

Chronic Venous Insufficiency in A Woman with Standing Profession

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Introduction

The prevalence of Chronic Venous Insufficiency (CVI) give a high social impact. In Europe, approximately 40% of the adult population affected by venous disorders. In general, the prevalence of CVI is about 5% - 15%. The prevalence is increasing by age.¹

Several risk factors have been associated with the occurrence of CVI, such as older age, female, family history, obesity, standing occupation, and pregnancy. CVI prevalence in women is 1% -40% in some areas, while in men 1% - 17%. Physical activity and ergonomic position contribute to the occurrence of CVI. Orthostatic type of work increase the prevalence and severity of CVI. Women with jobs that require standing for a long time about 4 hours will have a 2.7 times higher risk of going CVI.²

This case report will discuss the clinical manifestations, classification and management of CVI on 60 years old women with several risk factors including standing occupation.

Case

A 60 years old women came to the primary health care with intermittent leg swelling and pain on the afternoon. Since two months prior to the admission, patient had been complaining about her swollen feet after long duration on standing position at work, more often felt on her left leg. Swelling and stiffness often appeared in the afternoon. Patient then got pain on her foot. There was no worsening of pain during walking. Her blood glucose was checked and the result was normal. History of previous swollen at the feet was denied. History of leg trauma, blockage in the leg veins were also denied. She was referred to Sardjito Hospital for vascular examination and revealed a CVI. The risk factors of CVI that can be identified are: aged of 60 years, a teacher occupation with a long time standing position (about 4-5 hours) every day, obesity (her BMI was more than 30 kg/m²), and lack of physical activities.

On physical examination, the general status was good and full of consciousness. Blood

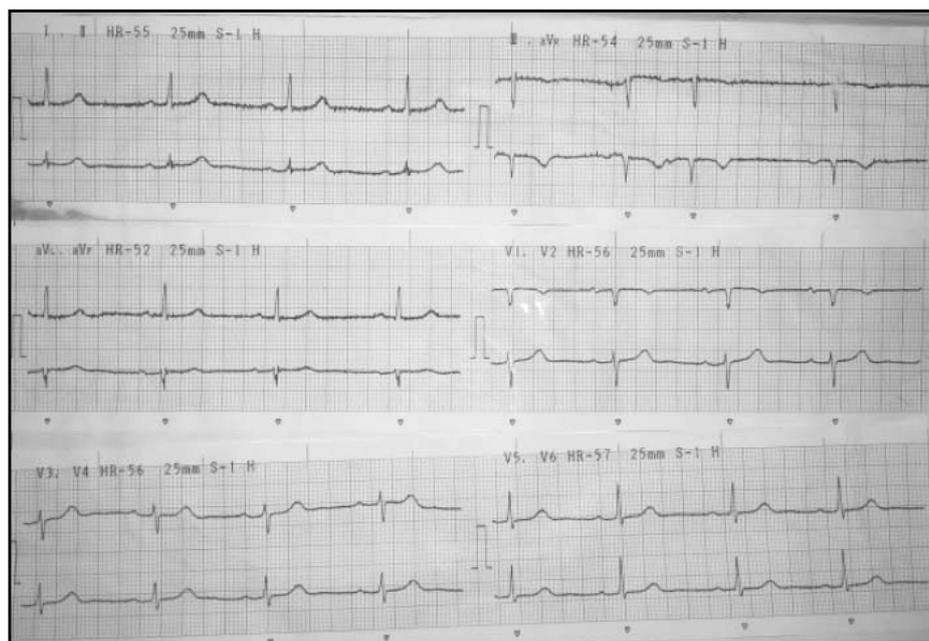


Figure 1. Electrocardiogram showed sinus rhythm with heart rate was 55 beats perminute.

