6 Venous Disorders

6.1 SUPERFICIAL VEIN THROMBOSIS

DEFINITION

Superficial vein thrombosis (SVT) is defined as thrombosis and inflammation of inner walls of the greater or lesser saphenous veins or their tributaries.

ETIOLOGY

- Trauma to the vessel wall—a drip needle or pressure externally due to tight garments or position of a limb.
- Circulating toxins from septic wounds.
- In association with deep venous thrombosis (DVT).
- It is associated with intravenous catheters and infusions.
- Occurs with varicose veins.
- Migrating SVT is often a marker for a carcinoma.
- May also occur in patients with vasculitides, such as thromboangiitis obliterans (TAO).

PATHOLOGY

Irritation produced changes in the tunica intima causing a thrombus to form. The thrombus becomes attached to the vein wall and rarely produces an embolus.

CLINICAL FEATURES

It can easily be distinguished from those of DVT. Patients complain of pain localized to the site of the thrombus. Examination reveals a reddened, warm and tender cord extending along a superficial vein. The surrounding area may be red and edematous. As the condition resolves, the skin become pigmented (brown) along the course of the vein.

INVESTIGATIONS

Phlebography is used to find out the thrombosis.

TREATMENT

Treatment is primarily supportive:

- Initially, patients can be placed at bedrest with leg elevation.
- Application of compression bandage in the form of crepe bandage or stockings from the toes to beyond the upper limit of the affected area.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) may be provided to relieve pain and inflammation.
- Antibiotics in case of infective phlebitis.
- *Anticoagulant therapy*: If a thrombosis of the greater saphenous vein develops in the thigh and extends toward the saphenofemoral vein junction and to prevent extension of the thrombus into the deep system and a possible pulmonary embolism.

6.2 DEEP VENOUS THROMBOSIS

DEFINITION

The presence of thrombus within a deep vein and the accompanying inflammatory response in the vessel wall is termed as *deep venous thrombosis* (DVT) or *thrombophlebitis*.

INCIDENCE

Deep vein thrombosis occurs less frequently in the upper extremity than in the lower extremity, but the incidence is increasing, because of greater utilization of indwelling central venous catheters.

ETIOLOGY

The factors that predispose to venous thrombosis were initially described by Virchow in 1856 and include stasis, vascular damage and hypercoagulability.

RISK FACTORS

- Recent surgery
- Neoplasms
- Trauma
- Fractures
- Immobilization
- Acute myocardial infarction (MI), congestive heart failure (CHF), stroke

- Postoperative convalescence
- Pregnancy
- Estrogen use (for replacement or contraception)
- Hypercoagulable states
- Previous DVT.

PATHOGENESIS

Damage to the intima causes platelets to be deposited on the vein wall. Venous stasis increases the accumulation of platelets, which adds to the size of the thrombus resulting in occlusion of the vessel lumen. Initially, the thrombus is composed principally of platelets and fibrin. Red cells become interspersed with fibrin and the thrombus tends to propagate in the direction of blood flow. The inflammatory response in the vessel wall may be minimal or characterized by granulocyte infiltration, loss of endothelium and edema. There is further extension of the thrombus (propagated thrombus) along the vessel to the next junction with a vein. A portion may break off giving rise to a pulmonary embolus or the thrombus may become organized and firmly attached to the vessel wall. Gradually, it is recanalized and circulation is re-established, but the valves are often destroyed leaving chronic venous insufficiency (CVI).

CLINICAL FEATURES

- Most common complaint is cramp-like pain in the calf.
- Unilateral leg swelling (edema around the joint distal to the area).
- Local warmth.
- Erythema.
- Tenderness may be present along the course of the involved veins.
- Palpable cord.
- Increased tissue turgor.
- Distention of superficial veins.
- Appearance of prominent venous collaterals.
- In some patients, deoxygenated hemoglobin in stagnant veins impart a cyanotic hue to the limb, a condition called *phlegmasia cerulea dolens*.
- In markedly edematous legs, the interstitial tissue pressure may exceed the capillary perfusion pressure, causing pallor, a condition designated as *phlegmasia alba dolens*.
- Unexplained systemic features, e.g. mild pyrexia, pleuritic pain, tachycardia in a patient recovering from surgery.
- Severe pulmonary embolus giving signs of extreme distress, breathlessness and shock may be the first indication of DVT.

