Deep Vein Thrombosis (DVT) in Pregnancy

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Case 1
Mrs. X, 27 years old, G4 P2+0+1+2 reported at 26 weeks of pregnancy with pain and swelling of right leg. On examination she was pale and had unilateral oedema of right leg with 26 weeks singleton pregnancy. Her haemoglobin was 6 gm% with peripheral blood picture of microcytic hypochromic anaemia. Colour doppler scan of lower limbs showed partial obstruction in right iliac vein, normal flow in right femoral and popliteal vessels (Fig. 1). Normal flow was documented in left iliac, femoral, and popliteal veins. She was found to be having thalassaemia trait and transfused two units of blood. Simultaneously she was started on intravenous heparin (unfractionated), 5,000 IU loading dose followed by 750 units per hour as continuous IV infusion for 5 days. Further, she was maintained on low molecular weight heparin (LMWH), nadroparin (Flaxiparine) 3,075 IU once a day for the remaining period of pregnancy until delivery. After 15 days, a repeat doppler study showed complete resolution of thrombus with normal flow (Fig. 2). Post partum period was covered by oral anticoagulants for six weeks.

Case 2
Mrs. Y, 21 years old, primipara reported on 23rd post-partum day with swelling and pain of left leg. Her clinical examination and colour doppler study showed deep vein thrombosis involving left ilio-femoral and popliteal veins. She was started on LMWH (flaxiparine) twice a day for 5 days and put on warfarin (10 mg) the same day. PT was kept more than twice the normal. It took 3 weeks for clot to resolve and oral anticoagulation was continued for 3 months. Within five months of delivery, patient reported with a second pregnancy. She refused medical termination of pregnancy and opted to continue pregnancy. After counselling her about the risk of recurrence of DVT, decision was taken to clinically monitor her for signs of DVT and cover her for thromboprophylaxis in post-partum period.

Venous thromboembolism in pregnancy presents...
a considerable diagnostic and therapeutic challenge. Cases such as these highlight several important management issues for DVT occurring during pregnancy, e.g., Which are the safest and most appropriate diagnostic tests? What types of therapy can be recommended? Are prophylactic measures warranted in patients with past history of DVT? Is aetiological evaluation warranted? We reviewed the literature to find answers to these frequently encountered clinical queries.

How common is DVT in pregnancy?

DVT of the lower extremities during pregnancy occurs at a rate of 0.13 to 0.61 per thousand pregnancies. Despite its relatively low incidence, DVT may lead to pulmonary embolism, the most common cause of maternal deaths in the developed countries. In the past, rates of fatal pulmonary embolism were highest in the post-partum period. Risk of thrombo-embolism was increased because of medical practices such as operative (caesarean or instrumental) delivery, prescription of prolonged bed rest after delivery, and use of oestrogens to suppress lactation. Changes with health care practices have reduced the incidence of iatrogenic pulmonary embolism in the puerperium. Other studies have documented that venous thrombo-embolism occurs with relatively equal frequency in all trimesters of pregnancy.

What predisposes a pregnant female to develop DVT?

Pregnancy itself is an independent risk for thrombo-embolism. The incidence of DVT is five times higher in pregnancy than in age matched non-pregnant females. Virchow's triad of hypercoagulability, stasis, and endothelial injury operates during pregnancy and puerperium. Increased levels of clotting factors (factor I, II, VII, IX, and X) together with decreased fibrinolysis and reduced levels of the natural anticoagulant, protein S contribute to this state of hypercoagulability during pregnancy. Venous stasis resulting from pressure of the gravid uterus on inferior vena cava and decreased venous tone are further predisposing factors present in all pregnant women. In an USG study of gestational changes of the venous system of the lower extremities, Macklon and associates documented decreased flow velocity and increased vessel diameter of the deep leg veins. At term, flow velocity of the femoral vein slowed to less than 1/3 of the velocity recorded in the 1st trimester and subsequently in the post partum period. Although pregnancy itself is not associated with endothelial injury, the trauma of operative delivery may result in vascular injury, leading to postpartum DVT. Other important risk factors are as shown in the table.

Table I: Risk factors for DVT.