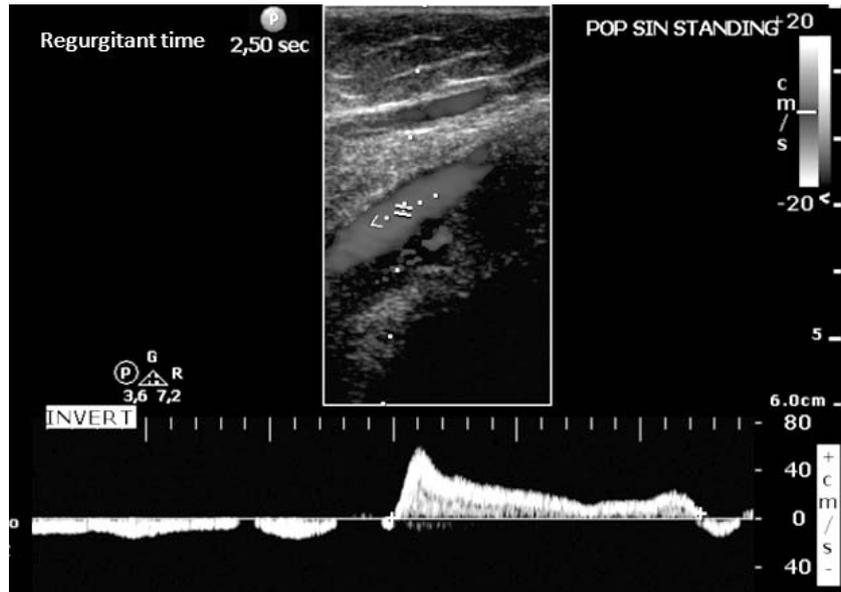


Figure 2. Pulse wave Doppler (DUS) examination showed a reflux at the left popliteal vein after compression distal to the vein (squeeze distal). The regurgitant time showing severe result (2.5 s).

pressure 120/80 mm Hg, regular heart rate at 80 beats/min, respiratory rate 20 breaths/min, and her temperature was of 36.5 ° C. Head examination was normal. There were no elevated jugular venous pressure or lymphadenopathy. Chest examination was normal, lung sound was vesicular, symmetric and no lag of motion. Examination of the heart revealed no abnormalities. Examination of the abdomen was within normal limits, there were no tenderness and liver or spleen enlargement. On extremity examination, there were strong arterial pulsation, warm skin, and found a scar and ulceration on the medial aspect of the left foot. There were no edema in both legs. Peripheral arterial oxygen saturation by pulse oximetry on inferior extremity was around 98% - 99%. An electrocardiographic examination showed sinus rhythm with the heart rate was 55 bpm (Figure 1).

Duplex ultrasonography examination found a venous regurgitation with regurgitation time was 2480 msec in the right femoral vein, 830 msec in left femoral vein, and 2500 msec in the left popliteal vein. Then patient was confirmed that she got a severe venous valve insufficiency on bilateral femoral vein and in the left popliteal vein. Arterial and venous flow were normal. Patients then treated with the installation of stocking compression on her right and left inferior extremity and given a micronized purified flavonoid fraction (Ardium®) 500 mg twice daily.

Discussion

Physiologically, vein serves primarily as a conduit to travel blood back from the capillaries to the heart. In addition, it also serves as a blood reservoir, vascular capacity regulator, part of peripheral pump, as well as involved in the regulation of body temperature. The vein is divided into superficial and deep vein. This division is based on the location of that vasculature. The superficial veins has large diameter, thick wall, muscular characteristic, and located beneath the skin. Deep vein generally runs along the artery and the diameter is about three times larger than the adjacent artery. Perforate veins connects superficial veins with the deep veins. One of the most important structures of the veins is a one way bicuspid valve that prevents blood reflux.¹

