DOWNLOAD PDF THROMBOEMBOLIC DISORDERS:

Chapter 1 : Thromboembolic Disease in Pregnancy

Warfarin is an oral anticoagulant used for treatment and prevention of venous thromboembolism and prevention of cardioembolic stroke. It was first synthesized as a rodenticide in and was FDA approved for human use in

Thrombosis is the process of formation of a fibrin blood clot thrombus. An embolus is a small fragment of a thrombus that breaks off and circulates until it becomes trapped in a capillary, causing ischemia or infarction to the area distal to the obstruction e. Major causes of thrombus formation are immobilization with venous stasis; surgery and the postoperative period; trauma to lower limbs; certain illnesses e. Normally, blood clot formation and dissolution constitute a fine balance within the cardiovascular system. The clotting proteins normally circulate in an inactive state and must be activated to form a fibrin clot. When there is a trigger, such as increased blood viscosity from bed rest and stasis or damage to a blood vessel wall, the clotting cascade is activated. Activated factor Xa, in the presence of calcium, PF3, and factor V, stimulates the conversion of prothrombin to thrombin Figure F, site of fibrinolytic action; Fu, fondaparinux; H, site of heparin action; LMWH, site of low-molecular-weight heparins; PF3, platelet factor 3; W, site of warfarin action. Sources outside the blood vessels, such as tissue extract or thromboplastin tissue factor , can trigger the extrinsic clotting pathway by activating factor VII to VIIa. Factor VIIa can also activate factor X, which results in the formation of thrombin. After stimulation from the intrinsic or extrinsic pathway, thrombin, in the presence of calcium, activates fibrinogen to soluble fibrin. With time and the presence of factor XIII, the loose fibrin mesh is converted to a tight, insoluble fibrin mesh clot. As the fibrin clot is being formed, it also triggers the release of fibrinolysin, an enzyme that dissolves fibrin, preventing the clot from spreading. Historically, thrombi have been classified into red and white blood clots. A red thrombus is actually a venous thrombus and is composed almost entirely of fibrin and erythrocytes red blood cells , with a few platelets. Venous thrombi generally form in response to venous stasis after immobility or surgery. The poor circulation prevents dilution of activated coagulation factors by rapidly circulating blood. The most common cause of red thrombus formation is deep vein thrombosis of the lower extremities. These thrombi may extend upward into the veins of the thigh and have the potential of fragmenting, subsequently causing life-threatening pulmonary emboli. White thrombi develop in arteries and are composed of platelets and fibrin. This type of thrombus forms in areas of high blood flow in response to injured vessel walls. Coronary artery occlusion leading to myocardial infarction is an example of a white thrombus.

Chapter 2 : Thrombotic Disorders: Diagnosis and Treatment

About Thrombotic/Thromboembolic Disorder: Thromboembolic Disorder is a condition in which the formation of a blood clot (thrombus) inside one of the blood vessels is carried by the blood from the site of origin to block another vessel.

Thromboembolic Disorders Jun 07, Viewed: In the United States, one half of all thromboembolic events in women younger than 40 years are related to pregnancy. The risk of deep vein thrombophlebitis DVT during pregnancy has been reported to be five times higher than in nonpregnant individuals and is probably even higher in the immediate postpartum period. The risk of pregnancy-associated thromboembolism is further increased in patients with prior DVT or Pulmonary embolism PE , advanced maternal age, multiparity, prolonged bed rest, varicose veins, or obesity, as well as those with a variety of inherited or acquired coagulation disorders. Postpartum risks are increased by cesarean delivery a fourfold to tenfold increase over vaginal delivery. The diagnosis of deep vein thrombophlebitis or Pulmonary embolism may be suspected clinically but usually requires radiographic confirmation. Noninvasive methods include high-resolution compression ultrasonography coupled with color Doppler imaging and impedance plethosmography to assess for DVT, as well as 99Tc ventilation-perfusion lung scan to assess for PE. The only examination contraindicated during pregnancy is I-fibrinogen scanning, because the dose of radiation to the fetal thyroid is clinically significant. Initial treatment of acute venous thrombosis and Pulmonary embolism requires intravenous heparin given in a high dosage sufficient to prolong the activated partial thromboplastin time PTT 1. The heparin requirements to treat acute venous thrombosis seem to be greater during pregnancy than in the nonpregnant state. In addition, failure to reach adequate anticoagulation in the first 24 hours of treatment greatly increases the risk of recurrent thrombosis. Intravenous treatment is usually continued for 5 to 10 days to allow for organization and firm attachment of the thrombus to the vessel wall. The patient then begins self-injected, adjusted high-dose subcutaneous heparin every 8 to 12 hours. The dose is determined by dividing the hour intravenous required dose into two to three subcutaneous injections daily. The goal again is to maintain the midinterval PTT at 1. Alternatively, continuous infusion of heparin can be arranged for the patient. Anticoagulation is continued throughout pregnancy and for approximately 3 months postpartum. Though heparin is the anticoagulant of choice during pregnancy, heparin-induced thrombocytopenia and osteoporosis are important adverse effects that must be watched. There are insufficient data to conclude which regimen provides the safest maximal protection against recurrent antepartum thrombosis. Antepartum thromboembolism prophylaxis should be considered, however, in pregnant women with a prior history of DVT or PE; women with antithrombin III, protein C, or protein S deficiency; and women with the antiphospholipid antibody syndrome. Unlike heparin, warfarin freely crosses the placenta. Use of warfarin during the first trimester has been associated with a variety of neonatal malformations, including nasal hypoplasia, frontal bossing, and short stature with stippled epiphyses. Use during the second and third trimesters has been associated with central nervous system abnormalities and fetal and placental hemorrhage resulting in fetal death. Warfarin should be avoided throughout pregnancy, and patients already taking warfarin and desiring to conceive should be switched to heparin before conception. Medical Disorders During Pregnancy.