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tr>
<td>Increased parity (&gt; 4)</td>
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<tr>
<td>Obesity</td>
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<tr>
<td>Operative or difficult instrumental delivery</td>
</tr>
<tr>
<td>Prolonged immobility</td>
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<tr>
<td>Previous thrombo-embolism</td>
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<tr>
<td>Thrombophilias</td>
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Multiple risk factors are often present in women who develop DVT during pregnancy and the risk factors are cumulative. In addition, an occult thrombophilia such as Factor V Leiden mutation may become unmasked during an otherwise normal pregnancy.
Which side is more prone during pregnancy for DVT?

During pregnancy venous thrombosis begins most frequently either in the calf veins or in the iliofemoral segment of the deep venous system. There is a striking propensity for the left leg, with approximately 80% of DVT’s in pregnancy occurring on this side. The venous drainage of the left leg follows a more tortuous course through the pelvis, with the left common iliac vein traversed by the right common iliac artery. It has been suggested that this renders the left leg more prone to DVT.

Are clinical features diagnostic enough to start treatment for DVT?

Clinical diagnosis of DVT and thrombo-embolism is notoriously unreliable. The intensity of the classical symptoms of pain, tenderness, and swelling of the affected limb depends on the extent of the vascular occlusion, existing collateral circulation, and the associated inflammatory response. The physiological changes of pregnancy further complicate interpretation of the patient’s history, physical findings, and test results. Lower extremity oedema and leg pain in pregnancy may be due to lymphatic obstruction and increase in intravascular blood volume rather than DVT. In fact, DVT in pregnancy may present atypically with diffuse abdominal pain. Dyspnoea, a common symptom of DVT, is experienced by nearly 3/4th of females during normal pregnancy. Such frequent and non-specific manifestations can easily create a diagnostic enigma for the clinicians.

Can non-invasive tests confirm the diagnosis of DVT?

In the last decade, non-invasive diagnostic studies such as impedance plethysmography, real time B mode USG, and duplex doppler scanning have replaced venography as the initial screening test in the diagnosis of DVT. These diagnostic studies have high sensitivity for the detection of thrombosis in the proximal ilio-femoral veins but not in the distal deep veins of the lower extremity.

Impedance plethysmography measures changes in electric resistance measured by two electrodes wrapped around the calf in relationship to changes in venous volume. Serial normal studies performed over 7-14 days have shown sufficient sensitivity and specificity to withhold therapy in both non-pregnant and pregnant patients.

Doppler ultrasonography: This technique has become the diagnostic study of choice in cases of suspected proximal vein occlusion. A 5 MHz transducer is placed over the veins. Venous flow produces a characteristic low pitched sound that is abolished by venous occlusion. Concomitant evaluation of venous anatomy, flow, augmentation (increased flow with muscular activity in the calf), and compression (elimination of residual lumen by firm pressure with the transducer probe using one hand) yields a correct diagnosis with high degree of sensitivity and specificity. The sensitivity and specificity in the evaluation of proximal vein thrombosis are 91% and 99% respectively.

Limitations: Doppler flow studies are less effective in calf vein thrombosis with a sensitivity and specificity of 36% and 95% respectively. Studies have shown that only 20% of these thrombi extend proximally and that calf vein thrombi are not life threatening as long as they remain confined to the calf. Therefore when DVT below popliteal vein is suspected, serial measurements are necessary to rule out propagation of a clot. Other limitation is that doppler USG is not sensitive for asymptomatic thromboses.

What is the role of venography and how safe is it in pregnancy?

Venography remains the diagnostic standard for DVT in both pregnant and non-pregnant patients. Venography has the advantage of accurately evaluating the entire lower extremity from the calf veins to the common iliac vessels. It is also more reliable than non-invasive
techniques in differentiating between intraluminal defects and external venous compression. Potential side effects are chemical phlebitis, leg swelling, pain, and skin necrosis secondary to dye extravasation. Procedure is invasive and is associated with risks of provoking thrombosis and contrast reaction. In addition, the procedure is relatively expensive and the results can be difficult to interpret. Estimated foetal radiation exposure is negligible: approximately 0.314 rad for a unilateral procedure without abdominal shielding\textsuperscript{15}. Limited venography, using an abdominal shield, can reduce the estimated foetal exposure to less than 0.05 rads.

The role of venography in diagnosing DVT during pregnancy remains unresolved. Venography may be helpful when the results of non-invasive imaging studies are equivocal or serial scanning is impractical.

