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DEEP VEIN THROMBOSIS

Vascular Sciences

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ABSTRACT

Deep vein thrombosis (DVT) usually affects the deep vein of the legs, though it may also occur in the veins of the arms, mesenteric and cerebral veins. Venous thromboembolism can cause sudden pulmonary embolism with instantaneous death. In patients who have developed deep vein thrombosis there is likelihood of recurrent thrombosis and post thrombotic syndrome. Deep venous thrombosis is preventable in majority of the cases. Understanding the etiopathogenesis, clinical presentation, evaluation and management is essential for both prevention and management thereby reducing the morbidity and mortality associated with the disease.

KEYWORDS

Deep Vein Thrombosis, Prevention, Treatment.

INTRODUCTION:

DVT is more common in the lower extremity with the incidence of 40% in the distal veins, 16% in the popliteal vein,20% in the femoral vein and 4% in the iliac veins.[1] The most dreaded complication of lower extremity is pulmonary embolism.

DISCUSSION:

A variety of risk factor predispose to DVT. [1, 2]

1. Sluggish circulation:

This is seen in patients who are rendered immobile by bedrest, general anesthesia, operations, long haul flights and in bedridden patients due to stroke.

2. Venous hypertension:

This can be caused by either compression of the veins or functional impairment as seen in pelvic growths, pregnancy and congenital anomalies.

3. Damage to the veins as seen in trauma surgery, previous DVT and intravenous drug abuse.

4. Hyperviscosity of the blood seen in thrombocytosis, polycythemia and dehydration.

5. Genetic deficiency: Deficiency of protein C and S, Anti-Thrombin 3 Deficiency and Factor V Leiden mutation

Acquired causes: These include cancer, myocardial Infarction, heart failure, anticoagulant therapy, inflammatory bowel disease, nephrotic syndrome, estrogen therapy, smoking, diabetes, hypertension

6. Obesity: Leads to hypercoagulable state by 2 mechanism:

- Increased fibrinogen level
- Sluggish venous circulation in the infra-diaphragmatic region and lower limbs.[3]

All risk factor for DVT can be categorized as transient, persistent and unprovoked group. [4]

- Transient factors: These surgery, general anesthesia, prolong hospitalization, C-section, hormone replacement therapy, pregnant state and injury to the lower extremity. Any surgery under general anesthesia lasting more than 30 min and hospitalization longer than 72hrs is considered a very important transient risk factor in the surgical patients.
- Persistent risk factors include active cancer and specific medical condition like SLE and IBD.
- Unprovoked factors are those that cannot be classified under transient or persistent category such as altered lipid level, High triglyceride level, etc. Advance age is another risk factor for DVT.

According to Virchow's triad the main mechanisms involved in DVT are:

- Damage to vessel wall
- Turbulence of blood
- Hyper viscosity of blood.

Any of the factor from the Virchow's triad serves as a trigger point for venous thrombosis. Thrombus once formed reacts with the endothelium. This stimulates the release of cytokines and increased leukocyte adhesion to the endothelium thereby promoting further venous thrombosis. DVT is commonest in the lower extremity below the knee and usually sets in from the soleal sinuses. A strong correlation between atherosclerosis and DVT is observed attributable to endothelial dysfunction. [5]

Histologically there is formation of an extensive thrombus followed by remodeling of the thrombus. Neutrophil and macrophages infiltrate the fibrin clot within the lumen of the vessel leading to cytokine release. Subsequently fibroblast and collagen replace the fibrin. This is followed by remodeling. Fibrosis diminishes the blood flow even after acute thrombosis resolves. The natural fibrinolytic system causes disintegration of the clot in the central portion of the vein. This causes re-canalization of the central portion of the vein. However, the residual clot in the periphery of the vein continues to remain thereby fixing and rendering the walls functionless. [6] The end result of a vein affected by DVT is a valve less vein which causes incompetency of the perforators due to persistent high back-pressure leak as well as varicose vein. In a few patients it causes skin changes giving rise to a post phlebitic limb.[7]

Clinical Features:

DVT commonly affects the lower extremity. Pain, redness and swelling are the common features. Physical examination will reveal edema of extremity, increased local rise of temperature and severe tenderness. In advanced cases with severe venous hypertension venous gangrene can set in, which is described as Phlegmasia Cerulea Dolens. In addition to venous dysfunction and edema, the lymphatics may also be compressed giving rise to pale white limb described as phlegmasia alba dolens typically seen in pregnant state. [5]

Investigation:

Venous Doppler is an extremely important investigation and helps in establishing the diagnosis of DVT. This reveals the extent of the thrombosis with respect to extension of the thrombus up to the iliac veins. D-Dimer is a supportive investigation which is seen in patient suffering from DVT. [6]

Management:

Aims of treating DVT:

1. Prevention of pulmonary embolism
2. Prevention of propagation of the clot
3. Reduction in the incidence of post-phlebitic limb

Treatment options:

Once the diagnosis is confirmed immediate anticoagulation is

essential. The choice of anti-coagulation depends on pre-existing medical status of the patient.

- DVT associated with cancer is best treated with low molecular weight heparin (LMWH) and Factor Xa inhibitor (Rivaroxaban).
- In patients suffering from liver disease LMWH is preferred. [5]
- Oral anticoagulant is contraindicated in patients with renal disease. Vitamin K antagonist are recommended.
- In patients with previous history of coronary artery disease Vitamin K or rivaroxaban is preferred.
- In patients who have dyspepsia, as there is a chance of GI bleeding, Vitamin K antagonist are preferred. [7]

Duration of treatment:

- Initial 5 days with LMWH until the INR is greater than 2
- Vitamin K antagonist for 3 months. In case of unprovoked DVT vitamin K antagonist therapy beyond 3 months is advisable.
- Rivaroxaban is preferred as it does not require regular INR monitoring.
- Platelet counts have to be meticulous monitored .If less than 75000 then heparin is replaced by fondaparinux. [8]
- Supportive treatment includes elevation of limbs and elasto-crepe bandage support,
- Patient is followed up periodically with INR report if on Vitamin K antagonist, INR to be maintained above 2. After 3 months ECG, 2D-ECHO and chest X-Ray along with venous doppler of lower extremity is essential. If the veins have recanalized and there is no evidence of pulmonary hypertension then anti-coagulants can be stopped. However if recanalization is not yet completed then extension of anticoagulant is considered. [9]

IVC Filter:

Indication:

- Recurrent venous thrombo-embolism despite adequate anticoagulation.
- Venous thromboembolic disease with absolute contraindication to anti-coagulants.
- Complications leading to cessation of anticoagulant therapy.

Contraindications to IVC filters:

- Uncorrectable coagulopathy and bacteremia.

Complications include: bleeding, thrombosis and filter tilt. IVC thrombosis and renal failure are dreadful complication of IVC filters. Retrievable IVC filters are preferred over permanent IVC filters. Due to lack of prospective randomized studies the use of IVC filter continues to be debatable. IVC filter should be used for specific indication wherein anti-coagulant are either contraindicated or don't work. [10]

CONCLUSION:

DVT is a dreaded complication seen in hospitalized surgical patients. Identification of risk factors essential for prophylaxis. Clinical features are suggestive. However, venous doppler of lower extremity is diagnostic. Parenteral followed by oral anti-coagulant is preferred. Continuous monitoring for complications is necessary. IVC filter may be used in certain situations where in anti-coagulants do not work or are contraindicated in patients and in those patients developing complication.

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