Our patient came with symptomatic manifestation as oedema and pain in the leg especially in the afternoon. The clinical manifestations of venous disorders range from asymptomatic to symptomatic, such as dilatation of the superficial veins until skin wounds. Some of the most frequent manifestation are teleangiectasis, varicose vein and CVI. Intramuscular or intermuscular deep veins are important in determining the occurrence of CVI. There are three pairs of intermuscular veins named in accordance with the arteries that accompany the posterior tibial vein behind the medial malleolus,

anterior tibial vein coming from the dorsalis pedis and peroneal veins found in the tibia and fibula. The popliteal vein is an important intermuscular vein that would later become the femoral vein. After passing through the inguinal ligament it becomes femoral vein and continues to iliac vein and the inferior vena cava. Venous flow is determined by venous valves and venous pump. Bicuspid venous valves drain blood from distal to proximal and from superficial to deep veins except at the foot. At the foot, blood flows from deep to superficial vein. Venous pump is a pump caused by leg muscle compression and causing venous flow. The blood flows into proximal deep veins by muscular contraction. The effectiveness of the pump is then determined by the conditions of competent venous valves. Competent venous valves have two functions, to prevent transmission due to high pressure in the superficial veins and capillaries during muscle contraction and prevents muscle contraction at the end of backflow into the superficial veins. When it goes well, mean venous pressure in the superficial veins is around 20-30 mmHg. If the average venous pressure rises to 60-90 mmHg, it will cause venous hypertension and may alter the anatomy, physiology, and histopathology associated with CVI.³

Epidemiology and prevalence of varicose veins in women is two times higher than in men. Increasing age is a risk factor for CVI and varicose veins. CVI prevalence rises to 21.2% in men aged > 50 years and 12% in women > 50 years. The presence of family history of varicose veins will increase the risk of varicose veins. Forty-two percent of women in Japan with varicose veins have family history, as compared to 14% if the downregulation of desmulin gene will affect the smooth muscle cells in the the saphenous vein wall. A job that requires standing position in a long period of time is an independent factor for CVI occurrence. It was found that the prevalence CVI in standing profession is 36% compared to 27% in others.⁴ Body mass index (BMI) > 30 kg/ m² increase the risk of CVI. In this patients, she had several risk factors that can induce the occurrence of CVI, such as women, age > 50 years, working with long-duration of standing position up to 4-5 hours per day, obesity with BMI > 30 kg/ m², while family history was denied.

Professions requiring long period of time in standing position were associated with increased of CVI compared to professions with various

different movements. Number of years in those profession has a positive correlation to the severity of CVI. This is because the calf muscle pump is not very often used in those workers. Muscle pump inactivity will increase venous pressure.⁵ Standing profession is defined as profession with duration of standing position > 75% of their work duration.⁴ Women with jobs that require standing position for a long time, about 4 hours will have higher risk of CVI.²

Several theories have been proposed as the basic cause of CVI. There are two theories that are universally accepted, the primary valve incompetence and congenital vein wall weakness. Some of the factors that influence the occurrence of CVI are venous stasis, venous hypertension, fibrin cuff, water hammer effect, and trapping leukocytes. Venous stasis occurs due to accumulation of the stagnant blood in the veins that become tortuous, wider and lead to tissue anoxia and cell death, causing skin changes and ulcerations. Venous hypertension causes muscle pump dysfunction and venous ulceration. The hydrostatic pressure between the deep veins and superficial veins at rest and standing position is the same. During the contraction of the calf muscles, deep vein pressure will be higher than the superficial veins. Valve closure needed to prevent transmission of pressure to the superficial vein. But in the incompetent valve and pump dysfunction, pressure was transmitted to the superficial veins, causing CVI symptoms and ulcerations. Pericapillary blockage by fibrin (fibrin cuff) was related to restriction of oxygen diffusion that passes through the vessel. It will cause edema and dermatoslerotic changes in the skin. Water hammer effect theory stated that reflux was transmitted mainly through the perforator into the superficial veins which time a break of 20 -25% in patients with normal venous pressure but venous hypertension will occur by the induction of valsava maneuver.⁶

Complaints sometimes are subjective and unclear in the beginning of disease, common symptoms including a heat-burning sensation, swelling, throbbing, cramping, aching, heavy sensation, restless legs, and tired legs sensation. Some varicose veins patients, 18% came with persistent symptoms and 50% with episodic symptoms. Pain caused by CVI may be provoked by walking, warm feeling of the foot and relieved by cold compression, and compression stockings will reduce pain. Ulceration can be a clinical