Chapter 3 : Browse or Search Notecards (Flashcards) | Easy Notecards

Thromboembolic disease is the leading cause of nonobstetric postpartum maternal mortality. In the United States, one half of all thromboembolic events in women younger than 40 years are related to pregnancy.

Primary hypercoagulable states thrombophilias. The growing list of established thrombophilias reflects our increasing understanding of the genetic basis of thrombosis but, ironically, it is also creating increasing uncertainty about how to use this information for diagnosis and management in clinical practice. A genome-wide linkage screen has identified a distinct region on chromosome 1q, genetic variability of which influences the level of free protein S in plasma. For example, another genome-wide linkage screening has found that polymorphisms in the ABO blood group genotype are major genetic determinants of plasma levels of von Willebrand factor, increases in which are associated with thrombotic risk. Individuals with inherited thrombophilias have a higher risk of VTE in the setting of acquired thrombogenic events than those without identifiable genetic defects. One study, for example, showed that the prevalence of the prothrombin-gene mutation was significantly higher in patients with venous thrombosis than in healthy controls odds ratio of about Likewise, the use of oral contraceptives was more frequent among women with thrombosis than among controls odds ratio, For women who were taking oral contraceptives and also had the prothrombin-gene mutation, the odds ratio for thrombosis rose dramatically to Possibly all patients with VTE have a genetic predisposition in the form of one or more thrombophilias. The level of lifelong, baseline hypercoagulability in any individual may be determined by the type s and number of thrombophilia s that are inherited. Likewise, it is possible that all episodes of VTE are precipitated by acquired thrombogenic triggers. Thus, in an individual with a relatively low level of baseline genetic hypercoagulability e. Thus, the precipitating event in such individuals is often clinically overt. In most cases, such thrombophilic individuals never suffer VTE throughout their lifetimes, and when they do have an episode it is unlikely to recur. In contrast, an individual with a high level of baseline genetic hypercoagulability with multiple thrombophilic mutations or polymorphisms is at such high risk that relatively minor acquired triggers can initiate a thrombotic episode. Furthermore, VTE in these high-risk individuals is more likely to recur. Thrombosis in Patients with Cancer Mark N. Tumors, through expression of tissue factor, can activate coagulation. This article focuses on the treatment of VTE in cancer patients. Treatment of cancer patients with VTE is difficult because these patients have an increased risk of both recurrent VTE and anticoagulant-induced bleeding compared with noncancer patients. In some instances of end-stage cancer, there is the difficult decision of whether one should even treat the acute thrombotic event. Initial Treatment of Venous Thromboembolism Based on the results of numerous randomized controlled trials, low-molecular-weight heparin LMWH has replaced unfractionated heparin UFH as the first-line treatment in the majority of patients with acute deep vein thrombosis DVT. Large meta-analyses of these clinical trials have shown that weight-adjusted subcutaneous LMWH is safer and probably more effective than UFH administered by continuous intravenous IV infusion and monitored by the activated partial thromboplastin time aPTT. Nonetheless, it would seem reasonable to generalize the results of these trials to cancer patients with acute VTE. In terms of optimizing treatment, the use of LMWH avoids IV administration of anticoagulant therapy and the need for laboratory monitoring, thereby improving the quality of life of the patient. There have been three clinical trials that demonstrated that patients with acute proximal DVT could be treated safely at home with subcutaneous LMWH without hospital admission. Clearly, some patients with acute VTE will require hospitalization because of symptoms and other complications related to their cancer. If patients are to be treated at home, they must be reliable and compliant and have a good support system. The use of inferior vena caval IVC filters reduces the short-term risk of PE but is associated with an increased long-term risk of recurrent DVT, despite concurrent oral anticoagulant therapy. In a large randomized trial conducted in France, in which patients with proximal DVT were treated with anticoagulant therapy and randomized to receive an IVC filter or not, there was a statistically significant reduction in PE during the first 2 weeks of treatment. This was likely a result of thrombosis that developed around and proximal to the filter. Filters should be reserved for cancer patients who are actively bleeding and cannot