What are the recent advances in the diagnosis of DVT?

**Magnetic resonance imaging:** MRI has recently been established to be a reliable method for diagnosing pelvic and lower extremity venous thromboses. It is at least as accurate as venography for proximal thromboses in the lower limb and possibly even more sensitive for pelvic vein thromboses. Obvious advantages in pregnancy include its non-invasiveness, lack of ionising radiation exposure, and excellent resolution of the IVC and the pelvic veins.

**Blood tests:** Several tests are available that reflect the formation of intravascular fibrin. Results are invariably positive when thrombosis has occurred. The assays for fibrinopeptide A and the fibrin degradation products: D-dimers are the most sensitive. At present, data are insufficient to determine the utility of this promising test in the setting of pregnancy. Elevations in D-dimers levels are found even in uncomplicated pregnancy, with levels increasing during the course of gestation\textsuperscript{16}. A finding of normal level of these however, essentially rules out DVT.

**Is diagnostic workup for congenital thrombophilias required in these cases?**

Activated protein C resistance due to factor V Leiden mutation is the commonest thrombophilia. The estimated prevalence among people of European ancestry is 5% but the defect is less common in other ethnic groups. It is rarely found in Asians and Africans\textsuperscript{17}. Therefore, diagnostic workup for thrombophilia is not indicated in all cases of DVT in pregnancy, but only in selected patients with clinical indicators of hypercoagulable state (Table II).

**Table II : Clinical indicators of thrombophilias.**

<table>
<thead>
<tr>
<th>Family H/O thrombosis</th>
<th>Recurrent thrombosis</th>
<th>Idiopathic thrombosis</th>
<th>Thrombosis at unusual sites (e.g., axillary, cerebral, mesenteric, portal, hepatic veins)</th>
<th>Skin necrosis after starting warfarin therapy</th>
</tr>
</thead>
</table>

**Management of DVT in pregnancy**

**What are the aims of the treatment of DVT?**

Aims are:

1. To prevent extension of the thrombus.
2. To restore venous patency and thus in the long run to prevent the post-phlebitic syndrome due to venous insufficiency with features like chronic pain, swelling, and sometimes ulceration of the affected limb.
3. The most important aim is to prevent pulmonary embolisation or its recurrence. Incidence of pulmonary embolism depends on whether or not DVT is adequately treated. Untreated, as many as 24% of the patients with antenatal DVT will have pulmonary embolism with a mortality rate of approximately 15%\textsuperscript{18}. If patients are treated with anticoagulants, embolisation occurs in...
only 4.5% with a mortality rate less than 1%, hence the importance of proper treatment.

Is bed rest absolutely necessary in all cases of acute DVT?

Bed rest with elevation of the affected extremity is invaluable initially because it promotes venous return and decreases oedema. Preferable is the Trendelenberg position, which involves elevating the foot end of the bed approximately 8 feet. As soon as symptoms permit, the patient should be encouraged to ambulate, since bed rest itself may enhance venous stasis. There is no evidence that bed rest will prevent embolus detachment. Sitting with legs dependent is contraindicated.

Should we advocate elastic bandage/stockings?

When correctly designed, elastic stockings increase the velocity of venous flow. The pressure gradient should decrease from ankle to thigh without a constricting garter at the top. Elastic bandages once in vogue, are best avoided because they are easily wrapped incorrectly with the greatest pressure ending up at the top, thus impeding venous return.

Does DVT limited to calf require treatment?

Although it is widely accepted than proximal DVT should be treated with anticoagulants, the need for treatment of thrombosis below the level of popliteal fossa remains in dispute as risk of pulmonary embolisation in such cases is very less (1%). Because approximately 20% of lower DVT’s extend proximally and anticoagulants can prevent this spread, many believe that treatment is required. An alternative is frequent doppler flow studies to detect extension and then treat.

How safe are the anticoagulants in pregnancy?

Anticoagulant therapy for pregnant patients is complicated not only by maternal risks, but also by the foetal risks of teratogenicity and foetal haemorrhage. The pregnancy categories of the most commonly used anticoagulants and thrombolytics are listed below.

Table III : FDA pregnancy categories of anticoagulants.

