# Thrombosis disorders and Deep Venous Thrombosis

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 Thrombosis is the development of a 'thrombus' consisting of platelets, fibrin, red cells and white cells in the arterial or venous circulation. If part of this thrombus in the venous circulation breaks off and enters the right heart, it may be lodged in the pulmonary arterial circulation, causing pulmonary embolism (PE).

 In the left-sided circulation, an embolus may result in peripheral arterial occlusion, either in the lower limbs or in the cerebral circulation (where it may cause thromboembolic stroke).

# A. Pulmonary embolism

 Pulmonary embolism is a potentially fatal complication of lower limb deep vein thrombosis. A clot from the lower limb veins becomes detached from its site of formation and passes via the inferior vena cava and right heart to the pulmonary arteries. Here it may totally occlude the perfusion to part or all of one or both lungs.

• This leads to collapse or sudden death in some patients and is a medical emergency. Treatment includes full, immediate anticoagulation with intravenous heparin combined with standard methods of resuscitation. Large emboli may be treated by infusion of fibrinolytic drugs into the pulmonary arteries via catheters inserted via arm or leg veins.

# **Prevention of pulmonary embolism**

 In some patients the risk of pulmonary embolism is very great, e.g. a large venous thrombosis in the lower limb where anticoagulation is contraindicated (e.g. following a hemorrhagic stroke). In others, pulmonary embolism occurs despite full anticoagulation with warfarin.  Pulmonary embolism may be prevented by the insertion of an inferior vena cava filter which traps large thrombi in its wires and prevents them from occluding the pulmonary arteries. These filters are usu-ally placed by a radiologist via the femoral or jugular vein under X-ray control without the need for an open surgical procedure

# **Venous thrombosis**

 Venous thrombosis of the deep veins is a serious life-threatening condition which may lead to sudden death in the short term or to long-term morbidity due to the development of a post-thrombotic limb and venous ulceration. All patients admitted to hospital or being treated for a serious illness should be assessed for the risk of deep vein thrombosis. These patients may he considered at low risk, moderate risk or high risk:

- low risk / young patients, minor illnesses, operations lasting for less than 30 minutes with no additional risk factors;
- moderate risk / patients over the age of 40 years with debilitating illnesses, undergoing major surgery but no additional risk factors;

 high risk / patients over the age of 40 years with serious medical conditions, such as stroke and myocardial infarction, and undergoing major surgery with additional risk factor, such as a past history of venous thromboembolism, extensive malignant disease or obesity.  Deep venous thrombosis (DVT) is a common cause of death. The true incidence of DVT difficult to determine because it clinical diagnosis can be inaccurate and often occurring in the setting of other critical illness. Approximately 20% of cases of calf DVT propagate to the thigh, and 50% of cases of thigh or proximal DVT embolize.

 In modern medical practice the most common cause of lower limb venous thrombosis is following hospital admission for treatment of medical or surgical conditions.





# **Risk factors for DVT**

- Endothelial injury by malignancy:
- Adhesion of tumor cells to endothelium .
- Chemotherapeutic drugs, such as bleomycin, carmustine, vincristine and doxorubicin can also cause vascular endothelial cell damage

- Venous stasis:
- This is caused by immobility, venous obstruction, increased venous pressure, and increased blood viscosity.
- Surgery and critical illness. E.g.:-Major chest surgery. Similarly, a prolonged nonambulatory state, such as fracture of the hip requiring bed rest can increase DVT risk.

- Drugs: Oral contraceptives (OCPs) and estrogen hormone replacement therapy:These have been linked to increased risk of venous thrombus formation.
- Hypercoagulable states.

#### **Clinical features**

DVT can be silent but typically symptoms and signs occur during the second postoperative week, although they may came earlier or later. The patient complains of pain in the calf, and on examination there is tenderness of the calf and swelling of the foot, often with edema, raised skin temp., and dilation of the superficial veins of the leg. This is accompanied by a mild pyrexia. If the pelvic veins or the femoral vein are affected, there is massive swelling of the whole lower limb.



# Investigations

- Venography: it is an invasive procedure.
- Duplex scanning: it can detect thrombi in all major veins at and above the knee. It is simple and noninvasive.
- Compression ultrasonogrphy: is highly sensitive in detecting thrombosis of the proximal veins but less sensitive in detecting calf vein thrombosis.

