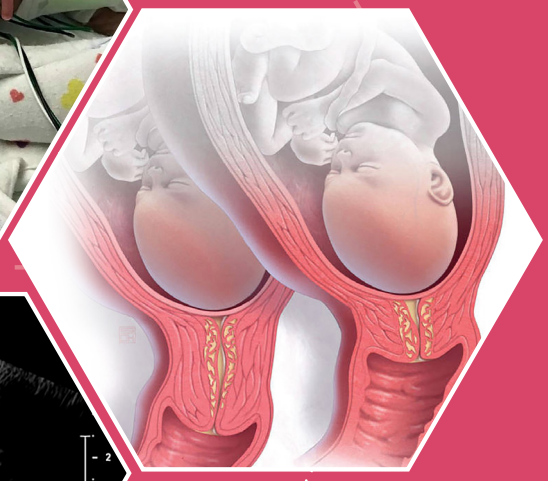
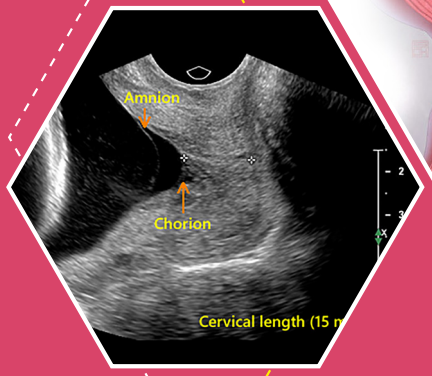
سازمان نظام پزشکی جمهوری اسلامی ایران





خلاصه مباحث

وبینار ترومبوز و بارداری

پنجشنبه ۲۲ خرداد ۱۳۹۹

سرفصل‌ها و مدرسان:

ترومبو پروفیلاکسی اسکورینگ در حاملگی و بعد از زایمان
دکتر زهرا سلیمانی؛ پری‌ناتولوژیست
عضو هیات علمی دانشگاه علوم پزشکی بقیه الله

ترومبوفلبیت و آمبولی ریوی پیشگیری، تشخیص و درمان در حاملگی
دکتر مریم نورزاده؛ پری‌ناتولوژیست
عضو هیات علمی دانشگاه علوم پزشکی تهران

ترومبوفیلی ارثی و حاملگی
دکتر ترانه ارباب‌زاده؛ پری‌ناتولوژیست

آنتی فسفولیپیدها و حاملگی
دکتر مریم مشفقی؛ پری‌ناتولوژیست
عضو هیات علمی جهاد دانشگاهی

آسپرین و حاملگی
دکتر نوشین اشراقی؛ پری‌ناتولوژیست
عضو هیات علمی دانشگاه علوم پزشکی ایران

معاونت آموزش سازمان نظام پزشکی کشور و تهران بزرگ

RISK ASSESSMENT FOR THROMBOEMBOLIC DISEASES IN PREGNANCY

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Pregnancy and the puerperium are well-established risk factors for deep vein thrombosis (DVT) and pulmonary embolism which are collectively referred to as venous thromboembolic disease. The need for thromboprophylaxis should be assessed antepartum, postpartum and at any time the patient transitions from the outpatient to the inpatient setting. When it is determined that thromboprophylaxis is warranted, an appropriate strategy should be selected and prescribed.

Thromboprophylaxis can be pharmacologic (anticoagulation) or mechanical (intermittent pneumatic compression devices or graduated compression stockings).

Pregnancy and the puerperium are risk factors for the development of venous thromboembolism. This risk is thought to be due to venous stasis of the lowerextremities, endothelial injury and the hypercoagulable state that occurs during pregnancy. The incidence of VTE is increased throughout all trimesters of pregnancy but is highest during the postpartum period. Factors that may further augment the risk include a prior history of VTE, hospitalization for an acute illness or cesarean delivery, and the presence of an inherited thrombophilia (factor V Leiden mutation, prothrombin gene mutation, or antithrombin III, protein C, or protein S deficiencie).

Pharmacologic prophylaxis may be considered in patients with a history of a single idiopathic, pregnancy-associated or estrogen-associated VTE, and in those with a history of multiple VTEs, regardless of the cause. Pharmacologic prophylaxis is also considered for patients with a known thrombophilia and in those with persistent risk factors and a prior history of VTE. The evidence to support pharmacologic thromboprophylaxis in the indicated populations is indirect, and largely based upon epidemiologic studies, small retrospective studies of thromboprophylaxis in high risk populations, and clinical experience. Nonetheless, studies consistently report the highest incidence of VTE in those with high risk variants of inherited thrombophilia and persistent risk factors in those with a prior history of VTE. Pregnant women who have had a prior VTE related to a high estrogen

state (prior pregnancy or estrogen-related VTE) are considered candidates for thromboprophylaxis

because these risk factors are likely to increase the chances of recurrent VTE during pregnancy. In contrast, for those women in whom a transient risk factor for prior VTE (trauma, immobility, surgery) is identified, the likelihood of recurrence is presumed to be lower. Thus, clinical surveillance is preferred over pharmacologic thromboprophylaxis for those without persistent risk factors, unless multiple VTEs have occurred.