<table>
<thead>
<tr>
<th>Agents</th>
<th>Pregnancy category</th>
</tr>
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<tbody>
<tr>
<td>Warfarin</td>
<td>X</td>
</tr>
<tr>
<td>Unfractionated heparin</td>
<td>C</td>
</tr>
<tr>
<td>Dalteparin sodium (LMWH)</td>
<td>B</td>
</tr>
<tr>
<td>Enoxaparin sodium (LMWH)</td>
<td>B</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>C</td>
</tr>
<tr>
<td>Urokinase</td>
<td>B</td>
</tr>
</tbody>
</table>

What is the role of oral anticoagulants in pregnancy related DVT and how safe are these?

Warfarin is the most widely used coumarin derivative for oral anticoagulation. Its therapeutic efficacy lies in its ability to inhibit the action of vitamin K. Vitamin K functions in the liver as a cofactor in the synthesis of 4 essential clotting factors – factor VII, IX, X, and prothrombin.

Dosage : Usual anticoagulation dose is 10-15 mg daily until a therapeutic prolongation of prothrombin time (PT) is achieved. An appropriate International Normalisation Ratio (INR) is 2.0-3.0. Initially PT is monitored daily for 5-7 days then twice weekly for 1-2 weeks and then weekly for several months depending on the response. Large loading doses should be avoided because of the increased likelihood of overcoagulation or quite possibly an early prothrombotic effect due to excessive protein C inhibition. During the first 5-7 days of warfarin therapy, heparin needs to be continued because its action is not immediate, as it is dependent on the synthesis of clotting factors.

Warfarin use during pregnancy is limited by the fact that it crosses placenta. There is 5% risk of embryopathy if taken between 6th and 12th week of pregnancy. First trimester exposure can lead to nasal hypoplasia, stippled epiphyses, and limb hypoplasia. Foetal exposure at any time during
pregnancy can lead to neurological abnormalities. Exposure to coumarin derivatives outside the first trimester has been associated with a variety of CNS abnormalities including midline dysplasia, midline cerebellar atrophy, and mental retardation. Ophthalmic abnormalities including optic disc atrophy, microphthalmia, and blindness also have been reported\textsuperscript{22}. It can also cause foetal and neonatal haemorrhage and placental abruption. Warfarin can also cause major maternal bleeding and its action is not as easily reversed as that of heparin. Therefore, its use during first trimester is absolutely contraindicated, though the use during 2nd and 3rd trimester is somewhat controversial. Preferably, its use during pregnancy should be avoided unless specifically indicated. It is not secreted in the milk, thus is safe for use during lactation. If used during pregnancy, it should be replaced by heparin after 36 weeks or at the onset of labour. If spontaneous labour occurs while the patient is still taking warfarin, administering vitamin K and fresh frozen plasma can reverse its effect.

Which is the most commonly used anticoagulant during pregnancy?

Unfractionated heparin, a naturally occurring mucopolysaccharide is the anticoagulant used most widely for DVT in pregnancy. In plasma, it combines with antithrombin III to become a potent inhibitor of thrombin and to increase the circulating levels of activated factor X inhibitor. If necessary, heparin effects can be reversed rapidly with protamine sulfate in a dose of 1 mg/100 units of administered heparin. No more than 50 mg should be given over any 10 min. period because it itself can cause bleeding.

What are the therapeutic doses of heparin?

Adequate dose of heparin affects the recurrence rate of thrombosis. Lack of adequate anticoagulation increases the risk of recurrence by 11 to 15 folds. Optimal anticoagulation is obtained with an aPTT of 60-80 sec (1.5 to 2.5 times control). Spontaneous haemorrhage frequently occurs if the aPTT exceeds 135 sec. for periods longer than 12 hours. Before heparin is initiated a complete blood count, platelet count, PT, aPTT, and urinalysis are obtained. For patients with uncomplicated DVT, the loading dose is 100 U/kg with a minimum of 5,000 units. Following it, the initial infusion rate should be 15-25 units/Kg/hour. An aPTT should be obtained 4 hours after the loading dose and appropriate adjustment should be made.

