Chronic Venous Disease – Management Update

MD. SAIF ULLAH KHAN¹, MOHAMMAD SAFIUDDIN², MD. MAHBUBUR RAHMAN¹

¹Department of Vascular Surgery, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, ²Department of Cardiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka

Address for Correspondence: Dr. Md. Saif Ullah Khan, Assistant Professor, Department of Vascular Surgery, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, E-mail: drsaif murad@yahoo.com

Introduction:

Chronic venous disease (CVD) of the lower limbs is manifested by a range of signs, the most obvious of which are varicose veins and venous ulcers. However, the signs also include edema, venous eczema, hyperpigmentation of skin of the ankle, atrophie blanche and lipodermatosclerosis. Chronic venous disease can be graded according to the descriptive clinical (C), etiological (E), anatomical (A), and pathophysiological (P) classification (CEAP classification, published in 1995 and updated in 2004), which provides an orderly framework for communication and decision making.² The clinical signs in the affected legs are categorized into seven classes designated C₀ to C₆ (Table 1. Revised CEAP Classification). Chronic venous disease encompasses the full spectrum of signs and symptoms associated with classes C₀ to C₆. Whereas the term "chronic venous insufficiency" is generally restricted to disease of greater severity (i.e., classes C_4 to C_6). Chronic venous insufficiency (CVI) may affect up to 20% of adult. 10% to 35% of adults in the United States have some form of chronic venous disorder.³ Overall, chronic venous disease has been estimated to account for 1 to 3 percent of the total health care budgets in countries with developed health care systems.4

Risk factors

- Deep vein thrombosis (DVT)
- · Family history of varicose veins
- · Sedentary lifestyle
- Overweight
- Pregnancy
- Not exercising enough
- Smoking
- Extended periods of standing or sitting
- Being female (related to levels of the hormone progesterone)
- Being tall
- Genetic factors
- Age over 50

Pathophysiology

Pathophysiology of CVD is classified as reflux, obstruction, or both. The vast majority of patients with

CVD have reflux.5 Despite the diversity of signs and symptoms associated with chronic venous disease, it seems likely that all are related to venous hypertension. In most cases, venous hypertension is caused by reflux through incompetent valves, but other causes include venous outflow obstruction and failure of the calf-muscle pump. 1 Reflux may occur in the superficial or deep venous system or in both. Valve damage may occur as the result of aging, extended sitting / standing or a combination of aging and reduced mobility. In many patients varicose veins will be present in conjunction with chronic venous insufficiency, but this is not always the case. There are many patients with typical changes of chronic venous insufficiency, but no obvious problem with their superficial veins. These patients may have abnormalities in the deeper veins which will only be apparent on special scans. The transmission of high venous pressures to the dermal microcirculation causes extravasation of macromolecules and red blood cells that serve as the underlying stimulus for inflammatory injury.³

Symptoms

- Ankle swelling
- Discomfort- tightness in calves, burning, itching, dull ache, or heaviness in affected calves or legs.
- Tired, restless legs
- Pain while walking or shortly after stopping (Pain that gets better when legs are raised).

Signs

- Swelling of the ankles and/or calves
- Change in skin color, usually around the ankle
- Rash
- · Signs Telengectasia
- Reticular veins
- Varicose veins
- Pigmentation
- eczema
- Thickening and hardening of the skin on the legs and ankles (lipodermatosclerosis)
- Atrophie blanche
- Skin ulcer

CEAP classification

The CEAP classification was developed at a 1994 international consensus committee meeting sponsored by the American Venous Forum. Venous disease of the legs can be classified according to the severity, cause, site and specific abnormality using the CEAP classification. Use of such a classification improves the accuracy of the diagnosis and improves communication between specialists.



Fig.-1:

In 2000, Rutherford proposed a new tool to measure the severity of venous disease⁶. Its aim was to quantify the progression and treatment of chronic venous disease. These scores were mainly developed for severe forms of CVD according to the CEAP clinical classification (grades C4, C5, and C6), ie, for cases designated traditionally in terms of chronic venous insufficiency (CVI). These severity scores for CVD grade clinical severity, anatomo-pathophysiologic



Fig.-2:

Table-IP Classification (Revised)

	CEAP Classification (Revised)
Clinical classification (C)	C0: no visible or palpable signs of venous disease
	C1: telangiectasias or reticular veins
	C2: varicose veins (>3 mm diameter)
	C3: edema
	C4: skin and subcutaneous tissue changes C4a: pigmentation or eczema C4b: lipodermatosclerosis or atrophie blanche
	C5: healed venous ulcer
	C6: active venous ulcer
Each clinical class is further characteri	zed by a subscript for symptomatic (S) or asymptomatic (A)
Etiologic classification (E)	Ec: congenital
	Ep: primary
	Es: secondary (postthrombotic)
	En: no venous cause identified
Anatomic classification (A)	As: superficial veins
	Ap: perforator veins
	Ad: deep veins
	An: no venous location identified
Pathophysiologic classification (P)	Pr: reflux
	Po: obstruction
	Pr,o: reflux and obstruction
	Pn: no venous pathophysiology identifiable

severity and disability. Without a doubt, these severity scores deserve to be tested and evaluated by their potential users.

