Review Article,

**Obesity and Venous Disorders-An Overview**

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**Abstract:**

The prevalence of obesity, particularly central obesity, is increasing globally. It has been identified as a risk factor for not only cardiovascular diseases but also venous disorders. This brief review attempts to revisit the pathophysiology, classification and management of venous disorders in the context of central adiposity.

**Keywords**- central adiposity, intra-abdominal pressure, coagulation, venous stasis

**Introduction:**

As per the ICMR-INDIAB study, the prevalence of central obesity lies between 16.9%–36.3%.¹ It is not only associated with elevated risk for cardiovascular disease (CVDs) but also predisposes individuals to venous stasis, which may trigger deep vein thrombosis and chronic venous insufficiency.²,³ Obesity increases the risk of venous insufficiency and associated complications by more than 6-fold.⁴

Excessive body weight puts an additional load on the circulatory system because the excess body mass implies our body must work harder to distribute, deliver and return blood to the cardiac pump. Impaired return of the blood increases the pressure in the lower extremity veins. Venous hypertension due to higher reflux or obstruction is a vital cause of chronic venous insufficiency. The increased pressure in the veins may lead to leaky valves causing varicose veins.

Increased body weight is also related to changes in the coagulation system, including fibrinolytic activity and increased plasma concentration of coagulation factors.

**Pathophysiology**

The complication of chronic venous insufficiency hinders the person’s ability to involve in routine occupation or social activities and also puts a financial burden on the patient and family due to loss of duty hours.⁵ As per Virchow’s triad of hypercoagulability, stasis and injury to veins, factors contributing to deep vein thrombosis formation lead to chronic venous insufficiency.⁶

Chronic venous insufficiency can lead to difficult-to-heal and recurrent venous ulcers. Danielsson et al. found a significant association between a BMI of 28.9kg/m² or more and higher clinical scores of chronic venous disease.⁷ The study by Kruger et al. showed a strong relationship between the cross-sectional areas of lower extremity veins and increasing BMI.⁸ Pathological changes due to hyperglycemia may be etiologically linked to vascular dysfunction in patients with diabetes.⁹ Local angiogenesis and reduced peripheral blood flow are some known vascular complications.

Several epidemiological studies strongly support the hypothesis that obesity is a risk factor for chronic venous insufficiency.¹⁰ Obesity is also related to changes in the coagulation parameters, including altered fibrinolytic activity and increased concentration of clotting factors.¹¹ These changes in endothelial function and coagulation are thought to be important not only for arterial but also for venous thrombosis.¹²

Besides, obesity is considered to cause venous stasis, which may trigger chronic venous insufficiency and deep vein thrombosis.
Arvfiddson et al. demonstrated that in morbidly obese patients, the pressure in the iliofemoral vein was significantly more than in non-obese individuals. The increased intra-abdominal pressure is thought to be transmitted to the extremities by the femoral veins causing venous stasis and distension of the veins in the lower extremities favouring venous valve dysfunction and thrombosis.

A waist circumference of >100cm may increase the risk of venous thromboembolism by 4 times. Similarly, post-thrombotic syndrome occurs more in obese patients after venous thromboembolism. Increased body weight is a risk factor for not only the first venous thromboembolism event but also for recurrent venous thromboembolism events. The prevalence of iliac vein compression is more in women than in men.

**Types of Venous Disorders:**

**Blood clots:** These can occur on legs, arms, kidneys (renal vein thrombosis), brain (cerebral vein thrombosis), lungs (pulmonary embolism) or other internal organs like the liver or pelvic organs.

**Deep Vein Thrombosis:** The blood clots in the deep veins of legs and arms may cause inflammation. It may be initially asymptomatic but may lead to a dangerous pulmonary embolism.

**Superficial thrombophlebitis:** The blood clots in a vein close to the skin’s surface cause inflammation and pain, but they usually do not lead to pulmonary embolism unless they move first to the deep venous system.

**Chronic venous insufficiency:** The condition causes blood stagnation, chronic leg swelling, increased pressure, increased pigmentation or discoloration of the skin, or even leg ulcers called venous stasis ulcers.

**Venous ulcers:** Ulcers are wounds or open sores that don’t heal or keep coming repeatedly. Venous stasis ulcers usually occur below the knee, on the inner aspect of the leg or just above the ankle. Venous leg ulcers are present in 1% of adults.

**Arteriovenous fistulas** are arteries and veins that connect directly with nothing in between.
Varicose veins are twisted, swollen veins near the skin's surface and formed when weak or defective valves allow blood to flow backwards, which stagnates in the vein. Chronic venous obstruction can also cause varicose veins. Women are affected twice as men. Almost 33% of adults have varicose veins.

