

Chronic venous insufficiency with lower extremity disease: Part 1

The “broken rungs and water balloon” disorder

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Chronic venous insufficiency (CVI) is the most common cause of lower extremity wounds. The venous tree is defective, incapable of moving all the blood from the lower extremity back to the heart. This causes pooling of blood and intravascular fluid at the lowest gravitational point of the body—the ankle.

This article has two parts. Part 1 enhances your understanding of the disease and its clinical presentation. Part 2, which will appear in a later issue, explores the differential diagnosis of similar common diseases, the role that coexisting peripheral artery disease (PAD) may play, disease classification of venous insufficiency, and a general approach to therapy.

The most common form of lower extremity vascular disease, CVI affects 6 to 7 million people in the United States. Incidence increases with age and other risk factors. One study of 600 patients with CVI ulcers revealed that 50% had these ulcers for 7 to 9 months, 8% to 34% had them for more than 5 years, and 75% had recurrent ulcers.

Thrombotic complications of CVI include thrombophlebitis, which may range from superficial to extensive. If the thrombophlebitis extends up toward the common femoral vein leaving the leg, proximal ligation may be needed to prevent clot extension or embolization.



Understanding normal anatomy and physiology

Lower extremity veins flow horizontally from the superficial veins to the perforating veins and then into the deep veins. Normally, overall venous blood flows vertically against gravity from the foot and ankle upward toward the inferior vena cava (IVC). This antigravity flow toward the IVC results from muscular contraction around nonobstructed veins and one-way valves that close as blood passes them. These valves prevent abnormal backward blood flow toward the foot and ankle region.

The lower extremities have four types of veins. *Superficial veins* are located within the subcutaneous tissue between the dermis and muscular fascia. Examples are the greater and lesser (smaller) saphenous veins. *Perforating veins* connect the superficial veins to the deep veins of the leg.

The *deep veins* are located below the muscular fascia. The *communicating veins* connect veins within the same system.

The *greater saphenous vein* is on the leg's medial (inner) side. It originates from the dorsal veins on top of the foot and eventually drains into the common femoral vein in the groin region. By way of perforating veins, the greater saphenous vein drains into the deep venous system of both the calf and thigh.

The *lesser saphenous vein* is situated on the lateral (outer) side of the leg and originates from the lateral foot veins. As it as-

Abnormally elevated venous pressure stems from the leg's inability to adequately drain blood from the leg.

cends, it drains into the deep system at the popliteal vein behind the knee. Communicating veins connect the greater saphenous vein medially and the lesser saphenous vein laterally.

Intramuscular veins are the deep veins within the muscle itself, while the *intermuscular veins* are located between the muscle groups. The intermuscular veins are more important than other veins in development of chronic venous disease. Below the knee, the intermuscular veins are paired and take on the name of the artery they accompany—for example, paired anterior tibial, paired posterior tibial, and paired peroneal veins. Eventually, these veins form the popliteal vein behind the knee, which ultimately drains into the femoral vein of the groin.

As the *common femoral vein* travels below the inguinal ligament of the groin, it's

called the external iliac vein. Eventually, it becomes the common iliac vein, which drains directly into the IVC.

Pathophysiology

Abnormally elevated venous pressure stems from the leg's inability to adequately drain blood from the leg toward the heart. Blood drainage from the leg requires the muscular pumping action of the leg onto the veins, which pump blood from the leg toward the heart as well as from the superficial veins toward the deep veins. Functioning one-way valves within the veins close when blood passes them, preventing blood from flowing backward toward the ankle. This process resembles what happens when you climb a ladder with intact rungs: As you step up from one rung to the next, you're able to ascend.

CVI and the "broken rung" analogy

If the one-way valves are damaged or incompetent, the "broken rung" situation occurs. Think how hard it would be to climb a ladder with broken rungs: You might be able to ascend the ladder, but probably you would fall downward off the ladder due to the defective, broken rungs.