Diagnostic evaluation

Diagnostic evaluation of CVI can be logically organized into 1 or more of 3 levels of testing, depending on the severity of the disease:

- Level I: History and clinical examination, which may include use of a hand-held Doppler scanner.
- Level II: noninvasive vascular laboratory testing, which now routinely includes duplex color scanning
- Level III: invasive investigations or more complex imaging studies, including ascending and descending venography, venous pressure measurements, computed tomography (CT), venous helical scanning, or magnetic resonance imaging (MRI).

Measurement of ambulatory venous pressure remains an important invasive diagnostic tool and identifies the standing pressure nadir as well as the rapidity of emptying and refill. In practice, these examinations are often supplanted by more patient-friendly, noninvasive plethysmographic studies, which provide an objective measure of volume changes related to posture and to function of the calf muscle pump. However, as duplex ultrasonographic diagnosis has assumed an increasingly dominant diagnostic role, disease classification is often assigned on the basis of duplex scanning information alone. Magnetic resonance venography (MRV) offers yet another alternative for clarification of proximal diagnostic questions. The evolution of new imaging techniques in MRV and computed tomography venography (CTV) holds as much or more promise for elucidation of venous disease than of arterial disease. Both modalities require only Venepuncture for image optimization.⁴

Treatment

A. Conservative treatment

- · Avoiding long periods of standing or sitting
- Intermittent elevation of the legs during the day and elevation of the legs at recumbency
- · While sitting, leg should be above the thigh
- · Maintaining ideal body weight
- Beginning a walking program
- Avoiding crossing the legs
- Compression therapy Compression therapy using pumps, bandaging, and/or graded compression stockings is the mainstay of treatment⁷.
- Micronised purified flavonoid fraction (Diosmin+ Hesperidin)

B. Vein ablation treatments

- a. Sclerotherapy-(Sodium tetradecyl sulfate, Sodium morrhuate, Hypertonic solution of sodium chloride, Polidocanol, Sodium iodide, Ethanolamine oleate)
- b. Radiofrequency ablation
- c. Laser ablation
- C. Surgical procedures
- Vein stripping
- Ambulatory phlebectomy
- · Bypass surgery
- Valve repair
- Valve Transplantation
- Valve Transposition
- Valve Substitutes
- Linton's operation
- Endoscopic ligation of perforators
- Stenting

Many patients without symptoms or signs of lipodermatosclerosis or ulceration simply require reassurance. 8 Others presenting for treatment are concerned about the cosmetic appearance of their limbs. Patients should be given a trial of graded compression stockings and further instructions to avoid prolonged standing and to elevate the feet above the heart while lying down as well as several times during the day.⁴ Stockings in the lower pressure range (class 1, 20 to 30 mm Hg) are sufficient to control edema, but higher pressures (class 2, >30 to 40 mm Hg; or class 3, >40 mm Hg) are recommended to control venous dermatitis or ulcers. 9 In a prospective randomized double-blind controlled trial, micronised purified flavonoid fraction did not change the symptoms of CVI except for nighttime cramping 10. Surgical therapy for CVI should be considered in patients who have failed less invasive treatments.⁴ Patients with varicose veins and superficial venous reflux can undergo saphenous vein high ligation and stripping or other endovascular techniques along with branch varicosectomy to remove the offending reflux. Alternatively, sclerotherapy or other minimally invasive techniques can be used in some cases. Surgical correction of superficial venous reflux in addition to compression bandaging does not improve ulcer healing but reduces the recurrence of ulcers. 11

Prevention

Certain factors such as genetic predisposition, gender and increasing age are unavoidable. Weight control, adequate

physical exercise, avoidance of smoking, avoidance of sedentary activities and control of hypertension may all be significant in preventing the development of varicose veins.

References:

- John J. Bergan, Geert W. Schmid-Schönbein, Philip D. Coleridge Smith, et al. Chronic Venous Disease. N Engl J Med 2006; 355: 488-498.
- Eklof B, Rutherford RB, Bergan JJ, et al. Revision of the CEAP classification for chronic venous disorders: consensus statement. J Vasc Surg 2004; 40: 1248-1252.
- Neglen P, Thrasher TL, Raju S. Venous outflow obstruction: An underestimated contributor to chronic venous disease. J Vasc Surg 38: 879–885, 2003.
- 4. Robert B. Rutherford. Vascular Surgery, 6th ed; Elsevier, Inc.
- Ruckley CV. Socioeconomic impact of chronic venous insufficiency and leg ulcers. Angiology1997;48:67-69.

- Rutherford RB, Padberg FT, Comerota AJ, et al. Venous Severity Scoring: An adjunct to venous outcome assessment. J Vasc Surg 31:1307–1312, 2000.
- Rathbun SW, Kirkpatrick AC. Treatment of chronic venous insufficiency. Curr Treat Options Cardiovasc Med. 2007 Apr;9(2):115-26.
- Norman S. Williams. Bailey & love's short practice of surgery, 25th ed. edward arnold (publishers) ltd.
- Seshadri Raju, and Peter Neglén. Chronic Venous Insufficiency and Varicose Veins. N Engl J Med 2009; 360:2319-2327
- Danielsson G, Jungbeck C, Peterson K, Norgren L: A randomised controlled trial of micronised purified flavonoid fraction vs placebo in patients with CVI. Eur J Vasc Endovasc Surg 2002; 23:73.
- Manjit S Gohel, Jamie R Barwell, Maxine Taylor. Long term results of compression therapy alone versus compression plus surgery in chronic venous ulceration (ESCHAR): randomised controlled trial. BMJ 335 : 83 doi: 10.1136/ bmj.39216.542442.BE (Published 1 June 2007).