receive anticoagulant therapy and for patients who develop multiple episodes of recurrent thromboembolism despite therapeutic LMWH. This would avoid the long-term potential complications of IVC filters. However, the results of additional studies on cancer patients are required. Long-Term Anticoagulant Therapy Long-term anticoagulant therapy using coumarin derivatives is required to prevent recurrent thrombosis. An oral anticoagulant such as warfarin is commenced on the first or second day of heparin treatment, and the aim is to achieve an International Normalized Ratio INR of between 2. Warfarin therapy is particularly complicated in the cancer patient for a number of reasons. It is often difficult to maintain the INR within the therapeutic range because cancer patients suffer from anorexia and vomiting. They may have chronic disseminated intravascular coagulation or extensive hepatic metastases. In addition, drug interactions e. Often it is necessary to frequently interrupt oral anticoagulant therapy because of thrombocytopenia and procedures such as thoracentesis and abdominal paracentesis. Finally, frequent blood sampling is required for the INR, and accessing veins can often be difficult in the cancer patient. There are certain features of long-term anticoagulant therapy with LMWH that are attractive in the cancer patient. LMWH does not require laboratory monitoring and can be administered once or twice daily, subcutaneously, based on body weight. There is the clinical experience that LMWH can be effective in patients who develop recurrent thrombosis despite therapeutic warfarin therapy. There have been a number of trials that have compared long-term oral anticoagulant therapy with long-term LMWH. No definitive conclusions can be drawn from these trials concerning long-term treatment with LMWH in the cancer patient. Several recent randomized trials, however, have provided new information concerning the long-term treatment of cancer patients with VTE. In the trial reported by Meyer et al, cancer patients with acute VTE were randomized to 3 months of enoxaparin or warfarin at a targeted INR of 2. This observed difference was mainly as a result of the rates of major bleeding in the 2 groups; The probability of VTE at 6 months was reduced from No statistically significant difference was detected in major bleeding between groups, which occurred in 3. Finally, in a subgroup analysis of a trial that compared long-term tinzaparin LMWH with oral anticoagulant therapy, both administered for 3 months, there was a statistically significant reduction in recurrent VTE in the subgroup of cancer patients. It substantially reduces the rate of recurrent VTE without an increase in bleeding, thereby improving the quality of life of the cancer patient. Duration of Long-Term Treatment Clinical trials have shown that longer-duration oral anticoagulant therapy is associated with lower rates of recurrent thromboembolism compared with shorter-duration treatment. Hence, anticoagulant therapy should be continued for as long as the cancer is active. In patients with metastatic disease, anticoagulant therapy should be continued indefinitely or until a contraindication to therapy develops. In patients with nonmetastatic disease, treatment should be for at least 6 months or for as long as the patient is on chemotherapy or hormonal therapy. Catheter-Related Thrombosis Long-term indwelling central venous catheters are commonly used in cancer patients. One of the complications related to central venous catheters is catheter-associated venous thrombosis. This can involve the catheter tip ball-valve clot , the length of the catheter fibrin sheath , the catheterized vessel in the upper limb, the central vasculature of the neck or mediastinum, or a combination of these sites. The incidence of catheter-related thrombosis varies among studies. Four randomized studies have been performed to evaluate the safety and efficacy of prophylactic anticoagulation in patients with central venous catheters. Venography was performed at 90 days or sooner if patients had symptoms suggestive of thrombosis. In the study by Monreal et al, cancer patients with central venous catheters were randomized to dalteparin IU subcutaneously once daily or no prophylaxis. Two more recent randomized trials reported low rates of thrombosis and no effect of prophylactic anticoagulant therapy. Reichart et al randomized cancer patients with central venous catheters to 16 weeks of dalteparin or placebo. The reasons for the much lower rates of catheter-related thrombosis reported in recent trials are uncertain, but improved catheter material and advances in insertion techniques may contribute. Currently, it is not clear whether cancer patients with central venous catheters should receive antithrombotic prophylaxis. Additional randomized trials are required. Treatment of central venous catheterâ€”related thrombosis remains a controversial and poorly studied area. Currently, cancer patients with symptomatic central venous catheterâ€”related thrombosis are treated with anticoagulant therapyâ€”that is, initial LMWH or UFH followed by long-term oral anticoagulant therapy. Routine removal of the catheter when there is a

