



**SREE BALAJI MEDICAL COLLEGE HOSPITAL
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**MEDICAL EDUCATION UNIT
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**A BOOK ON DEEP VENOUS
THROMBOSIS & PULMONARY EMBOLISM**

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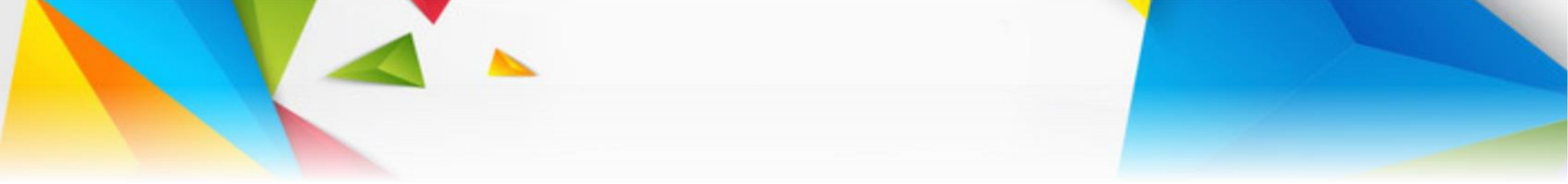
ABOUT THE BOOK

Hypercoagulability is an increased tendency to cause thrombosis. The aetiology can be in acquired and inherited factors. It predisposes to arterial, venous and micro vascular complications. Hypercoagulability itself is a risk factor for venous thrombotic and atherothrombotic events. Some prevalent inherited hypercoagulable states are *factor v Leiden mutation* and *prothrombin G20210A* which is due to factor v and prothrombin gene mutation. The other inherited hypercoagulable states are due to deficient of antithrombin, protein C and protein S. Mutation in methyl tetrahydrofolate reductase cause raised homocysteine which is a misconception of inherited hypercoagulable state. Antiphospholipid syndrome is a significant factor for acquired hypercoagulability. It's a secondary response to rheumatological and lymphoproliferative conditions. Other acquired conditions are heparin inducing thrombocytopenia, paroxysmal nocturnal hemoglobinuria, myeloproliferative malignancies etc,. Malignancy associated venous thromboembolism differs with type of malignancy and management.

Hypercoagulability can be suspected, when the patient presents with

- A. Idiopathic thrombotic events at any age
- B. Family history of venous thromboembolism
- C. Thrombosis at unusual sites
- D. Recurrent unprovoked thrombosis
- E. Recurrent unexplained foetal loss
- F. Warfarin induced skin necrosis
- G. Purpurin fulminans
- H. Recurrent superficial thrombophlebitis

This book focus on the overview of hypercoagulable conditions and its manifestations in a precise manner. It is to fetch the knowledge among the medical practitioners and students for preventing the cascade of thrombotic events and to get better prognosis in management.



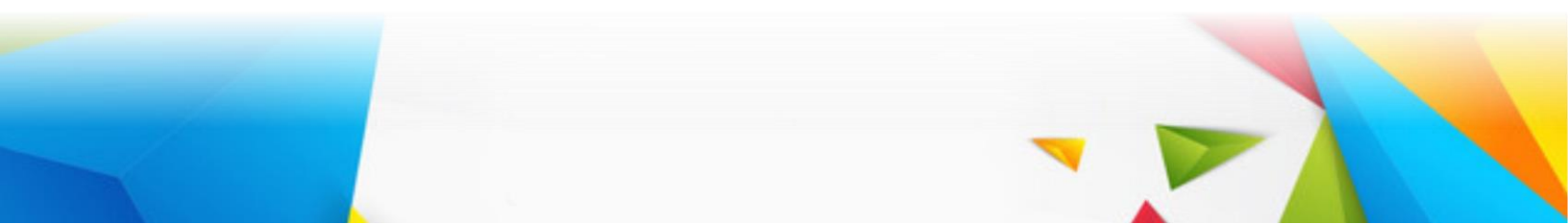
There is no measureable radiation exposure associated with ultrasound or magnetic resonance imaging. However there are risks to the fetus and mother associated with radiation exposure for contrast venography, and with iodinated and gadolinium contrast for venographic studies. **DIAGNOSIS**

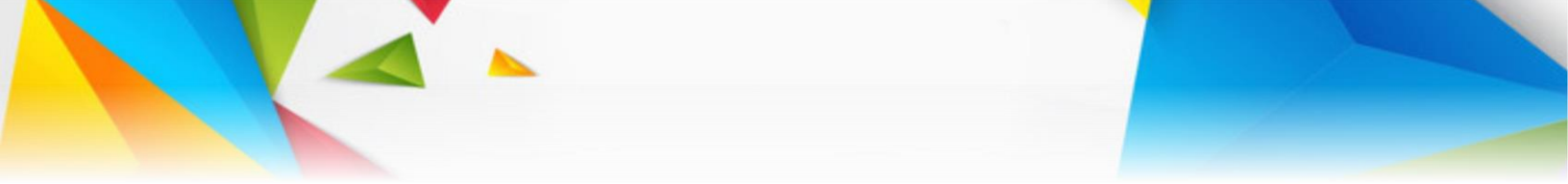
The diagnosis of deep vein thrombosis (DVT) in pregnancy is made by demonstrating a lack of compressibility of the proximal veins on compressive ultrasound (femoral vein thrombosis) or poor flow on Doppler imaging of the femoral-iliac vein (iliac vein thrombosis). The diagnosis is rarely made by the demonstration of a filling defect on contrast or magnetic resonance venography. D-dimer levels and clinical exam cannot be used alone to diagnose DVT.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of DVT in pregnancy is similar to that in non-pregnant patients. It includes other entities that present with erythema, warmth, swelling and tenderness of the lower extremity and/or flank, lower abdomen, buttock, or back. However, the clinical signs and symptoms of DVT in pregnancy are masked by many of the physiologic changes of normal pregnancy (eg, lower extremity swelling and cramping).

The clinical suspicion for DVT should be high in the setting of pregnancy. Although not always present, features that are highly suggestive of the diagnosis include unilateral signs and symptoms and the classic symptoms of iliac vein thrombosis (flank/pelvic/buttock pain and swelling). Such symptoms should prompt immediate investigation for DVT with compressive or Doppler ultrasound. It is important to note that DVT can coexist with other conditions. Thus, although the





finding of an alternative diagnosis (eg, cellulitis) will lower the clinical suspicion for DVT, it does not always obviate the need for diagnostic imaging.

The differential diagnosis of DVT in nonpregnant patients and the physiologic changes of pregnancy that can mimic DVT are discussed separately.