Examination may reveal posterior calf tenderness, warmth, increased tissue turgor or modest swelling and rarely a cord. Cuff test, Homan's sign and Mose's sign will be positive.

INVESTIGATIONS

- D-Dimer, a degradation product of cross-linked fibrin is often elevated in patients with venous thrombosis.

- *Duplex venous ultrasonography:* By imaging the deep veins, thrombus can be detected either by direct visualization or by inference when the vein does not collapse on compressive maneuvers.
- The Doppler ultrasound measures the velocity of blood flow in veins. This velocity is normally affected by respiration and by manual compression of the foot or calf. Flow abnormalities occur when deep venous obstruction is present.
- *Magnetic resonance imaging (MRI):* It is useful in patients with suspected thrombosis of the superior and inferior vena cava or pelvic veins.
- *Venography*: Contrast medium is injected into a superficial vein of the foot and directed to the deep system by the application of tourniquets. The presence of a filling defect or absence of filling of the deep veins is required to make the diagnosis.

DIFFERENTIAL DIAGNOSIS

Deep vein thrombosis must be differentiated from a variety of disorders that cause unilateral leg pain or swelling, including muscle rupture, trauma or hemorrhage; a ruptured popliteal cyst and lymphedema. It may be difficult to distinguish swelling caused by the postphlebitic syndrome from that due to acute recurrent DVT. Leg pain may also result from nerve compression, arthritis, tendinitis, fractures and arterial occlusive disorders.

TREATMENT

- Bedrest with a cradle and the end of the bed elevated (15–22 cm) until all the local signs subside may be up to 7 days.
- *Anticoagulants prevent thrombus propagation and allow the endogenous lytic system to operate:*
  - This includes either unfractionated heparin or low-molecular–weight heparin (LMWH).
  - A direct thrombin inhibitor, such as lepirudin or argatroban may be used as initial anticoagulant therapy for patients in whom heparin is contraindicated, because of heparin-induced thrombocytopenia (HIT).
  - Warfarin is administered during the first week of treatment with heparin and may be started as early as the first day of heparin treatment.
- *Thrombolytics*: Thrombolytic drugs such as streptokinase, urokinase and tissue plasminogen activator (tpA) may also be used.
- *Vena cava filter* (*Greenfield filter*): To prevent pulmonary embolism.

PROPHYLAXIS

Prophylaxis should be considered in clinical situations where the risk of DVT is high.

- Low-dose unfractionated heparin (5,000 units 2 h prior to surgery and then 5,000 units every 8–12 h postoperatively).
- Warfarin.

- External pneumatic compression (TED antiembolic stockings) applied to the legs are used to prevent DVT.

COMPLICATIONS

- Pulmonary embolism
- Chronic venous insufficiency.

6.3 VARICOSE VEINS

DEFINITION

Varicose veins are dilated, tortuous superficial veins that result from defective structure and function of the valves of the saphenous veins.

INCIDENCE

- The most common in 40–50 years
- Females are more affected than males.

TYPES

Varicose veins can be categorized as primary or secondary. Primary varicose veins originate in the superficial system and occur two to three times as frequently in women as in men. Approximately half of patients have a family history of varicose veins. Secondary varicose veins result from deep venous insufficiency and incompetent perforating veins or from deep venous occlusion causing enlargement of superficial veins that are serving as collaterals.

ETIOLOGY

- Failure of development of valve in the vein
- Damage to the valve due to thrombosis
- From intrinsic weakness of the vein wall
- From high intraluminal pressure
- Rarely from arteriovenous fistulas.

PREDISPOSING FACTORS

- *Pregnancy*:
  - Compression of pelvic vein due to enlarged womb **(Fig. 6.3.1)**
  - Estrogens relax the muscles in the veins and this also increases the tendency of the veins to expand.
- Occupation necessitating constant standing, e.g. shop assistant, traffic police, teachers, etc.