Continuous IV infusion of heparin should be administered for 3-5 days for active thromboembolic disease or until symptoms have resolved and there is no recurrence. It is followed by adjusted dose regimen of heparin for 4-6 months followed by a prophylactic dose for the remainder of pregnancy and 6-12 weeks postpartum. An adjusted dose regimen is accomplished by administration of heparin s c every 12 hr. and adjustment of dose to achieve an aPTT of 1.5-2 times control at 6 hrs. Once a stable dosage is reached, a mid-interval aPTT should be checked weekly at the pre-natal visits. It should be anticipated that heparin requirements would increase during pregnancy until term.

Should anticoagulant be given during labour?

If DVT occurred 3 or more months before the expected due date, anticoagulation during labour is not indicated. Patient can be instructed to discontinue therapy with the onset of labour. Therapy should be restarted promptly 4-6 hrs post-partum. Regional anaesthesia is not contraindicated if aPTT is normal and heparin has not been administered within 4-6 hr. of procedure\textsuperscript{23}. If anticoagulation is required during delivery, the dose should be adjusted to achieve an aPTT of 1.5 times control during labour and delivery. This does not increase the incidence of PPH in a normal delivery; however, there is a slight increase in the incidence of episiotomy haematoma. Conduction anaesthesia is contraindicated in these patients.
What are the complications of heparin therapy?

Heparin does not cross placenta so there are no foetal hazards. However, its long-term use, as is required in DVT, can have some adverse effects in the mothers. Bleeding is an obvious risk with any anticoagulant, but the rate of serious bleeding in pregnant patients treated with heparin (2%) is comparable to the rate in non-pregnant patients. Subcutaneous heparin can cause persistent anticoagulation after cessation of use. Therefore it is recommended that subcutaneous heparin be converted to IV heparin 24 hr. before elective induction of labour.

Osteoporosis: It is particularly relevant to pregnancy because heparin treatment is frequently prolonged. In addition, pregnancy itself and breast feeding affect bone demineralisation. The risk of symptomatic osteoporosis with prolonged heparin treatment is low (about 2%) but subclinical osteopenia may occur in up to a third of women. Fortunately, these effects appear to be at least partially reversible and bone density generally improves with discontinuation of heparin.

Thrombocytopenia: It exists in 2 forms – an early benign form with mild thrombocytopenia occurring after one to several days of treatment and delayed condition which occurs 6-10 days after commencing treatment. The latter is an immune-mediated reaction associated with severe thrombocytopenia, paradoxical arterial, or venous thromboses with significant morbidity and mortality and requires immediate withdrawal of heparin treatment. Patients on long term heparin treatment should have their platelet count checked 1 week after commencing treatment and monthly thereafter.

What is heparin resistance?

It is arbitrarily defined as need for more than 20,000 units per day. Mainly occurs in patients with large venous thrombo-emboli. Mechanism of Resistance:

i) Inherited or acquired decrease in AT III levels.

ii) Increased plasma levels of factor VIII.

iii) Increased plasma levels of heparin binding proteins.

In patients with heparin resistance, monitoring with the aPTT is not recommended. Instead, monitoring with antifactor Xa assay can be used. Alternatively, low molecular weight heparin appears to be effective because they have less protein binding.

What are low molecular weight heparins (LMWH)?

These are fragments of conventional heparin produced by enzymatic or chemical breakdown. Like heparin they do not cross placenta, are non-teratogenic and are not secreted in milk. By virtue of their shorter and lighter structures, LMWHs produce a predominantly anti-thrombotic effect through their inhibition of factor Xa with little anticoagulant activity.

Are different LMWHs similar in action?

LMWHs have a molecular weight between 4,000 and 6,000. Various formulations of LMWHs differ in mean molecular weight, glucosaminoglycan content, and anticoagulant activity. The various fractions of heparin have different pharmacological profiles in terms of bioavailability, plasma clearance, and release of tissue factor pathway inhibitor. Each LMWH requires individual evaluation. Properties of a particular LMWH cannot be extrapolated to a different LMWH.

What are the advantages of LMWH over unfractionated heparin (UFH)?

1. Studies have conclusively proved that LMWHs are as safe and effective as UFH. Their anticoagulant effects are predictable with minimal alterations in PT and aPTT. This translates into a lower risk of haemorrhagic complications.

2. A lower risk of heparin-induced thrombocytopenia because they are less likely to activate resting platelets to release PF4 and
bind less well to PF4.

