excessive venous distension due to hormonal effects or high pressure. [2,5] Deep venous obstruction can restrict the flow out of the blood then increased venous pressure due muscle contraction and and secondary muscle pump dysfunction. Obstruction may occur due to intrinsic venous processes as in patients with a history *Deep vein thrombosis* (DVT) with inadequate recanalization and venous stenosis, or due to extrinsic compression, such as in May-Thurner syndrome (compression of the left vena iliaca communists were passing between right arteries iliacacommunis and lumbosacral region.[6] Muscle pump dysfunction causes venous blood not effectively emptied of the distal extremity. This incidence is rare due to a neuromuscular primary disorder or *muscle wasting syndrome*. However, clinically significant muscle pump disfunction often occurs in severe reflux or obstruction. Muscle pump dysfunction is a major mechanism of superficial venous incompetence and the complication such as venous ulcers.[1.7]

3. Clinical Manifestations

Vein varicose is adilatation of the superfisial vein and progressively can be *tortuous* and bigger. It is susceptible to invading superficial thrombophlebitis. Edema on varicose veins initially occurs in the *perimalleolar region* and progressively rises along the top of the leg depending on the accumulation of the liquid. Leg pain or discomfort is described as severe and stabbing pain after long standing and the pain is reduced by elevating the foot. Edema may produce pain due to increased *intercompartmental* and subcutaneous pressure and volume. Pain also along the varicose veins due to venous distention. Obstruction of the deep venous system can cause venous claudication, or severe leg cramps.[8] Skin changes including skin hyperpigmentation from hemosiderin deposition and eczematous dermatitis.

Clinical classification has seven categories (0-6) and further categories with presence or absence of symptoms. Classification based on congenital, primary and secondary causes of venous dysfunction.[9] Anatomical classification based on superficial venous system involvement, deep or perforating with multiple venous segment. Pathophysiological classification describes the basic mechanisms causing chronic venous disease, including reflux, obstruction of a vein or both.

Tabel 2: Classification of chronic venous disease based on clinical, etiological, anatomical, and pathophysiological.[3, 10]

Classification	Description/Definition
C.Clinical (subdivided into A for asymptomatic, S for symptomatic)	
0	No venous disease
1	Telangiectases
2	Varicose veins
3	Edema
4	Lipodermatosclerosis or Hyperpigmentation
5	Healed ulcer
6	Active ulcer
E Etiologic	
Congenital	Present since birth
Primary	Undetermined etiology
Secondary	Associated with pos thrombotic traumatic
A.Anatomic distribution (alone or in combination)	
Superficial	Great and short saphenous veins
Deep	Cava, iliac, gonadal, femoral, profunda, popliteal tibial, and muscular veins
Perforator	Thigh and leg perforating veins
P. Pathophysiological	
Reflux	Axial and perforating veins
Obstruction	Acute and chronic
Combination of both	Valvular dysfunction and thrombus

Diagnosis of Varicose Veins

The diagnosis of varicose veins obtained through patient history, physical examination and non-invasive examination. Invasive tests can also be used for diagnosis but usually invasive tests are used only to assess the severity of the disease or to determine the time when surgery should be performed.[2,8,11-13]

a. Physical examination

Physical examination has an important role in directing varicose veins therapy Skin surface is examined to see any abnormalities or bulges, hyperpigmentation, stasis dermatitis, atrophic blanche (white scar tissue at the site of previous ulcers accompanied by reduced capillaries). The distribution of varicose veins follows the course of the affected superficial vein. A standing position examination should be performed to provide the maximum distention effect on the vein. The presence of edema and its severity should be evaluated. Edema is usually *pitting*. The consistency of the calf muscles must evaluated and limb circumference should be measured which is initial sign. Ulcers are usually seen in chronic diseases. Venous ulcers are usually obtained in the medial supramalleolar area in the vein perforations and most large hydrostatic pressures area. [2.5.13]

b. Non invasive examination

Venous Duplex Imaging

Venous Duplex Imaging is an excellent method for the diagnosis of DVT. This technique is also used to confirm the diagnosis, assess the etiology and severity varicose veins. *Venous Duplex Imaging* combining B-mode imaging in superficial veins and deep veins with *Doppler assessment of flow*. The examination will provide information about the anatomical level of the disease including the superficial venous system, the deep vein, and the perforator vein. A standard duplex venous examination is performed for DVT exclusion or venous obstruction. Flow characteristics due to venous suppression are important elements of examination for thrombotic exclusion. Examination of flow direction is done with position 30 ^{0}*reversetredelenberg* at the same time performed *provocative manouvers* such as valsavamanouvers or increased flow with emphasis. The *cuff* method *inflation-deflation method* to perform rapid deflation of the cuff in the standing posi preferred for m enimbulkan reflux. [1.13]

Plethysmography

Plethysmography is one of the earliest methods for measuring blood flow in the extremities, using the principle of volume change in certain organs or regions. There are various *plethysmography* techniques but few are popular. *Air plethysmography* (APG) and *photoplethysmography* (PPG) are commonly used techniques.