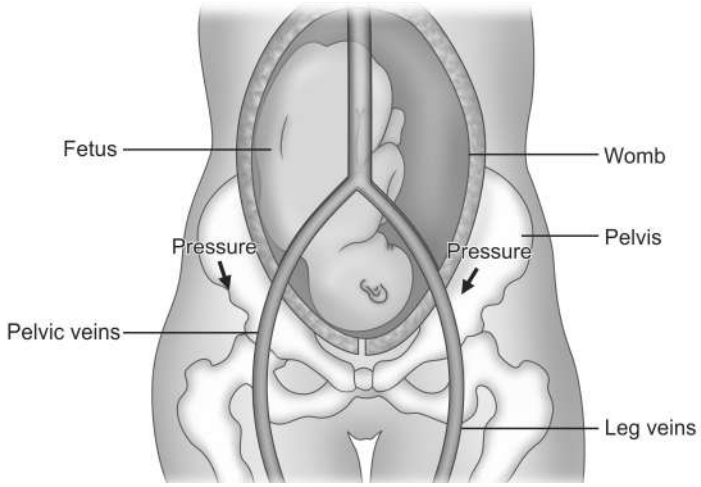


Fig. 6.3.1 Uterus pressing the pelvic veins

- Low-fiber diet, which results in constipation. If the bowel is loaded with constipated stool it compresses the large (iliac) veins in the pelvis, which in turn dilates the veins of the leg.
- *Constipation:* Staining during constipation builds up pressure and transmits the pressure to the legs. This increased pressure build-up in the leg veins over the years will lead to a deterioration of the valves and hence, will lead to varicose veins.
- Tight corsets or garters.
- Contraceptive pills which have estrogen.
- Family history.
- Secondary to DVT.

PATHOLOGY (FIG. 6.3.2)

The vein wall dilates at weak areas and the valves become incompetent. Normally as the calf muscle contracts, there is pressure on deep veins which forces the blood proximally. This pressure is not transmitted to the superficial veins, because of valves in communicating veins. When these valves become incompetent the pressure pushes the blood into a superficial vein, which dilates and lengthens. A vicious cycle is set up, the ineffectual valves permitting regurgitation and the increasing amount of blood thus left in the veins still further dilating them and making the valves more incompetent.

During standing, the force of gravity tends to keep the blood in lower parts of the body, aggravating the condition. There is loss of elastic tissue, muscle atrophy of the media layer and hypertrophy of the outer layer. Dilated veins and abnormally high pressure in the capillaries increases exudation of lymph, which results in edema.