3. Their increased bioavailability and longer half-life permits once or twice daily administration.

4. Bone density loss is comparable to physiological loss of pregnancy.

5. Anti Xa, activity is correlated with body weight, which permits administration in a fixed dose.

6. No need for lab. monitoring for coagulation tests like PT and aPTT.

Table IV: Comparison of UFH and LMWH.

<table>
<thead>
<tr>
<th>Effect</th>
<th>UFH</th>
<th>LMWH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean molecular wt. (Daltons)</td>
<td>15,000-30,000</td>
<td>3,000-9,000</td>
</tr>
<tr>
<td>Anti Xa : Antithrombin ratio</td>
<td>1 : 1</td>
<td>2-4 : 1</td>
</tr>
<tr>
<td>Protein binding</td>
<td>Significant</td>
<td>Minimal</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Not rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Bioavailability</td>
<td>Approx. 30%</td>
<td>Approx. 90%</td>
</tr>
<tr>
<td>Route of administration</td>
<td>S/c or i/v</td>
<td>S/c</td>
</tr>
</tbody>
</table>

Does LMWH treatment require lab. monitoring?

There is no need for lab. monitoring as weight adjusted dosage of LMWH has very predictable effect. Monitoring antifactor Xa levels: can be altered but are not very readily available and are very costly. Periodic complete blood counts and stool for occult blood are indicated to monitor for possible bleeding complications. Since these agents are eliminated primarily through the kidneys, accumulation of antifactor Xa activity may occur in patients with chronic renal insufficiency. Plasma antifactor Xa concentration should be monitored in patients with renal dysfunction and possibly in those weighing less than 50 kgs or more than 80 kgs.

Can LMWH be used for acute treatment of DVT?

Currently use of LMWHs in pregnancy is mainly confined to the chronic phase of treatment and to thromboprophylaxis. Clinical trials suggest that LMWH given SC can replace the standard IV application of unfractionated heparin in the initial treatment of DVT, granting equal or even better efficacy and potentially lower rates of adverse side effects. Full anticoagulation doses of LMWH are:

- ENOXAPARIN 1 mg/kg SC bd
- DALTEPARIN 100 iu/kg SC bd

Do all patients with DVT require hospitalization?

Advent of LMWH for treatment of DVT with advantages of subcutaneous once or twice daily dosage without monitoring has made it possible to treat patients in an out-patient setting. But pregnant state is a contraindication for such treatment.

What is the role of thrombolytics in pregnancy?

No controlled trials on the safety and efficacy of thrombolytics in pregnant patients have been done. Because of the risks of maternal haemorrhage and foetal loss, thrombolytic therapy should be reserved for patients with massive pulmonary embolism and severe haemodynamic instability. In DVT, it is a reasonable option when viability of the affected limb may be in jeopardy, e.g., phlegmasia alba dolens and phlegmasia cerulea dolens. In these conditions, it is reasonable to accept the risk of bleeding to potentially save the limb.

Is thromboprophylaxis required in subsequent pregnancy?

Risk of recurrent venous thromboembolism in...
pregnancy for a woman who has a prior episode is not known definitely. Retrospective studies have estimated the risk to be 4-15%30. Recent data suggests that these women may have an increased risk of recurrent thrombosis because, compared to controls, they have higher plasma levels of biochemical markers of activation of the coagulation cascade during subsequent pregnancy.

**Prophylactic doses of UFH and LMWH** : Higher doses are needed for effective prophylaxis during pregnancy to offset the characteristic increase in plasma volume, renal clearance, and blood levels of coagulation factors and to counteract the alterations in metabolism of heparin that occurs during pregnancy. Subcutaneous heparin 7,500 to 10,000 U should be administered twice daily. Prophylactic dosage of LMWHs is administered once daily. In cases of DVT during previous pregnancy if there are associated risk factors like thrombophilia or antiphospholipid syndrome, prophylaxis should be started in 1st trimester and continued until 6 weeks postpartum. If there is no other risk factor except for H/O DVT in previous pregnancy, the different strategies recommended are:

- Clinical surveillance followed by warfarin postpartum x 4-6 weeks
- Or
- Heparin or LMWH throughout pregnancy followed by warfarin/LMWH x 4-6 wks.

**References**


23. Herlocker TT. Central neural blockage for patients