Editorial

DEEP VEIN THROMBOSIS

Deep Vein thrombosis has been a common complication encountered in chronically ill patients and those having undergone major surgery. The pathogenesis of this problem was postulated as the triad of stasis, changes in the vessel wall and hypercoagulable state (Virchow 1856). Stasis or pooling of blood, such as occurs in sinuses found in the soleus muscles, can be a cause of deep vein thrombosis (Sevitt and Gallagher, 1959). Phlebographic studies show collection of the dye in the veins within the soleus muscle. These veins are devoid of valves and their filling source is the arterial supply plus some reflux from the tibial veins (Almen and Nylander, 1962). Also because they drain anteriorly, blood is pooled longer in the supine position and with the calf muscles in a resting state.

The changes taking place in the vessel wall have been attributed to intimal damage promoting platelet aggregation (Spaet and Erickson, 1966). This may be the sequence in cases undergoing surgery. But there have been situations where prolonged bed rest without associated tissue injury, has lead to the development of venous thrombosis. In such cases, stasis has been said to promote a chemotactic gradient leading to leucocyte migration, which in turn get entrapped between the endothelial cells and the basement membrane (Stewart 1975).

Local hypercoagulability is favoured by the endothelial damage which leads to the formation of a nidus of erythrocytes, platelets and fibrin. Recent studies of platelet physiology, particularly of factors responsible for platelet aggregation have shown the process to take place in arteries (French, 1969; Mustard et al., 1964).

No strong evidence is there for a similar process in veins. But in various studies (Hume, 1966; Obrien et al., 1972), the alteration of platelet function has been proved in instances of deep vein thrombosis, giving cause to believe that platelets play an important role in this condition.

The clinical diagnosis of deep vein thrombosis is not always reliable. Venograms are still considered as the standard tests, although the disadvantages of cost, discomfort and chemical phlebitis exist. This 125_1-labeled fibrinogen method based on the theory that thrombi could be detected by the uptake of the isotopically tagged material (Hobbs 1962) was found to be eighty eight percent more sensitive than the venogram (Gallus 1975). This technique being insensitive for pelvic veins is only useful for thrombi in the lower limbs. The Doppler ultrasound studies and plethysmographic methods are also useful in the detection of venous thrombi. Both these procedures are not useful for thrombi in the muscular veins, the deep femoral vein and the pelvic venous plexus, which are the silent areas. Contrast venography thus remains the best technique, but should be used only when absolutely necessary to establish the diagnosis.

To prevent the development of venous thrombosis it should be aimed at reducing the aiding factors of venous stasis, local hypercoagulability and platelet activity. The venous stasis could be minimized by periodic emptying of the sinuses in the muscles. This can be had either by external compression or direct electrical stimulation of the calf muscles. Active patient participation is difficult and the results of elastic stockings are conflicting.

The interference in the coagulation mechanism has been tried with good results using phenindione in patients with fractured hips (Sevitt and Gallagher, 1959). Low dose Heparin used prophylactically reduced the incidence of thrombo-embolism post-operatively (Sharnoff 1966). The two regimes employed are 5000 units subcutaneously twice daily or 5000 units thrice daily, the initial dose being given two hours prior to the operation. Trials carried out have shown a low incidence of venous thrombosis and subsequent pulmonary embolism than in controls undergoing surgery (Gallus et al., 1976). Bleeding complications and a greater requirement of blood could be expected in this treatment.

An alteration in platelet activity to reduce the incidence of venous thrombosis has been achieved in selected patients by using Dextran, Aspirin, Hydroxychloroquin and sulfin pyrazone.

Heparin plays the chief role in the treatment of established deep vein thrombosis. Administration by continuous infusion is as effective as intermittent injections, with lower incidence of haemorrhagic complications (Salzman et al., 1975). Monitoring may be done by keeping the partial thromboplastin time to one and a half to two times the normal (Hirsh 1975). It is recommended to continue heparin therapy for ten days followed by oral anticoagulants which should have reached a therapeutic level by this time. The duration of oral anticoagulation therapy is debated. The proposed regime is four to six weeks for calf vein thrombosis, three to six months for deep vein proximal to the calf, four to six weeks for mild pulmonary embolism and six months for major pulmonary embolism (Hirsh 1975). Urokinase and streptokinase are used only in patients with massive embolization causing associated complications (Urokinase-Streptokinase Trial, 1974). Surgery in the treatment of deep vein thrombosis is now considered obsolete due to the high rate of reocclusion

(Haller and Abrams, 1963; Mahorner et al., 1957). The role of heparin in adequate doses is therefore the most important step in treating patients with established deep vein thrombosis.

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