

VENOUS THROMBOSIS AND PREGNANCY

Pages with reference to book, From 1 To 2

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Pulmonary embolism is a major complication of pregnancy and is responsible for maternal deaths in a large number of patients (Friend and Kakkar, 1972). Venous thrombosis both superficial and deep with an episode of pulmonary embolism is likely to occur in one out of 70 pregnancies (Aaro and Juergens, 1971). Deep venous thrombosis alone with embolic episodes occurs between 1.6 and 4.7 in 1000 deliveries (Friend and Kakkar, 1972). Morris and Mitchell (1978) report that pregnancy is associated with sixfold increase in the risk of thromboembolism.

Obstetric complications increase the risk of venous thrombosis (Aaro and Juergens, 1971). The common complications associated with an increased risk for thromboembolism are prolonged labour, difficult forcep delivery, toxemia of pregnancy, haemorrhage and Cesarean section. Gallus (1976) in a survey of fatal pulmonary embolism either during pregnancy or after delivery found that these patients had one or more risk factors which included obesity, high parity, prolonged bed rest, operative delivery and age greater than 30 years. Jeffcoate and Tindall (1965) consider cesarean section as the most important single factor which seems to increase the risk of venous thrombosis. Seventy five percent of the fatal pulmonary embolism in Finnerty and Mackay's (1962) series was associated with cesarean section.

The factors which produce venous thrombosis viz venous stasis, hyper coagulable blood and venous intimal injury are well known. Venous flow is known to become sluggish during pregnancy which is brought by mechanical obstruction due to increased intra-abdominal pressure and the pressure of the foetal head on the veins of the pelvis (Wright et al., 1950). A decrease in venous tone is also a contributing factor in slowing the venous circulation (Flessa et al., 1974).

It is known that during pregnancy there is an increase in the clotting factors, II, VII, VIII, IX and X (Bonnar et al., 1970) and thus there is a state of hyper-coagulability. Several studies have been carried out to study plasma fibrinogen during pregnancy and it has been proved that plasma fibrinogen levels increase with the length of gestation (Bonnar, 1973). Yet another factor which increases the hyper coagulability is the reduction of fibrinolytic activity (Bonnar, 1973). A change in the concentration of anti thrombin III which is capable of inactivating thrombin, and factors IX, X and XI which thus profoundly influences the process of venous thrombosis (Moseley, 1980).

Evidence of intimal injury in pregnant patients with deep vein thrombosis is not available. The diagnosis of deep venous thrombosis is not always easy and clinical history and physical signs may not be elicited in as many as fifty percent of the patients. Phlebography provides the conclusive evidence of the presence of venous thrombosis but injection of contrast material and radiography during pregnancy is undesirable and could be dangerous. Similarly the use of radionuclide venograms carry considerable risk during pregnancy. Noninvasive techniques such as Doppler ultrasonic velocity detector and plethysmography have been used extensively in the detection of venous thrombosis. The most useful of these techniques is Doppler ultrasonic velocity detector (Bergqvist et al., 1976). The obvious drawback in these techniques is non-recognition of non-occlusive thrombosis, otherwise the technique is noninvasive, inexpensive, portable and reasonably accurate.

Anticoagulants are indicated in patients with a clinical diagnosis of iliofemoral vein thrombosis. Drugs with molecular weight of less than 1000 pass the placental barrier (Quenneville et al., 1959). Heparin with a molecular weight of 20,000 does not cross the placental barrier, Warfarin anticoagulants however, do so. The foetus has low levels of Vit K-dependent Factors II, VII, IX and X and Warfarin anticoagulants could increase the risk of haemorrhage in the foetus and there is some evidence of birth defects associated with oral anticoagulant therapy during the first trimester of pregnancy (Moseley,

1980).

Heparin does not cross the placental barrier or is known to produce teratogenesis, however, a large dose for prolonged periods produces osteoporosis (Hirsh et al., 1972). Haemorrhagic complications such as haematuria vaginal bleeding or postpartum haemorrhage are known to occur in patients on heparin therapy. All these complications however, can be prevented by careful control of patient's partial thromboplastin time. Heparin thus remains the drug of choice although its prolonged use is undesirable on account of its parental route of administration.

Elastic bandages and bed rest is considered sufficient treatment in patients where venous thrombosis is limited to calf (Wessler, 1972).

Several regimes of heparin therapy have been devised (Hill and Pearson, 1971). Mueller and Lebherz (1969) suggest that an initial episode of deep venous thrombosis be treated with seven to 14 days of heparin administered intravenously. In patients with suspicion of pulmonary embolism long term heparin administered sub-cutaneously or through a heparin lock is recommended.

Villasanta (1965) reported 297 patients with thromboembolism and of these 134 were treated with anticoagulants. He found that maternal mortality was 12.8 percent in the group not given anticoagulants as compared to 0.7 percent in those treated with anticoagulants.

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