Photoplethysmography

Photoplehysmography (PPG) uses a *probe* that emits infrared light on the skin and a *photoelectric* detector to measure light reflections. The relative changes in blood vessel volume on the skin (dermis) of the leg may be determined by measuring the light emitted from the probe. The PPG probe is applied to the sole of the foot, before a maneuver is performed to remove the blood on the leg with *calf muscle contraction*. Blood backflow detection by increasing the light emission and calculate the charging time. Vessel filling time is the time required for PPG *tracing* to return 90% of baseline after calf muscle contraction is stopped. The measurement is not quantitative, but has shown a correlation with invasive examination. Vessel filling time < 18-20 seconds, dependent on the patient's position, is indicated as CVI. Venous filling time> 20 seconds as normal indication. Tourniquet ingestion or low pressure of the cuff can distinguish superficial venous disease and deep venous disease.[1,8,11,13]

Air Plethysmography

The main tool in water *plethysmography* (APG) or air *plethysmography* is a blood pressure *cuff* on inflation do to optimize contact with the leg, the transducer and instrument recording. Cuff inflation in calves is only minimal, then venous emptying by elevating the limbs or moving the fingers. The patient is then instructed to stand without burdening the limbs attached to the *cuff* so that the calf muscles do not pump blood. The volume of venous filling is measured and timed. Patients with venous reflux have a shorter time. Deep venous fluxes show a greater effect than superficial reflux.

c. Invasive Inspection

Phlebography

Phlebography or venography can be ascending or descending phlebography. Ascending phlebography is doing contrast injection on the dorsum of the foot and showing the ascendering contrast within the venous system in the lower extremities. Although ascending phlebography is preferred as gold standard to determine venous patency, its role has been taken over by noninvasive imaging, where noninvasive imaging is able to provide detailed information about vein anatomy that can be useful in surgical intervention and helps to distinguish between primary and secondary diseases. [1.8,11]

Ambulatory venous pressure

An *ambulatory venous pressure* (AVP) monitor is gold standard in hemodynamic assessment vein. This technique is done with injected the needle into the *vein pedal* and connected to the *pressure transducer*. Pressure rated at rest and after activity, usually by removal of the finger. The pressure is also monitored before and after the placement of the *cuff* on the *ankle* to help distinguish deep venous reflux from the superficial vein. AVP is very important in assessing the severity and *clinical outcomes* in CVI. The

average ambulatory venous pressure (normal: 20-30 mmHg) and charging time (normally 18-20 seconds) are the most useful measurements. AVP is rarely used in clinical practice because it is an invasive and alternative method. [1.8,11]

Management of Varicose Veins

The initial therapy of varicose veins includes conservative therapy to reduce symptoms and help prevent secondary complications and progression of the disease. It is advisable to position the limbselevationally and reduce intraabdominal pressure to minimize edema. The use of *compressive stockings* is the basis of conservative therapy. If conservative therapy fails or the response is not satisfactory, further treatment may be considered depending on the anatomical and physical features.[8]Specific therapies based on the severity of the disease, with CEAP clinical classes 4-6 often require invasive therapy. Refers to the vascular specialist should be done in CEAP classes 4-6 (and possibly CEAP grade 3 with extensive edema).

Conservative Therapy

Compressive Leg Garments

The aim of this therapy is to provide a gradual external compression of the limbs and counteract the hydrostatic pressure against venous hypertension. Some of the *compression garments* available are *graded elastic compressive stockings*, *gauze boots paste*, *layered bandaging*, and *adjustable layered compression garments*. It has been established to use the total pressure of 20-50 mmHg stockings in patients with varicose veins. Therapy with a pressure of 30-40 mmHg gives significant results on the improvement of pain, swelling, skin pigmentation, and activity if the patient's compliance rate reaches 70-80%. [12]Compression s*tocking* has shown a reduction in the residual volume fraction, which is an indicator of improved calf muscle pump function, and to reduce reflux in the venous segment.[1,2]

Exercise

The abnormality of fung the calf muscle pump has a significant role in the pathophysiology of varicose veins. A gradual training program has been used as an attempt to rehabilitate muscle pumps and improve symptoms of varicose veins. Padberg et al concluded that structured exercise to improve calf muscle pump function in varicose veins provides beneficial effects as adjunctive therapy of medical therapy and surgery in severe disease.[1,2]

