

Chronic Venous Insufficiency, Venous Hypertension and its complications

Chronic venous insufficiency (CVI) is a common condition, affecting 2-5% of the population. It is related to primary or secondary dysfunction of the musculo-venous system resulting in chronic ambulatory venous hypertension and eventual microangiopathic changes of the skin and pannicular with associated complications

Primary dysfunction result in reflux from

- Varicose vein disease with valvular and or venous wall abnormalities of the superficial and deep system

Secondary changes from

- Deep and Superficial Venous Thrombosis with subsequent recanalization resulting in obstruction and or reflux due to valvular damage

Chronic venous insufficiency is caused by malfunction of the venous system associated with chronic venous hypertension. When the venous system is normal, exercise such as walking activates the calf muscle pump, reducing the venous pressure in the foot from 90 mm Hg on standing to around 30 mm Hg during walking.

With every thigh and calf muscle contraction, blood is expelled out of the leg. During muscle relaxation, the competent valves prevent reflux. The venous pressure remains low because of slow filling from arterial inflow. Obstruction to venous outflow or reflux due to valve damage interferes with this normal mechanism and results in high ambulatory venous pressure during exercise

Many previous pathophysiological explanations for the changes seen in CVI are now considered to be out of date.

Dysfunctional Venous systems are now believed to be related to injury to the vein walls and venous valves. This injury is largely due to a sterile inflammatory process. There are other contributing factors and these may include

- Genetics
- Obesity
- Female gender
- Pregnancy
- Standing occupations in females.

Vein wall injury results in the clinical presentation of tortuosity and dilatation seen in varicose veins.

Increasing venous diameter leads to functional valvular failure and reflux.

The effect of this reflux is chronic ambulatory venous hypertension leading to microangiopathic skin changes.

Clinically it manifests itself as lower extremity oedema, pain, itch, skin colour change and ulceration.

The earliest sign of venous hypertension are elongated and dilated veins in the epidermis and dermis called *telangectasia*. Deeper subcutaneous *reticular veins* may dilate and

elongate. Above the superficial fascia are the *varicose veins* which become tortuous, dilated and incompetent.

The exact mechanism of vein wall and valvular dysfunction seen in CVI is still controversial.

However, we can consider this to be a chronic inflammatory process triggered by venous hypertension, in which there is a white cell /endothelial interaction with the markers of chronic sterile inflammation.

There is evidence that leukocyte migration occurs into the parenchyma of venous valves and vein walls, resulting in destruction of elastin and possibly collagen leading to perforated scarred, non functioning valves and elongated, thinned wall varicose veins.

The other manifestation of venous hypertension is in the skin, where again leukocyte activation and inflammatory responses have been implicated as playing a major role in the pathophysiology of CVI

Complications of CVI

- Varicose veins
 - Cosmetic problem
 - Symptoms
 - Complications SVT, Bleeding. Varicose ulcers
- Oedema
- Pain
 - Venous hypertension in muscles and fascial compartments of the lower leg from exercise and prolonged standing results in the characteristic ache of CVI. The discomfort is described as pain, pressure, burning, itching, dull ache, or heaviness in affected calves or leg
- Venous dermatitis,
- Lipodermatosclerosis:
 - These characteristic skin changes in the lower extremities include capillary proliferation, fat necrosis, and fibrosis of skin and subcutaneous tissues. Skin becomes reddish or brown because of the deposition of hemosiderin from red blood cells
- Ulceration

Current theory implicates inflammation as the cause for valve, vein wall and advanced skin changes in chronic venous dysfunction. This is triggered by chronic ambulatory venous hypertension.

Hence the main stay of treatment is correction of this venotensive state.