Normally, one-way valves ensure that blood flows from the lower leg toward the IVC and that the superficial venous system flows toward the deep venous system. The venous system must be patent (open) so blood flowing from the leg can flow upward toward the IVC. Blockage of a vein may result from an acute thrombosis (clot) in the superficial or deep systems. With time, blood may be rerouted around an obstructed vein. If the acute thrombosis involves one or more of the one-way valves, as the obstructing thrombosis opens up within the vein's lumen, permanent valvular damage may occur, leading to post-thrombotic syndrome—a form of CVI.

CVI may result from an abnormality of any or all of the processes needed to drain blood from the leg—poor pumping action

of the leg muscles, damage to the one-way valves, and blockage in the venous system. CVI commonly causes venous hypertension due to reversal of blood flow in the leg. Such abnormal flow may cause one or more of the following local effects:

- leg swelling
- tissue anoxia, inflammation, or necrosis
- subcutaneous fibrosis
- compromised flow of venous blood or lymphatic fluid from the extremity.

“Water balloon” analogy

The effect of elevated venous pressure or hypertension is worst at the lowest gravitational point (around the ankle). Pooling of blood and intravascular fluid around the ankle causes a “water balloon” effect. A balloon inflated with water has a thin, easily traumatized wall. When it bursts, a large volume of fluid drains out. Due to its thicker wall, a collapsed balloon that contains less fluid is more difficult to break than one distended with water.

In a leg with CVI, subcutaneous fluid that builds up requires a weaker force to break the skin and ulcerate than does a nondistended leg with less fluid. This principle is the basis for compression therapy in treating and preventing CVI ulcers.

Effects of elevated venous pressure or hypertension

Increased pressure in the venous system causes:

- abnormally high pressure in the superficial veins—60 to 90 mm Hg, compared to the normal pressure of 20 to 30 mm Hg
- dilation and distortion of leg veins, because blood refluxes abnormally away from the heart and toward the lower leg and may move from the deep venous system into the superficial veins.

Abnormal vein swelling from elevated pressure in itself may impair an already abnormally functioning one-way valve. For

instance, the valve may become more displaced due to the increase in intraluminal fluid, which may in turn worsen hypertension and cause an increase in leg swelling. Increased pressure from swollen veins also may dilate the capillary beds that drain into the veins; this may cause leakage of fluid and red blood cells from capillaries into the interstitial space, exacerbating leg swelling. Also, increased venous pressure may cause fibrinogen to leak from the intravascular plasma into the interstitial space. This leakage may create a fibrin cuff around the capillary bed, which may decrease the amount of oxygen entering the epidermis, increase tissue hypoxia, trigger leukocyte activation, increase capillary permeability, and cause local inflammation. These changes may lead to ulceration, lipodermatosclerosis, or both.

Visible changes may include dilated superficial veins, hemosiderin staining due to blood leakage from the venous tree, atrophie blanche, and lipodermatosclerosis. (See *CVI glossary*.) Both atrophie blanche and lipodermatosclerosis result from local tissue scarring secondary to an inflammatory reaction of the leg distended with fluid.

Lipodermatosclerosis refers to scarring of subcutaneous tissue in severe venous insufficiency. Induration is associated with inflammation, which can cause the skin to bind to the subcutaneous tissue, causing narrowing of leg circumference. Lymphatic flow from the leg also may become compromised and inhibited in severe venous hypertension, causing additional leg swelling.

Patient history

In a patient with known or suspected CVI, a thorough history may lead to a working diagnosis. Be sure to ask the patient these questions:

- Do you have pain?
- Is your pain worse toward the end of the day?
- Is the pain relieved with leg elevation at night?

CVI glossary

Atrophie blanche: atrophic (thin) skin with ivory-white, depressed, discolored tissue with red dots of the dermal capillaries, typically seen around the ankles. It may arise within the fibrotic area of lipodermatosclerosis and doesn't represent a healed ulcer.

Claudication: cramping leg pain with exercise, such as walking, from ischemia of the leg muscle groups. It results from proximal narrowing of the arterial tree associated with peripheral arterial disease (PAD). Exercising muscles need more oxygen than resting muscles. In persons with PAD, these muscles don't get the extra oxygenated blood they need, leading to tissue ischemia and pain. Claudication resolves with rest.