Pharmacological Therapy

Four groups of medications have been evaluated for the treatment of varicose veins including coumarins (α-benzopyrones), flavonoids (γ-benzopyrones), sponosides (*horse* extracts *chestnuts*), and other plant extracts. These drugs have *venoactive* abilities and are widely used in Europe but not in the USA.[4] The principle use of *venoactive* drugs in varicose veins is to improve venous tone and capillary permeability, although the exact mechanisms of these drugs are unknown. It is thought flavonoids have an effect on leukocytes and endothelium with modification of inflammatory degrees and reduced edema. The purified flavonoids fraction of daflon has been shown to reduce complaints associated with edema used as primary therapy or surgical therapy.[15]There are several drugs that are also used in the treatment of venous diseases with ulcers. Some studies suggest the use of pentoxifylline for healing of venous ulcers, although the effects are few and the role remains unclear. Aspirin and *platelet-derived growth factor* in healing or preventing recurrent venous ulcers have not been studied. There is no any data supporting the use of aspirin in the prevention of thromboembolic events in varicose veins.[3]

Invasive Therapy

Sclerotherapy

Sclerotherapy is a sclerosing agent injection into varicose veins to spoil endotelium causing thrombosis which closes the vein. *Sclerotherapy* is a therapeutic modality to *spider veins*, venous reticular and small varicose veins (<6mm). *Sclerotherapy is* not effective in large-diameter varicose veins (8-12mm) due to incompetence of large saphenous veins.[8] A common complication of *sclerotherapy* is hyperpigmentation of the surrounding skin due to hemosiderin degradation, skin necrosis, thrombophlebitis, and allergic reactions. Attempt to minimize the complications of *sclerotherapy* has been done with *microthrombectomy* with several small incisions directly on clogged varicose veins to remove thrombus. A *randomizedmulticenterstudy* found that *microthrombectomy* 1-3 weeks after injection in small varicose veins (<1mm) generate minimal hyperpigmentation, decrease the pain and inflammation.[3.8]

Surgery

In severe varicose veins, venous ulcers may require therapy for 6 months before complete healing is achieved, and recurrence often occurs especially if compression therapy is not maintained. In varicose veins which is refractory to the medical and without any invasive therapy facility, surgical treatment options should be considered to addition of*compressive stocking*, including patients who are feel uncomfortable or with venous ulcers that do not heal even with maximal medical therapy. [16]

Ligation, *Stripping* and venous *Phlebectomy*

The removal of saphenous vein with high ligation from *saphenofemoral junction* may be considered and is a standard for varicose veins. In addition, large venous varicose groups associated with incompetent saphenous vein may be avulsied by the same arrangement with a technique called *stab phlebectomy*.[3]

Ablation Therapy with Radiofrequency and Laser Endovena

Recent venous ablation therapy is to use heat energy in the

form of radiofrequency or laser for obliteration of the veins. This technique is often used in large saphenous venous reflux as an alternative therapy of *stripping*.[3] Heat causes local thermal injury to the vein wall resulting in fibrosis and thrombosis. Radiofrequency ablation in large saphenous veins, obtained 85% complete obliteration after 2 years with 11% comitization, 95% of patients reported satisfied and showed improvement of symptoms.[17]

Endovascular therapy

Endovasculartherapy on varicose veins become very important to restore the *outflow* of the venous system and relieve obstruction. Before endovascular therapy venous iliac stenosis and obstruction causing varicose veins are treated by surgical procedures such as *cross-femoral venous bypass* or iliac vein reconstruction with prosthetic material. Because the venous *stent* procedure provides good results, venous bypass surgery is rarely performed.[2, 20]

Subfascial Endoscopic perforator surgery(SEPS)

The principle of surgery in advanced varicose veins is perforator venous ligation that contributes to focal high pressure in superficial veins. This ligation will be difficult to do with traditional surgical techniques due to tissue damage at the site of surgery. *Subfascial endoscopic perforator surgery* (SEPS) provides a tool for incompetent perforator venous ligation by obtaining access from distant sites of *lipodermatosclerosis* or ulcers. [2.3]

Veins reconstruction

Injury or dysfunction of the venous valve contributes to the development and progression of varicose veins. Deep vein reconstruction has been performed in certain patients with advanced stage varicose with ulcers that often recur with severe symptoms. The open valvuloplasty technique has been refined, and the closed technique for venous repair is developed with *transcommisuralvalvuloplasty*. *Vein Valvuloplasty* has demonstrated 59% competence and is free from recurrence of 63% ulcer within 30 months.[1,2]

4. Summary

Varicose veins diseases are part from chronic venous disease, about 23% of population adults in America States suffering from varicose vein. More often in women than men. Venous pathology occurs when venous pressure is increased and reverse blood flow is impaired, this may be due to several mechanisms: venous valve incompetence (superficial veins, venous, or perforated veins), venous or combination obstruction. The use of *plethysmography is* often used to assist in assessing the severity of the disease. Treatment of varicose veins is based on the severity of the disease and guided by anatomical and pathophysiological considerations. *Compressive garments* are the main therapies. The classical surgical techniques and the latest intervention methods become consideration if conservative therapy does not provide satisfactory results.

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