Women who are already receiving anticoagulant therapy should have the need for ongoing therapeutic anticoagulation reassessed at the beginning of the pregnancy.

All postpartum women should be subjected to vigilant clinical surveillance for the signs and symptoms of VTE.

Data are insufficient to support routine outpatient pharmacologic thromboprophylaxis for most women in the postpartum period. Pharmacologic prophylaxis may be considered in patients with a history of prior VTE (single or multiple) regardless of the provoking factor (transient or persistent, inherited thrombophilia) and in a subset of patients with inherited thrombophilia without a personal or family history of VTE. The rationale for the use of thromboprophylaxis in the postpartum period is similar to that provided for the antepartum period However, the threshold for anticoagulation is lowered in the postpartum setting largely because the risk of VTE is increased.

تشخیص بیماری ترمبوآمبولی در حاملگی

مریم نورزاده | پریناتولوژیست

علائم بالینی PTE در حاملگی غیراختصاصی است و با حاملگی همپوشانی دارد. علائم DVT و PTE شامل تورم اندام تحتانی، تپش قلب، هموپتزی، تنگی نفس و درد سینه می‌باشد.

- **تشخیص DVT:** در صورت شک به DVT توصیه به شروع فوری درمان DVT با LMWH یا UFH همزمان با اقدامات تشخیصی می‌شود. جهت تشخیص باید اقدام به Comparing ultrasonography شود، اگر نتیجه CUS منفی شد، درصورتیکه بیمار High risk باشد با ادامه درمان باید اقدام به سونوگرافی داپلر عروق ایلیاک کرد. اما درصورتیکه بیمار Low risk باشد میتوان درمان را قطع کرد و CUS را در روز ۳ و روز ۷ تکرار کرد.

- **تشخیص PTE:**

ECG: غیراختصاصی است.

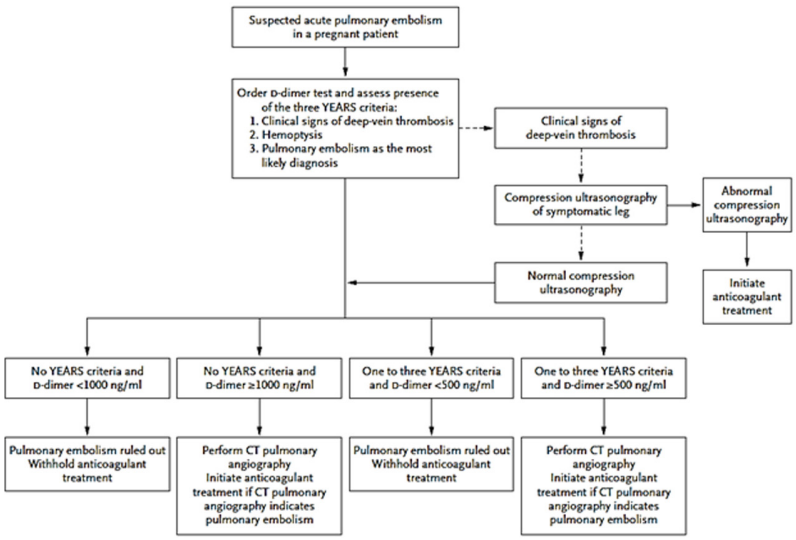
ABG نرمال رد کننده PTE نمی‌باشد.

CXR: تشخیصی نمی‌باشد.

V/Q scan: اقدام choice می‌باشد.

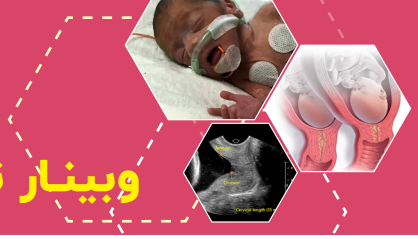
CTPA: از بالاترین sensivity و specifity برخوردار است.

الگوریتم تشخیص PTE



- **درمان:** جهت درمان می‌توان از LMWH یا UFH استفاده کرد. IV UFH با دوز 80mg/kg آغاز و سپس 18mg/kg ادامه می‌یابد و ۶ ساعت بعد APTT چک می‌شود. رنج درمان APTT=۱/۵-۲/۵ برابر نرمال است. دوز LMWH: 1 mg/kg q 12h می‌باشد.

ادامه درمان برحسب مورد بین ۳ تا ۶ ماه می‌باشد.