catheter-associated thrombosis is a controversial subject. However, if the patient has a continued need for central venous access, a functioning catheter does not have to be removed. Often, routine removal of the catheter is not practical because reinsertion of another catheter is difficult and can be associated with considerable morbidity. Conclusion There have been many advances in the management of cancer patients with acute VTE. The use of long-term LMWH instead of oral anticoagulants can substantially reduce the risk of recurrent VTE in this high-risk group of patients without increased bleeding. A number of novel agents that target specific coagulation proteases are currently undergoing investigation for both the prevention and the treatment of VTE. Women referred to hematologists for thrombosis-related questions often present complex clinical scenarios. In this section, using a patient case presentation, we will review the currently available data on which to make our recommendation. Patient Presentation A year-old female is referred for consultation by her obstetrician. She is early in her third pregnancy. She has a history of polycystic ovarian disease and required hormonal ovarian stimulation for conception of her first 2 children, but not for the current pregnancy. Two years earlier, during her last pregnancy, she developed right arm and neck swelling 7 weeks after insemination and she was diagnosed with a right jugular, innominate and subclavian vein thrombosis. She was treated with heparin during the pregnancy, followed by warfarin post-partum. A thrombophilia evaluation demonstrated heterozygosity for prothrombin GA. She now presents for advice on management of this pregnancy. Assisted Reproductive Treatment and Thrombotic Risk Thrombosis has been described as a risk with these treatments, particularly in association with ovarian stimulation. Thrombosis is more common in the setting of ovarian hyperstimulation syndrome OHSS and following a treatment cycle that results in pregnancy. Most were not associated with venous catheters. Although there may be some reporting bias in publishing cases of upper extremity thrombosis given its rarity, these reports suggest that upper extremity thrombosis is not an uncommon site of thrombosis in this clinical situation. Twenty-five percent of patients in this report suffered arterial thromboses, most commonly intracerebral. Why these individuals develop thrombosis is not clear. The hormonal stimulation used typically results in estradiol levels up to 10 times the normal levels. Coagulation studies have been limited but generally show increased coagulation factors, particularly fibrinogen and factor VIII, and decreased fibrinolysis. Underlying thrombophilias generally have not been detected in these women, although most patients reported were not tested for the common thrombophilias, including factor FV Leiden and prothrombin GA. However, of 54 cases reviewed by Stewart et al, 1 6 had a history of prior thrombosis and 2 had a strong family history of thrombosis. Given the risks, it seems prudent to institute prophylaxis in a woman with a documented thrombophilia or a history of prior thrombosis who is to receive assisted reproductive treatment, although the optimal drug and dosing regimen is unknown. The risk is higher after cesarean section than after a vaginal delivery, including a fold higher risk of fatal pulmonary embolism PE.

DOWNLOAD PDF THROMBOEMBOLIC DISORDERS:

Chapter 4 : 4: Thromboembolic disorders | Nurse Key

Medical Definition of Thromboembolism Thromboembolism: Formation in a blood vessel of a clot (thrombus) that breaks loose and is carried by the blood stream to plug another vessel. The clot may plug a vessel in the lungs (pulmonary embolism), brain (stroke), gastrointestinal tract, kidneys, or leg.