Tests to diagnose venous disorders
- Ankle-brachial index
- Ultrasound

Chronic venous disease severity is generally evaluated using the CEAP (Clinical, Etiologic, Anatomic, Pathophysiologic) classification system, with a predominant focus on the clinical element. 18 Table 1

<table>
<thead>
<tr>
<th>CLINICAL STAGE</th>
<th>KEY CHARACTERISTICS</th>
<th>AETIOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>C0</td>
<td>C0: no visible or palpable signs of venous disease</td>
<td>Congenital (Ec)</td>
</tr>
<tr>
<td>C1</td>
<td>Telangiectasias or reticular veins</td>
<td>Primary (Ep)</td>
</tr>
<tr>
<td>C2</td>
<td>Varicose veins</td>
<td>Secondary (Es)</td>
</tr>
<tr>
<td>C3</td>
<td>Edema</td>
<td>None identified (En)</td>
</tr>
<tr>
<td>C4a</td>
<td>Pigmentation or eczema</td>
<td></td>
</tr>
<tr>
<td>C4b</td>
<td>Lipodermatosclerosis (venous stasis results in fat necrosis due to ischemia), or sclerosing panniculitis and hypodermitis sclerodermiformis</td>
<td></td>
</tr>
<tr>
<td>C5</td>
<td>Healed venous ulcer</td>
<td></td>
</tr>
<tr>
<td>C6</td>
<td>Active venous ulcer</td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>Symptomatic, ache, pain, tightness, skin irritation, heaviness, muscle cramps, and other complaints due to venous dysfunction</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>Asymptomatic</td>
<td></td>
</tr>
</tbody>
</table>

Anatomical Pathophysiology
- Superficial (As) | Reflux (Pr)
- Perforator (Ap) | Obstruction (Po)
- Deep (Ad) | Reflux and obstruction (Pr,o)
- No location detected (An) | No pathophysiology identified (Pno)

As: Superficial veins Telangiectasies or reticular veins, Great saphenous vein above the knee, Great saphenous vein below the knee, Small saphenous vein, Non-saphenous veins, Ap perforating veins: Thigh Calf Ad: Deep veins Inferior vena cava, Common iliac vein, Internal iliac vein, External iliac vein Pelvic veins, Common femoral vein, Deep femoral vein, Femoral vein, Popliteal vein, Crural veins (all paired) Muscular veins;

The patient evaluation includes a physical examination, comorbidity conditions, relevant medical history, and clinical manifestations. Noninvasive and invasive tests include Doppler ultrasonography and thorough imaging (ascending and descending venography, venous pressure measurements, computed tomography, venous helical scanning, or magnetic resonance imaging), which may be used alone or in combination, depending on the severity of the venous disease.18

Applying varying positions and levels of exogenous compression during imaging facilitates the display of obscure venous reflux regions.

Management:
Lifestyle: It is most important to adopt a healthy lifestyle regularly. Caloric intake should be limited, and the diet should be low in simple carbohydrates and high in fibre. The diet should include foods rich in antioxidants and essential fatty acids, especially omega 3, to protect the wall of the veins. The use of tobacco should be stopped. Non-pharmacological interventions can significantly alter the blood flow, consequently inducing positive variations in the endothelium.19 Weight loss decreases venous pressure, and regular exercise increases the tone of the calf muscles to support the vein wall. Exercise also ensures energy expenditure and promotes weight loss. Walking, swimming, and cycling increases...
venous tone. Sports such as tennis and squash exert pressure by jerking and compressing the calf. Heat dilates the veins; therefore, one should avoid sunbathing, saunas and hot baths. However, water at a lower temperature provided in a direction from the ankles towards the calves up to the thighs may be helpful.

Prolonged standing should be avoided, and legs should be elevated at the end of the day. Compression stockings or bandages may be used. Massage may improve lymphatic drainage, which relieves the pain.

**Pharmacologic Therapy:** Antibiotics may be given to clear skin infections or ulcers. Pentoxifylline may improve blood flow. Anticoagulants or blood thinners may be used to treat or prevent clot development. Injection heparin may be used for 3 to 6 months. Occasionally thrombolytics such as urokinase or tissue plasminogen activators may be used.

**Endovenous laser ablation:** It is preferred in patients who do not respond to non-invasive therapy like compression stockings. Endovenous laser ablation is a minimally invasive procedure that applies laser heat to reduce varicose veins. A catheter placed in the vein provides heat and obliterates the vein. Thus, less blood pools in the leg and overall blood flow improves.

**Sclerotherapy:** In serious cases, a sclerosant is injected into the affected veins, which become sclerosed. Blood then flows back to the heart via the other veins. The scarred vein is resorbed.

**Vein Surgery:**

a) Ligation and stripping: The vein may be tied off (ligation), or if it or its valves are heavily damaged, it may be removed (stripping).

b) Microincision/ ambulatory phlebectomy: This is a minimally invasive procedure in which superficial varicose veins near skin surfaces are targeted by making punctures using needles or incisions. Subsequently, a phlebectomy hook is used to eliminate the affected veins.

c) Subfascial Endoscopic Perforation Surgery (SEPS): It is a minimally invasive procedure for targeting perforating veins above the ankle. A clip is used to block the veins, thus preventing blood flow. It helps to heal ulcers and prevent their recurrence.

**Vein Bypass:** The healthy veins from another part of the body create a bypass to re-route the blood from a damaged vein. This method is only used in severe cases when other treatment options fail.

**Conclusions**

Obesity is a well-established risk factor for the development and progression of chronic venous disorders. It has a relevant influence on venous hemodynamic parameters of the lower extremities. Abdominal adipose tissue has a mechanical role in the elevated risk of venous disorders.

**ACKNOWLEDGEMENTS** - Nil

**CONFLICTS OF INTEREST** - Nil

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**References:**


[9] Das Evcimen N, King GL. The role of protein kinase C activation and the vascular...


