Superficial Vein Thrombosis (SVT) of Pregnancy

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Superficial Vein Thrombosis

- Concomitant DVT 25-30% of patients at presentation
- PE in 4-7% of patients at presentation
- Subsequent VTE 2-6% patients depending on follow-up duration
- Another SVT in different saphenous system 37.5%
- Recurrent SVT same saphenous system 26%
- All SVT patients need bilateral duplex scan to confirm diagnosis
- Determine precise location and extent of SVT
- SVT SHOULD NOT BE CONSIDERED A BENIGN CONDITION

Superficial Vein Thrombosis

- Symptomatic SVT at least 5 cm length prophylactic dose of low molecular weight heparin for 45 days over no anticoagulation (grade 2B 2012 ACCP guidelines)
- Pregnancy defined as a subgroup with higher incidence of VTE after a SVT
- Patients with assisted reproductive technologies have slightly higher incidence of VTE in pregnancy than those after natural conception
- On average 1-2 women per 1,000 will get a venous thrombosis during pregnancy.

Thromboembolic Complications of SVT

- More often SVT involved GSV extended to within 10 cm SFJ
- Involved veins above the knee
- Involved patients with history of VTE

Pregnancy Changes

- Smooth muscle relaxation
- Vasodilation
- Valve incompetence
- Expanded blood volume
- Decreased blood flow velocity
- Venous stasis
- Varicose veins developing during pregnancy – 8 – 20%
  - 13% primiparous
  - 16% secundiparous
  - 57% multiparous

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Nothing to Disclose
I have no relevant financial relationship(s) with any proprietary entity producing health care goods or services related to the content of my talk.


Pregnancy Changes

- Compression of iliac veins by enlarging uterus
  - Venous pressure increases
  - Venous flow patterns less responsive to respiration
  - Hypotension
  - Reduced cardiac output
  - Turn on side reversible

Hemodynamic Changes Causing Stasis

- Increased venous distensibility
- Increased capacity
- Reduction in velocity of lower limb blood flow
- Hypercoagulability

Autonomic Circulatory Control During Pregnancy

- Maternal hemodynamic alterations begin 4-5 weeks gestation plateau 2nd trimester
- Decreased systemic vascular tone
- Cardiac output rises (~40%)
- Expansion blood volume (activation renin – angiotensin – aldosterone system)
  - Decreases after load
  - Increased preload
- Uteroplacental vascular circuit can be likened to an arteriovenous fistula retention of fluids leading to expansion of blood volume and primary systemic vasodilation

Coagulation changes in Pregnancy

- Virchow’s Triad - all three components
  - Vessel wall injury, hypercoagulation, venous stasis
- Fibrinogen levels double in pregnancy
- Factors VII, VIII, IX, X and XII increase
- fibrinolytic activity decreased
- 40% decrease in free Protein S, an inborn anticoagulant
- Increased venous stasis
- Vascular injury associated with delivery
- Increased activation of platelets

Changes during Pregnancy that Affect Coagulability

- Increase in von Willebrand factor
- Increased generation of fibrinogen and fibrin split products
- Increase in plasminogen activators (i.e., plasminogen activator inhibitor 1 and 3)
- Inhibition of fibrinolytic system (i.e., decrease in activity of factors XI and XII and antithrombin III)
- Reduction in free protein S
- Progressive resistance to activated protein C
- Peak in the second trimester persist until 6 weeks postpartum

Common Risk Factors for Venous Thromboembolism in Pregnancy

- Patient Factors
  - Age more than 35 years
  - Obesity (BMI >29kg/m²) in early pregnancy
  - Thrombophilia
  - Past history of VTE (especially if idiopathic or thrombophilia associated)
  - Gross varicose veins
  - Significant current medical problems (e.g., nephrotic syndrome, anemia, diabetes mellitus, cardiac disease, hypertension)
**Common Risk Factors for VTE in Pregnancy**

- **Patient Factors**
  - Current infection or inflammatory process (e.g., active inflammatory bowel disease or urinary tract infection)
  - Immobility (e.g., bed rest or lower limb fracture)
  - Paraplegia
  - Recent long-distance travel
  - Dehydration
  - Intravenous drug abuse

- **Pregnancy/Obstetric Factors**
  - Ovarian hyperstimulation
  - Cesarean section, particularly as an emergency in labor
  - Complicated vaginal delivery
  - Major obstetric hemorrhage
  - Multiparity (four or more deliveries)
  - Hyperemesis gravidarum
  - Preeclampsia
  - Use of estrogen to suppress lactation

**PEARLS of Wisdom**

- Examination not reliable to establish proximal extent of SVT, especially in the thigh
- Non-occlusive thrombus may propagate into the deep venous system without significant venous flow disturbance and absence clinical features to suggest DVT (non-occlusive thrombi account for up to 60% of DVT associated with SVT)
- Thrombotic extension may complicate 5-10% of those treated with prophylactic anticoagulants – careful monitoring and rescanning
- Therapeutic or prophylactic dosing controversial as is the duration of treatment
- Risk assessment needed but remember thromboembolism still leading cause of maternal death

**Practice Points**

- PTE is the major cause of maternal mortality in the U.K. and USA
- Complications of SVT frequently clinically occult
- Congenital thrombophilias underlie about 50% of episodes of VTE in pregnancy

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