Inherited Thrombophilias in Pregnancy

Dr. Taraneh Arbabzadeh | perinatologist
Fellowship of perinatalogy, freelance

Abstract

Because there is insufficient clinical evidence that antepartum prophylaxis with UFH or LMWH prevents recurrence in women with a history of fetal loss or adverse pregnancy outcomes, screening for inherited thrombophilias is not recommended.

Among women with personal histories of VTE, recommended screening tests for inherited thrombophilias should include factor V Leiden mutation; prothrombin G20210A mutation; and antithrombin, protein S, and protein C deficiencies.

All patients with inherited thrombophilias should undergo individualized risk assessment, which may modify management decisions regarding VTE prevention.

Because of the lack of association between either heterozygosity or homozygosity for the MTHFR C677T polymorphism and any negative pregnancy outcomes, including any increased risk of VTE, screening with either MTHFR mutation analyses or fasting homocysteine levels is not recommended.

Warfarin, LMWH, and UFH do not accumulate in breast milk and do not induce an anticoagulant effect in the infant; therefore, these anticoagulants may be used in women who breastfeed.

Antiphospholipid syndrome (APS) and pregnancy

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Antiphospholipid syndrome (APS) is a systemic autoimmune disorder characterized by venous or arterial thrombosis and/or pregnancy loss in the presence of persistent antiphospholipid antibodies (aPL).

Pregnancy morbidity in antiphospholipid syndrome (APS) is defined by: ≥1 unexplained fetal deaths ≥10 weeks of gestation with normal morphology by prenatal ultrasound examination or direct postnatal examination

≥1 preterm deliveries of a morphologically normal infant before 34 weeks of gestation due to severe preeclampsia, eclampsia, or features consistent with placental insufficiency

≥3 unexplained, consecutive, spontaneous pregnancy losses <10 weeks of gestation, after exclusion of maternal anatomic and hormonal abnormalities and paternal and maternal chromosomal abnormalitie.

Nonpregnant women with a definite diagnosis of APS, based on laboratory criteria for aPL and a history of arterial or venous thrombosis, are at high risk of recurrent thrombosis and are generally treated with warfarin for an indefinite period that may be lifelong. We suggest a therapeutic dose of LMWH throughout pregnancy rather than prophylactic-dose LMWH.

We also prescribe low-dose aspirin (ASA) to reduce the risk of preeclampsia, whether or not they have a history of APS-defining pregnancy morbidity. For women with laboratory criteria for aPL and ≥1 fetal losses ≥10 weeks of gestation or ≥3 unexplained consecutive, spontaneous pregnancy losses <10 weeks of gestation but no history of venous or arterial thrombosis, we suggest combined therapy with low-dose ASA (50 to 100 mg per day) and prophylactic-dose LMWH rather than low-dose ASA alone. For women with laboratory criteria for aPL and ≥1 preterm deliveries of a morphologically normal infant before 34 weeks of gestation due to severe preeclampsia, eclampsia, or other findings consistent with placental insufficiency but no history of venous or arterial thrombosis, we suggest low-dose ASA therapy rather than no therapy or heparin. For pregnant women with the incidental finding of persistent aPL without meeting any of the clinical criteria for APS, we suggest low-dose ASA alone rather than no therapy. Our approach to postpartum venous thromboembolism prophylaxis depends on past medical and obstetric history, antepartum therapy, and route of delivery.

Aspirin and pregnancy

Dr.Nooshin-Eshraghi | perinatologist
perinatal flowship, Assistant professor Iran University Medicine

Low-dose aspirin (81 mg a day) is used as part of the management of antiphospholipid syndrome and for prevention of hypertension and preeclampsia as well ..

Candidates: It is reasonable to use the US Preventive Services Task Force (USPSTF) high risk criteria:

- Previous pregnancy with preeclampsia, especially early onset and with an adverse outcome
- Multifetal gestation
- Chronic hypertension
- Type 1 or 2 diabetes mellitus
- Chronic kidney disease
- Autoimmune disease (antiphospholipid syndrome, systemic lupus erythematosus)

offer low-dose aspirin for preeclampsia prevention with two or more of the following "moderate" risk factors:

- Nulliparity
- Obesity (body mass index >30 kg/m2)
- Family history of preeclampsia in mother or sister
- Age ≥35 years
- Sociodemographic characteristics (African American race, low socioeconomic level)
- Personal risk factors (eg, previous pregnancy with low birth weight or small for gestational age infant, previous adverse pregnancy outcome [eg, stillbirth], interval >10 years between pregnancies)

Timing of initiation: low dose aspirin for preeclampsia prevention at ≥12 weeks of gestation, and ideally prior to 16 weeks. Early therapy (before 16 weeks) may be important since the pathophysiologic features of preeclampsia develop early in pregnancy, weeks before clinical disease is apparent. However, possibly because aspirin has major effects on prostacyclin production and endothelial function throughout gestation. If aspirin is not initiated at the end of the first trimester, initiation after 16 weeks (but before symptoms develop) may also be effective.