# Management

#### • A. Prophylaxis

- Treat avoidable risk factors.
- Active mobilization: stimulation of blood flow by encouraging early mobilization reduces the risks.
- Intermittent calf compression: using inflatable cushions wrapped around the lower legs may be used intraoperatively to reduce the incidence of thrombosis.
- Mechanical methods are effective (Graduated compression stockings TED) have been shown in clinical trials to reduce the incidence of deep vein thrombosis. Also elevation of the legs to increase venous return are simple and effective.

- Low-dose unfractionated heparin: this is given SC at 5000 units 2 hrs. before surgery every 8 or12 hrs. Postoperatively. It should not be used for patients undergoing cerebral, ocular or spinal surgery.
- Low- molecular weight heparins: such as enoxaparin.
- Newer medications: such as the direct thrombin inhibitors represent a possible alternative to the unfractionated and LMWHs in the prevention of thromboembolic disease.

#### **B. Treatment**

 The aim is to minimize the risk of pulmonary embolism and encourage the thrombus to resolve. Pharmacological methods evaluated include: low-dose heparin, low-molecularweight heparin, dextran and adjusted dose warfarin.  In treating an established deep vein thrombosis it is important to make the correct diagnosis. Twenty per cent of patients with clinical signs and symptoms of a deep vein thrombosis have normal deep veins. When the diagnosis of DVT is made postoperatively, begin full-dose heparinization (bolus of 5000-10000IU, followed by continuous infusion of 1000-1500IU/hr) with the dose adjusted according to the weight of the patient and controlled by the activated partial thromboplastin time (APTT), if surgical hemostasis is achieved, also most studies suggest that the use of 5000 units of heparin, given subcutaneously two or three times a day, is effective.

 This should be continued for at least 5 days. Once on therapeutic heparin (aPPT of 1.5-2), warfarin should be initiated and the dose adjusted to maintain an appropriate INR (ie, The INR should be prolonged to between 2.5 and 3.5 times the control value.). Heparin and a therapeutic level of warfarin should overlap for at least 48 hrs. before discontinuing heparin.

 The aim here is to reduce the risk of a further recurrence of venous thrombosis. Warfarin does not remove the clot from blocked veins and the duration of treatment (usually 3-6 months) is selected to prevent further episodes of venous thrombosis. Patients with recurrent venous thromboembolic problems should be anticoagulated for life.

If edema is present, the patient should remain on bed rest with the affected limb elevated above the level of the heart for several days. The patient should remain on bed rest for 2-3 days even if no pain or edema is present and even if the aPPT is at a therapeutic range to allow fixation of the clot to the vessel wall. Administer 3-6 months of therapy in the case of proximal DVT, assuming that surgery was the only predisposing risk factor.

- Daltapain sodium is administered at 200 IU/kg/day SC. With a single dose not to exceed 18000 IU.
- The use of subcutaneous injections of lowmolecular-weight heparin for the treatment of deep vein thrombosis is an alternative method of anticoagulation. The dose is based on the patient's weight and treatment given without blood tests to control the dose. This has been found to produce reliable anticoagulation without the risk of hemorrhage.

 This may in future become a very convenient way to manage patients with acute deep vein thrombosis, either in hospital or at home.
Warfarin treatment is commenced at the same time and controlled using the INR in the same way as for the intravenous heparin regime.  Enoxaparin sodium is administered at 1mg/kg q12 hrs. SC. Or at 1.5mg/kg/day SC. The single daily dose should not exceed 150 mg. The advantages of low-molecular-weight heparin over standard heparin include once a day administration and a lower risk of bleeding complications, making it more suitable for out of hospital use.

# Venous thrombectomy:

 Occasionally if massive venous thrombosis in the lower limb can leads to severe impairment in the blood supply to the limb, leading to ischemia and, eventually, gangrene. This is a surgical emergency and requires rapid relief of the venous obstruction. This can be achieved surgically by opening the femoral vein via an incision in the groin and removing all clots from the deep veins of the leg and pelvis.

 This operation used to be more widely performed on the assumption that it would reduce the severity of post-thrombotic vein damage following a deep vein thrombosis. However, very few surgeons now perform this operation. The more modern treatment of thrombolysis, achieved by passing a catheter into the affected vein and infusing a fibrinolytic drug such as streptokinase or tissue plasminogen activator (TPA), is reducing the need for this operation.