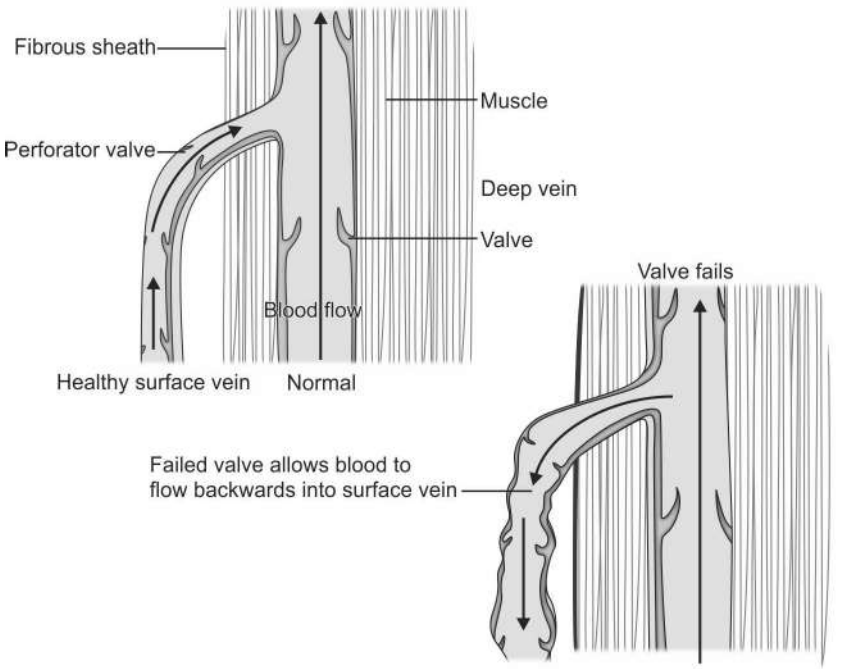


Fig. 6.3.2 Varicose vein caused by valve failure

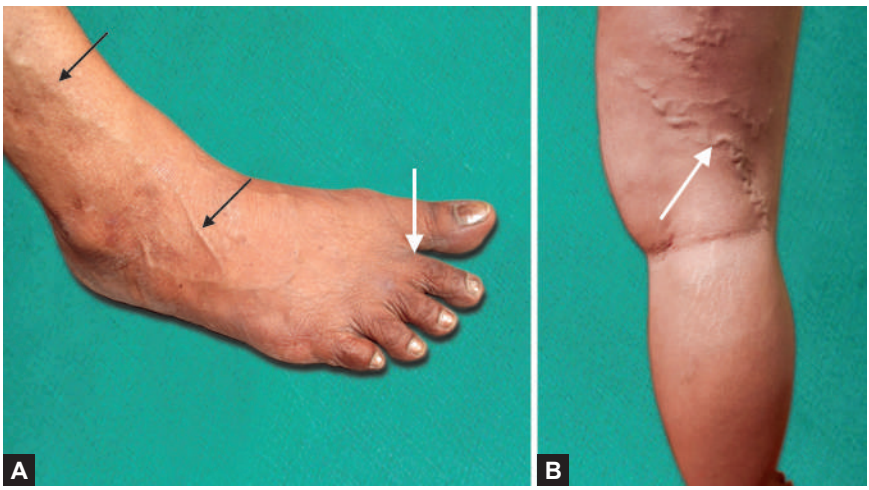
CLINICAL FEATURES

- Patients with venous varicosities are often concerned about the cosmetic appearance of their legs.
- Patient may complain of a dull ache, pain or pressure sensation in the legs after prolonged standing; it is relieved with leg elevation.
- Fatigue in the legs with difficulty in walking.
- Cramp in the calf muscles, especially at night.
- The legs feel heavy and mild ankle edema develops occasionally.
- The skin of the leg may become pigmented and indurated **(Figs 6.3.3A and B)**.
- Extensive venous varicosities may cause skin ulcerations near the ankle.
- Superficial venous thrombosis may be a recurring problem.
- Rarely, a varicosity ruptures and bleeds.

Visual inspection of the legs in the dependent position usually confirms the presence of varicose veins.

INVESTIGATIONS

- *Doppler ultrasound scan:* It can easily identify reflux or back-flow of blood in the veins.
- *Varicogram*: A cuff will be placed around the lower calf and the dye injected into the veins on the back of the foot. Usually, the site of connection of the varicose vein to the deep venous system need to be identified and the



Figs 6.3.3A and B (A) Varicose vein on female's leg, black arrows: dilated vein; white arrow: skin changes; (B) Varicose vein on female's thigh, white arrow: spider vein

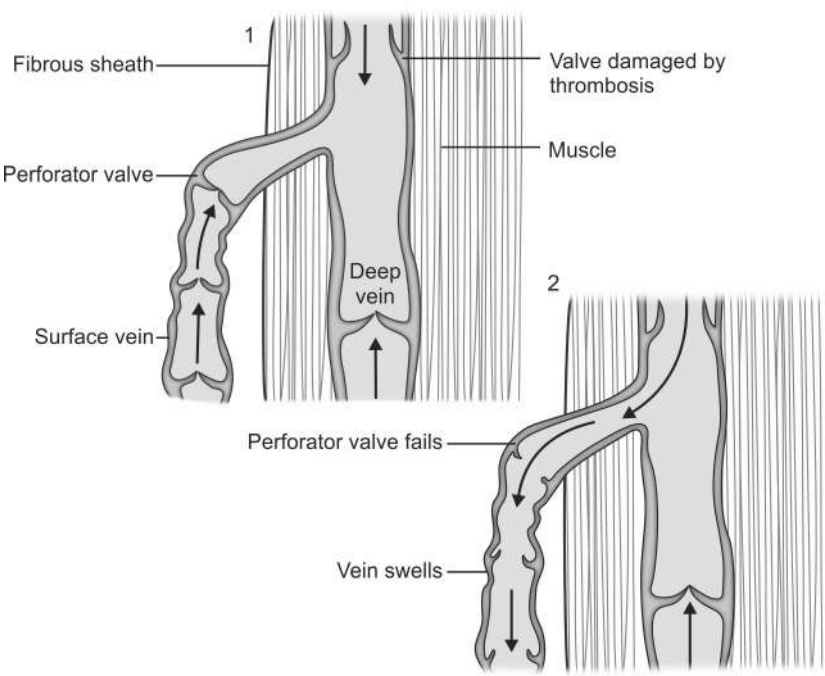


Fig. 6.3.4 Varicose veins caused by deep vein damage

needle is then placed within the varicosity. If the test is done to look at whether the valves in the leg are working, the needle may be placed either in the foot or in the groin, and the table will be tilted to see if the dye passes backwards through the valves **(Fig. 6.3.4)**.

