Dvt and treatment

Deep Vein Thrombosis and Treatment Introduction Formation of blood clots in the deep veins which often break loose to travel to the lungs is known as deep vein thrombosis (DVT) (Craig, 2005). Its prevalence and incidence can not be precisely predicted because it can go undetected as in many cases it is asymptomatic. Its prevalence is highest in those patients who are hospitalized and at bed rest with serious coexisting illnesses (Craig, 2005).

Etiology and pathogenesis

The primary mechanism for development of DVT is venous stasis, vessel wall injury and hypercoagulable state, commonly known as Virchow triad. The single most powerful risk factor for DVT is a prior history of venous thromboembolism. Other risk factors for DVT include postoperative period, pregnancy, puerperium, local trauma and stasis, smoking, obesity, lupus anticoagulant, post-stroke or neurological trauma, type-A blood group, malignancy, deficiencies of protein C, protein S or antithrombin III, impaired fibrinolysis as in post operative patients, those on estrogens, acute myocardial infection and congestive heart failure, hyperlipidemia, increasing age, inflammatory bowel disease and homocystinuria. Studies have shown that patients receiving general anesthesia have a 500% increased risk of DVT compared with patients receiving epidural anesthesia for the same surgical procedure (Craig 2005).

Pathophysiology

Vascular endothelial injury, even though minimal, exposes amorphous electron-dense substance which stimulates platelet adhesion and aggregation. The release of amorphous electron-dense substance is enhanced by activity of the intrinsic coagulation cascade. Platelet adhesion and aggregation causes formation of hemostatic plug after which

coagulation pathways are activated and thrombin is generated. Fibrin cross-linking builds a true thrombus out of what was initially a loose aggregation of blood elements (Craig, 2005). Normally, these series of events are opposed. In conditions as discussed above, these events are unopposed resulting in propagation of thrombus throughout the venous system. This is further accentuated by presence of reduced blood flow wherein the activated coagulation factors will accumulate.

Clinical manifestations

In many cases DVT may remain asymptomatic. The classic signs and symptoms of DVT are pain, tenderness, and unilateral leg swelling, due to obstruction to venous drainage. Other signs include warmth, erythema, a palpable cord, pain upon passive dorsiflexion of the foot, and spontaneous maintenance of the relaxed foot in abnormal plantar flexion (Craig 2005). Cellulitis can occur and in severe cases, pulmonary embolism can occur which is the dreaded complication of DVT.

Investigations

The standard diagnostic evaluation for patients with possible DVT is contrast venography. The most sensitive and specific test for the diagnosis of DVT is magnetic resonance venography (Craig 2005). Nuclear scintigraphic ventilation-perfusion scanning of the lungs remains the initial study of choice for most patients with possible PE. Though it usually yields nondiagnostic findings, in many cases it is capable of confirming or excluding the diagnosis with acceptable confidence levels. Blood tests have no role in either diagnosing or ruling out DVT. However, D-dimers may be raised in DVT, though it is not of much diagnostic value. D-dimer levels remain elevated in DVT for about 7 days (Schreiber 2007).

Treatment

The main treatment for DVT is anticoagulation. Initially, rapid anticoagulation is done with heparin followed by long-term anticoagulation with warfarin. Warfarin should never be started without prior heparinization because warfarin reduces the levels of anticoagulants before it reduces the levels of procoagulant proteins and this produces a hypercoagulable state during the first 5-7 days which can worsen thrombosis if given before heparinization (Craig 2005). Other anticoagulants which may be used are fondaparinux sodium, dalteparin and tinzaparin. In those in whom thrombolysis is not contraindicated, thrombolysis may be done before the initiation of anticoagulation therapy for prompt resolution of symptoms. Common fibrinolytics are tenecteplase, urokinase, streptokinase and alteplase (Schreiber 2007). In cases which have failed to standard anticoagulation therapy, clot removal and partial interruption of the inferior vena cava to prevent pulmonary embolism may be undertaken. Below-the-knee elastic stockings along with early ambulation are routinely recommended to assist calf muscle pump and reduce venous hypertension and venous valvular reflux (Schreiber 2007).

References

Feied, Craig. 2005. “ Deep vein thrombosis.” eMedicine from WebMD. 12th October 2007 Schreiber, Donald. 2007. “ Deepvein thrombosis and thrombophlebitis.” eMedicine from WebMD. 12th October 2007