Characteristic	Arterial ulcer*	Venous ulcer	Neuropathic ulcer*
Location	Over toe joints, malleoli (over the bony prominence), anterior shin, base of heel, pressure points	Medial and lateral malleolar area above bony prominence, posterior calf, may be large, circumferential	Plantar surface of foot over metatarsal heads, heel, pressure points
Appearance	Irregular margins, base dry and often pale or necrotic (brown/black fibrous tissue)	Irregular margins, pink or red base that may be covered with yellow fibrinous tissue, exudate common (may be heavy); ulcers can be large, sometimes circumferential	Punched out ulcer, usually superficial but sometimes deep, red base
Ulcer within callus	Rare	No	Calloused border, ulcer can be underlying a callus
Foot temperature	Warm or cool	Warm	Warm
Pain	Yes, may be severe	Yes, usually mild but may be severe	No
Arterial pulses	Absent	Present	Present or absent
Sensation	Variable	Present	Absent tactile, pain, temperature and vibratory sensations
Foot deformities	No	No	Often
Skin changes	Shiny, taut, loss of hair Dependent rubor of leg and foot that becomes pale with leg elevation	Erythema, brown-blue hyperpigmentation can be spotty or diffuse; "stasis" changes; atrophie blanche (white sclerotic areas), edema; dry skin; varicose veins common; if lipodermatosclerosis is present, skin may be bound down; bilateral lower extremities often affected	Waxy or shiny, loss of hair, may be taut; dry skin; may have non-pitting edema, especially on dorsal foot
Reflexes	Present	Present	Absent

Table 1. Differences in characteristics related injuries veins, arteries.⁸

Table 2. Classification of venous anatomy segment¹⁰

Superficial vein	Deep vein	Perforate vein
1. Reticular vein	1. Inferior vena cava	1. Thigh
2. DSV above knee	2. Common iliac vein	2. calf
3. GSV below knee	3. Internal iliac veins	
4. Lesser aphenous veins	4. Esternal iliac veins	
5. Nonsaphenous veins	5. Pelvic	
	6. Common femoral veins	
	7. Deep femoral vein	
	8. Femoral veins	
	9. Popliteal veins	
	10. Crural: antero posterior tibial, peroneal veins	
	11. Muscular/other	

manifestation which usually appears in the medial malleolus, where there is a maximum venous pressure caused by the presence of large vein perforation. Lipodermatosclerosis is skin discoloration on the lower extremity including capillary proliferation, fat necrosis, and fibrosis of the skin in the subcutaneous tissue. The skin becomes red and brown due to hemosiderin deposition of the red blood cells. CVI clinical course was consistent with progressivity of chronic venous stasis and hypertension. Swelling is caused by reflux and obstruction. The oedema type is pitting. Flushed skin discoloration or brownish is because CVI is localized along the medial part of the lower leg. Unhealed ulcer localized to the medial part of the lower extremity can be a symptom of CVI.⁷ On this patient, the initial complaints were swelling in the legs and soreness of the foot, especially in the afternoon. Later she complained about her injured left leg below the medial portion that was consistent to the CVI ulcer characteristic.

Patients with CVI and ulcerations may also have an arterial disease at the same time. Thus in patients with ulceration, measurements of ankle-brachial index (ABI) is also required. Distinguishing CVI ulceration from arterial disease can be seen in table 1.³ According to this table it can be concluded that the characteristics of the wound in our patient was venous-associated ulceration, with ABI score 0.9 still in normal ranges.