CONSERVATIVE TREATMENT

- External compression stockings has to be worn which provide a counter-balance to the hydrostatic pressure in the veins.
- Walking is encouraged.
- Avoid prolonged standing.
- Symptoms often decrease when the legs are elevated periodically.
- *Laser therapy*: The laser pulse is of a very short duration and destroys the veins, which are broken into very small particles. These are then removed by the body's immune system. There may be some reaction to the laser for the first 24 hours, with redness at the site, but this resolves quite quickly. This treatment is given on an out-patient basis and does not require any anesthesia. The complications of this therapy include pigmentation at the site and loss of pigment at the site. There is usually loss of hair growth in the area if the laser treatment is repeated. Very occasionally, there may be some crusting of the skin together with blistering. But these side-effects do not last long.
- *Endovenous obliteration of the saphenous vein*: A newer treatment for varicose veins is to insert a long, thin catheter that emits energy (most commonly heat, radiowaves or laser energy). The released energy collapses and scleroses the vein. A variety of techniques and protocols are used. Because it is easier to insert a catheter through a vein in the same direction that the valves open, the catheter is most commonly inserted into a more distal portion of the vein and threaded proximally. Energy is released from the catheter tip. As the catheter is pulled out, the vein lumen collapses. Bruising, tightness along the course of the treated vein, recanalization and paresthesia are possible complications.

SURGICAL MANAGEMENT

Surgical therapy may also be indicated for cosmetic reasons:

- *Sclerotherapy*: It is a procedure in which a sclerosing solution is injected into the involved varicose vein and a compression bandage is applied. The sclerosant solution produces inflammation in the vein so that no blood can pass through causing the lumen to be obliterated.
- Surgical therapy usually involves extensive ligation and stripping of the greater and lesser saphenous veins and should be reserved for patients, who are very symptomatic, suffer recurrent superficial vein thrombosis, and/or develop skin ulceration.

COMPLICATIONS

- *Hemorrhage*: Bleeding following rupture of a vein.
- *Ulceration*: Venous ulcer due to devitalized skin.
- *Phlebitis*: Superficial venous thrombosis.
- Edema particularly of the foot and ankle.
- Pigmentation.

6.4 PULMONARY EMBOLISM

DEFINITION

The term 'pulmonary embolism' implies clinically significant obstruction of a part or the whole of the pulmonary arterial tree, usually by thrombus that becomes detached from its site of formation outside the lung and is swept downstream until arrested at points of intrapulmonary vascular narrowing.

TYPES

- Thrombotic pulmonary embolism
- Nonthrombotic pulmonary embolism.

Thrombotic Pulmonary Embolism

As a complication of deep venous thrombosis: If a thrombosis breaks off in a deep vein it travels in the venous system to the right side of the heart, where it enters the pulmonary artery and passes into the pulmonary circulation where it blocks a vessel, the lumen of which is too narrow to let it pass through. The factors that predispose to a deep vein thrombosis also predispose to a pulmonary embolism. Stasis being the most important initiating factor in initiating and venous thrombi rather than any damage to vessel wall.

Other: A small proportion of pulmonary emboli may arise from the pelvic veins including the prostatic venous plexus in men and also from the right side of the heart (following myocardial infarction and in right ventricular failure).

Septic pulmonary emboli may arise from bacterial endocarditis in patients with septal defects, from the tricuspid valve in drug abusers, and from foreign material such as central venous lines, ventriculoatrial and arteriovenous shunts, internal cardiac defibrillators and pacemaker wires.

Nonthrombotic Pulmonary Embolism

Fat embolism: It is probably a common subclinical event following bony trauma. Following such trauma, neutral fat may pass into the circulation from injured long bones, be carried to the lungs and become lodged in the pulmonary vasculature.

Tumor emboli: Pulmonary vessels are occluded by macroscopic emboli, but it differs in its lack of responsiveness to anticoagulation. Tumors that have been implicated most frequently include carcinoma of the breast, stomach, colon and cervix, hepatomas, choriocarcinomas and hypernephromas.

Air embolism: It may be the result of faulty cannulation of the neck veins, therapeutic insufflation of air into a fallopian tube or intrauterine manipulations, including criminal abortion in which a frothy solution may be introduced into the uterus under pressure. Small amount of air are reabsorbed without harm,