Communicating veins: veins that connect to other veins within the same anatomic plane; for example, a vein that connects the superficial greater saphenous vein medially to the superficial lesser saphenous vein laterally.

Hemosiderin staining: permanent brown staining of the skin caused by death of leaked red blood cells (RBCs) from an incompetent and hypertensive venous tree. Hemoglobin from the RBCs is converted to the permanent pigment hemosiderin.

Lipodermatosclerosis: change in the skin of the lower legs, resembling an inverted bowling pin or champagne bottle, common in patients with chronic venous insufficiency (CVI);

a constricted area of adherent fibrotic subcutaneous tissue and skin. More proximal to this narrowing is a swollen area of the leg caused by CVI and edema.

Perforating veins: veins that extend from the superficial venous system, passing through the muscular fascia, and linking to the deep venous system; may be above or below the knee.

Reticular veins: bluish, dilated subdermal veins 1 to 3 mm in diameter.

Telangiectasia: dilated intradermal veins less than 1 mm in diameter.

Varicose veins: dilated, tortuous subcutaneous veins of 3 mm or more in diameter.

- Is it relieved with leg elevation during the day?
- Do you have leg pain that awakens you at night?
- How would you describe the pain?
- Does the skin on your leg feel tight or irritated?
- Have you noticed visible changes of your leg?
- Do you have a leg ulcer?
- tired, "heavy" legs that feel worse toward the end of the day
- discomfort that worsens on standing
- legs that feel best in the morning after sleeping or after the legs have been elevated during the day.

Also determine if the patient has comorbidities that may exacerbate CVI, including PAD, renal failure, venous thrombosis, lymphedema, diabetes mellitus, heart failure, or malnutrition. (See *CVI risk factors*.)

Common CVI symptoms

Approximately 20% of CVI patients have symptoms of the disease without physical findings. These symptoms may include:

Although patients may report leg discomfort, the history indicates that it doesn't awaken them at night. Be aware that discomfort from CVI differs from that caused by PAD. With PAD, patients may report pain on exercise (claudication), pain with elevation (nocturnal pain), or constant pain (resting pain).

Signs of CVI (with or without ulcers) include:

- leg swelling (seen in 25% to 75% of patients)
- skin changes (such as hemosiderin staining or dermatitis)
- telangiectasia, reticular veins, or both;

while these are the most common signs, they represent an overall less severe finding

- varicose veins with or without bleeding, occurring in one-third of patients with CVI.

Venous ulcers

Venous ulcers are the most common type of lower extremity ulcer. They're commonly found on the medial aspect of the lower extremity, from the ankle to the more proximal calf area. Usually, they arise along the course of the greater saphenous vein, but also may be lateral and may occur at multiple locations. They aren't found above the knee or on the forefoot. Venous ulcers are shallower than arterial ulcers and have considerable exudate consistent with drainage from a ruptured water balloon. They may extend completely around the leg.

CVI: From a heavy sensation to visible changes

In patients with CVI, blood flows within a lower extremity in an abnormal, reverse direction, causing build-up of blood and intravascular fluid around the ankle. Initially, this may cause only a sensation of heavy legs toward the end of the day, with no visible changes. Eventually, it may lead to venous ulcers or other visible changes. This abnormal blood flow results from dysfunction of the normal mechanisms that drain blood from the leg against gravity into the IVC. ■

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CVI risk factors

Risk factors for chronic venous insufficiency (CVI) include:

- increasing age
- family history of venous disease
- history of localized trauma to the extremity, including saphenous-vein removal during cardiopulmonary bypass surgery or peripheral vascular bypass surgery
- localized radiation therapy
- previous venous thrombotic episode
- surgical lower-extremity arteriovenous shunt for renal dialysis
- repetitive prolonged standing
- overweight
- cigarette smoking
- sedentary life style
- pregnancy.

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