There are several criteria used for the classification and severity of CVI. The CEAP (Clinical, etiology, anatomic, Pathophysiology) is a classification module that is used to report, diagnose and treat CVI. As a note, CVI is a progressive disorder so serial CEAP classification was necessary. For report writing, a basic and advanced format CEAP can be used, where the difference is in the advanced CEAP classification, venous anatomy segment was included.⁹ Clinical manifestations in our patient were skin changes with healed ulceration, no history of post

CEAP classification for chronic venous disorders	
Clinical classification	
C0	No visible or palpable signs of venous disease
C1	Telangiectasias, reticular veins, malleolar flares
C2	Varicose veins
C3	Edema without skin changes
C4	Skin changes attribute to venous disease (eg, pigmentation, venous eczema, lipodermatosclerosis)
C4a	Pigmentation or eczema
C4b	Lipodermatosclerosis or atrophie blanche
C5	Skin changes as defined earlier with healed ulceration
C6	Skin changes as defined earlier with active ulceration
S	Symptomatic, including ache, pain, tightness, skin irritation, heaviness, and muscle cramps, and other complaints attributable to venous dysfunction
A	Asymptomatic
Causal classification	
Ec	Congenital
Ep	Primary
Es	Secondary (postthrombotic)
En	No venous cause identified
Anatomic classification	
As	Superficial veins
Ap	Perforator veins
Ad	Deep veins
An	No venous location identified
Pathophysiologic classification	
Pr	Reflux
Po	Obstruction
Pr,o	Reflux and obstruction
Pn	No venous pathophysiology identifiable

Figure 4. CEAP classification for CVI.¹¹

Revised venous clinical severity score				
Attribute	Severity Score			
	None: 0	Mild: 1	Moderate: 2	Severe: 3
Pain or other discomfort (ie, aching, heaviness, fatigue, soreness, burning) Presumes venous origin	—	Occasional pain or other discomfort (not restricting regular daily activities)	Daily pain or other discomfort (interfering with but not preventing regular daily activities)	Daily pain or discomfort (limits most regular daily activities)
Varicose veins Varicose veins must be ≥ 3 mm in diameter to qualify in the standing position	—	Few: scattered (ie, isolated branch varicosities or clusters) Also includes corona phlebectatica (ankle flare)	Confined to calf or thigh	Involves calf and thigh
Venous edema Presumes venous origin	—	Limited to foot and ankle area	Extends above ankle but below knee	Extends to knee and above
Skin pigmentation Presumes venous origin Does not include focal pigmentation over varicose veins or pigmentation caused by other chronic diseases (ie, vasculitis purpura)	None or focal	Limited to perimalleolar area	Diffuse over lower third of calf	Wider distribution above lower third of calf
Inflammation More than just recent pigmentation (ie, erythema, cellulitis, venous eczema, dermatitis)	—	Limited to perimalleolar area	Diffuse over lower third of calf	Wider distribution above lower third of calf
Induration Presumes venous origin of secondary skin and subcutaneous changes (ie, chronic edema with fibrosis, hypodermatitis). Includes white atrophy and lipodermatosclerosis	—	Limited to perimalleolar area	Diffuse over lower third of calf	Wider distribution above lower third of calf
Active ulcer number	0	1	2	3
Active ulcer duration (longest active)	N/A	<3 mo	>3 mo but <1 y	Not healed for >1 y
Active ulcer size (largest active)	N/A	Diameter <2 cm	Diameter 2–6 cm	Diameter >6 cm
Use of compression therapy	Not used	Intermittent use of stockings	Wears stockings most days	Full compliance: stockings

Figure 5. Severity score of venous disorders.¹²

thrombotic, located in the femoral and popliteal veins, with the reflux pathophysiology. Based on CEAP classification, our patients is C5; Ep; Ad; Pr 13, 14, level II.

In the DUS examination, the limit for deep venous reflux is more than 1000 msec, the superficial vein is more than 500 msec and the perforator veins are more than 300 msec. Doppler