Thrombophilia Hypercoagulability or thrombophilia, is caused by, for example, genetic deficiencies or autoimmune disorders. Recent studies indicate that white blood cells play a pivotal role in deep vein thrombosis, mediating numerous pro-thrombotic actions. The main mechanism is exposure of tissue factor to the blood coagulation system. In addition, arterial and cardiac clots are normally rich in plateletsâ€”which are required for clot formation in areas under high stress due to blood flow. Blood flow Causes of disturbed blood flow include stagnation of blood flow past the point of injury, or venous stasis which may occur in heart failure , [19] or after long periods of sedentary behaviour, such as sitting on a long airplane flight. Also, atrial fibrillation , causes stagnant blood in the left atrium LA , or left atrial appendage LAA , and can lead to a thromboembolism. For an occlusive thrombus defined as thrombosis within a small vessel that leads to complete occlusion , wound healing will reorganise the occlusive thrombus into collagenous scar tissue, where the scar tissue will either permanently obstruct the vessel, or contract down with myofibroblastic activity to unblock the lumen. For a mural thrombus defined as a thrombus in a large vessel that restricts the blood flow but does not occlude completely , histological reorganisation of the thrombus does not occur via the classic wound healing mechanism. Instead, the platelet-derived growth factor degranulated by the clotted platelets will attract a layer of smooth muscle cells to cover the clot, and this layer of mural smooth muscle will be vascularised by the blood inside the vessel lumen rather than by the vasa vasorum. A venous thrombus may or may not be ischaemic, since veins distribute deoxygenated blood that is less vital for cellular metabolism. Nevertheless, non-ischaemic venous thrombosis may still be problematic, due to the swelling caused by blockage to venous drainage. In deep vein thrombosis this manifests as pain, redness, and swelling; in retinal vein occlusion this may result in macular oedema and visual acuity impairment, which if severe enough can lead to blindness. Embolus A thrombus may become detached and enter circulation as an embolus , finally lodging in and completely obstructing a blood vessel, which unless treated very quickly will lead to tissue necrosis an infarction in the area past the occlusion. Venous thrombosis can lead to pulmonary embolism when the migrated embolus becomes lodged in the lung. In people with a "shunt" a connection between the pulmonary and systemic circulation , either in the heart or in the lung, a venous clot can also end up in the arteries and cause arterial embolism. The tissue can become irreversibly damaged, a process known as necrosis. This can affect any organ; for instance, arterial embolism of the brain is one of the cause of stroke. Thrombosis prevention The use of heparin following surgery is common if there are no issues with bleeding. Generally, a risk-benefit analysis is required, as all anticoagulants lead to an increased risk of bleeding. In patients admitted for surgery, graded compression stockings are widely used, and in severe illness, prolonged immobility and in all orthopedic surgery , professional guidelines recommend low molecular weight heparin LMWH administration, mechanical calf compression or if all else is contraindicated and the patient has recently suffered deep vein thrombosis the insertion of a vena cava filter. Anticoagulant Warfarin and vitamin K antagonists are anticoagulants that can be taken orally to reduce thromboembolic occurrence. Where a more effective response is required, heparin can be given by injection concomitantly. As a side effect of any anticoagulant, the risk of bleeding is increased, so the international normalized ratio of blood is monitored. Self-monitoring and self-management are safe options for competent patients, though their practice varies. This carries an increased risk of bleeding so is generally only used for specific situations such as severe stroke or a massive pulmonary embolism.

Chapter 5 : Thrombosis - Wikipedia

Quantitative latex assay was used to measure D-dimers, which reaches sensitivity of greater than 95% for detection of

thromboembolic disease, with a cut-off value of ug/L.

Chapter 6 : Thromboembolic Disorder Assessment: DVT/PE

Thrombosis (from Ancient Greek Î¸Ï•ÏŒÎ¼Î²Ï‰ÏƒÎ¹Ï, thrÃ³mbÅ•sis "clotting") is the formation of a blood clot inside a blood vessel, obstructing the flow of blood through the circulatory system.

Chapter 7 : Thromboembolic Disorder Treatments: Without DVT/PE - MPR

In thromboembolic disorders, blood clots (thrombi) form in blood vessels. An embolus is a blood clot that travels through the bloodstream and blocks an artery. In the United States, thromboembolic disorders are a common cause of death in pregnant women.

Chapter 8 : Thromboembolic Disorder Treatments: Without DVT/PE

A comparative medication chart of thromboembolic disorder treatments in patients without DVT/PE.

Chapter 9 : Thromboembolic Disorder Treatments: DVT/PE - MPR

Pulmonary Embolism Explained Clearly - Risk factors, Pathophysiology, DVT, Treatment - Duration: MedCram - Medical Lectures Explained CLEARLY , views.