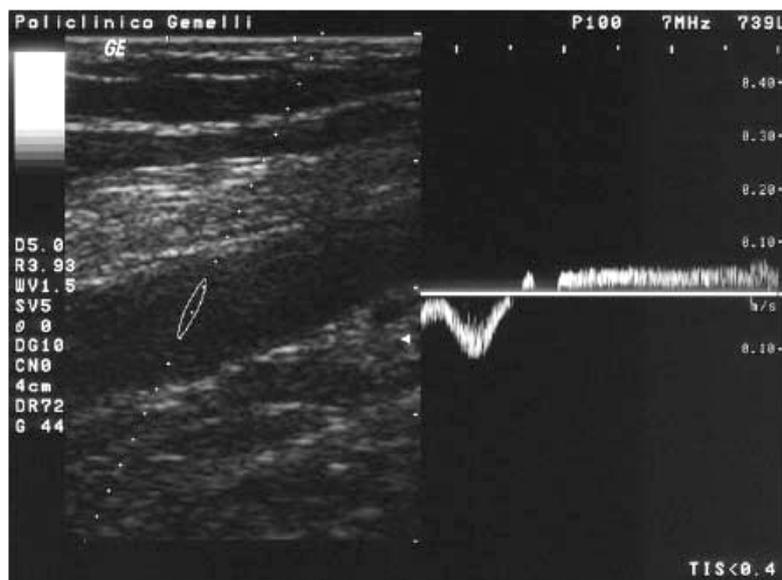


Figure 6. Doppler reflux pulse wave examination result with compression maneuver.¹³

examination was done while lying and standing by using color Doppler and pulsed wave Doppler. The existence of a reflux is the predominant finding in primary CVI. To identify CVI, reflux may be provoked by performing compression on the calf and Valsalva maneuver.¹⁴ In our patients doppler examination, the regurgitation time measured on the right femoral vein was 2480 msec, on the left femoral vein was 830 msec and on the left popliteal vein was 2500 msec.

Management of CVI is intended to reduce symptoms and repair the cause of the abnormality. Ranging from the use of drugs, use of compression such as compression stockings, and surgery. Some drugs used to relieve symptoms in patients with CVI were divided into 4 groups, those are venoactives, hemorrhheologic agents, antiplatelet agents, and other agents. Venoactive drugs suchn as phlebotonic agent works to increase the venous tone and capillary permeability. Hemorrhheological such as Pentoxifylline, can be used to treat CVI by inhibiting cytokines and reducing white blood cell adhesion to the endothelium and releasing free radicals. Other agents such as zinc supplements, prostaglandin E1 was found to be ineffective to overcome ulcer due to venous disorders.⁹ The use of compression stockings is intended to put pressure 30-40 mmHg or 40-50 mmHg on the ankle and gradually reduce pressure on the proximal part of the foot to restore venous flow pattern both on the superficial and deep veins. Putting the foot in higher position when

sitting or lying down will reduce the pressure on the veins and reduce edema.¹⁵ In patients with symptomatic CVI who fail conservative therapy, invasive advance management might be required, as in CVI classification CEAP class 4 to 6 with recurrent or unhealed ulceration and progressive infection and lymphedema. Superficial venous reflux can be healed with a cool touch laser ablation, RFA therapy, venous sclerotherapy, and venous phlebectomi. Deep venous reflux valve can be healed by reconstruction surgery or valvuloplasty. It also can be done by valve replacement and valve transposition procedure to patient whose valvuloplasty cannot be done. Subfascial endoscopic perforator surgery (SEPS) can be done in case of any incompetence of the perforator vein. In case of CVI with venous obstruction, endovascular procedure such as stenting on iliac vein can be done.⁶

In our patients, conservative therapy with the use of medications and compression stockings were conducted. Evaluation report showed a good results thus invasive intervention did not required. Education regarding control of risk factors such as reducing the time associated with long-duration of standing during working and reducing weight had been done to improve the outcome.

Conclusion

We reported 60 years old women with severe chronic venous insufficiency (CVI) present with

symptomatic complaints and several risk factors such as: age > 50 years, standing occupation, obesity with BMI more than 30 kg/m². This diagnosis cannot be obtained by physical examination alone but need Doppler Ultrasonography as further examination. Management of CVI was carried out according to CEAP classification standards that can be used to determine the progression and further management plan. CVI management aims to reduce symptoms and repair the cause of the abnormality which consists of the use of drugs, use of compression as compression stockings until the invasive action.

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