UDK 616.14-005.

SPECIAL ARTICLE-CLINICAL PRACTICE

Risk Factors for Venous Thromboembolism and Duration of Anticoagulation Therapy

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ABSTRACT

An adequate regimen for prophylaxis of venous thromboembolism (VTE) requires indentification of reversible and irreversible risk factors. Recent data confirm that the greatest number of pulmonary emboli (PE) occur in non-surgical patients. VTE also develops in many surgical patients upon hospital discharge. These findings emphasize the need for adequate VTE prophylaxis in inflammatory diseases, acute medical illness, and other conditions, as well as the need to optimize anticoagulant regimens after surgery. Establishing VTE risk factors, identifying acquired or inherited thrombophylias and occult or previously undiagnosed malignancy will help design an adequate anticoagulant regimen as secondary VTE prophylaxis for surgical and other patients. Follow up measures should include D-dimer values, ultrasonographic assessment of residual venous thrombosis and echocardiographic parameters, along with other relevant clinical data to assess the risk of VTE reoccurrence. These procedures will ensure the optimal duration of individually tailored anticoagulant therapy, with special attention to comorbidities and tendency to hemorrhage.

KEY WORDS

Venous thromboembolism, thromboprophylaxis, recurrent thrombosis, risk for bleeding.

DOI: 10.7251/SMD1301035A *(Scr Med 2013;44:35-42)*

Submitted: October 13, 2012
Accepted: January 17, 2013

Venous thromboembolism (VTE) is the third leading vascular cause of mortality after myocardial infarction and cerebrovascular insult.[1] It is the most preventable disorder. Thus improving measures to prevent VTE remains a top clinical priority.[2] Over two-thirds of all symptomatic VTE occur in patients that were not subjected to surgical procedures.[3] It was reported that 47-76% of all clinical VTE events after hip and knee surgery occur after hospital discharge, and it is recommended to extend VTE prophylaxis in such patients.[3,4]

Although venous thromboembolism is often present in surgical patients during the postoperative period, 70-80% of fatal pulmonary emboli (PE) develop in nonsurgical hospital patients. In 40% of such cases, an age factor is associated with other risk factors, such as previous VTE, malignancy, cerebrovascular accident, heart failure, chronic obstructive pulmonary disease, sepsis and immobilization or confinement to bed.[5] The incidence of venous thromboembolism increases with age, ranging between 1/10,000 per year in younger patients and 5-6/1,000 per year in people over 80 years.[5] An increase in VTE-related morbidity correlates with a number of associated comorbidities, such as inflammatory conditions, elevated acute-phase reactants and reduced anticoagulant proteins.[5]

Prevention and treatment of VTE requires key decisions for further management. These include determining the duration of anticoagulant treatment, selection of measures to prevent recurrent venous thromboembolism and VTE sequelae (pulmonary hypertension, post-thrombophlebitic syndrome) as well as appropriate diagnostic screening for thrombophilia and occult malignancy, along with defining reversible and irreversible risk factors for VTE.[6] A number of authors give priority to establishing optimal anticoagulant treatment over detecting possible congenital thrombophilic states that indicate clinical risk factors for VTE.[6]

Classification of risk factors for VTE

The various classifications and categorizations of risk factors for VTE are a mainstay for tailoring the optimal treatment of VTE patients to their individual characteristics.[7]

For example, Kaatz's categorization of VTE is particularly useful: VTE provoked by risk factors, cancer-related, idiopathic, thrombophilia-related and recurrent VTE.[7] Another classification is based on the strength of risk factors for VTE: **strong** risk factors for VTE with odds ratio > 10 include trauma or fracture, major orthopedic surgery, oncology surgery; **moderate** risk factors with odds ratio 2-9 include non-oncology surgery, use of oral contraceptives and hormone replacement therapy, pregnancy and puerperium, hypercoagulability state and previous VTE; **weak** risk factors with odds ratio <2 include advanced age, bed confinement for longer than three days, imobility on long trips, metabolic syndrome and air pollution.[8] The life-style or disease-associated risk factors for arterial and venous thromboembolism include obesity, diabetes mellitus, hypertension and smoking,[8] with special consideration given to the impact of dyslipidemia on VTE occurence. (Commonly known VTE risk factors[4-10] are presented in Table 1.)

Table 1. Risk factors for VTE

Surgery, trauma (major or lower limb trauma)
Immobility, lower limb paresis
Malignancy (active or occult), malignancy therapy (hormonal, chemotherapy, radiation, treatment with angiogenesis inhibitors)
Previous VTE
Use of estrogen-containing contraceptives, hormone-replacement therapy, or selective estrogen receptor modulators, agents stimulating erythropoiesis
Acute medical illness
Inflammatory bowel disease, nephrotic syndrome, myeloproliferative disease, chronic obstructive pulmonary disease, congestive heart failure, paroxysmal nocturnal hemoglobinuria
Dehydration, transfusion
Venous compression (tumor, hematoma, arterial abnormalities)
Obesity, advanced age
Pregnancy and puerperium
Congenital or acquired thrombophilia
Application of central venous catheters

Risk factors for VTE often overlap with those for coronary heart disease (smoking, obesity, high consumption of red meat instead of a healthier diet of fish, fruit and vegetables, psychosocial stress, hypertension). The JUPITER study (Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin) provides a convincing example of how risk factors for arterial and venous thromboembolism converge. Results of that study showed a reduction in VTE by 43% in the group treated with rosuvastatin (20 mg daily) compared to the control (untreated) group.[9]

Bauer and Previtali[8,10] group the VTE risk factors into **acquired** (including antiphospholipid syndrome, myeloproliferative neoplasms, paroxysmal nocturnal hemoglobinuria, inflammatory bowel disease, Wegener granulomatosis, paresis/paralysis of lower extremities, etc.), **inherited** (deficiencies of antithrombin, proteins C and S, factor V Leiden and prothrombin G20210A mutations, disfibrinogenemias) and **mixed** risk factors (hyper-homocysteinemia, resistance to activated protein C in the absence of factor V Leiden mutation, increased activity of factors VIII, IX, XI, thrombin-activated fibrinolysis inhibitor (TAFI), reduction of tissue factor pathway inhibitor (TFPI), and fibrinolytic activity).

Patients heterozygous for factor V Leiden have a three times higher risk for an initial VTE while homozygous individuals carry a 15-20-fold increased risk.[9] Goldhaber considers the combination of homozygous factor V Leiden mutation, double heterozygotes for factor V Leiden and prothrombin G20210A mutation, deficiencies of proteins C, S and antithrombin as well as antiphospholipid syndrome to be a particularly ominous setting for thrombophilia.[9]

Although estrogens in the form of oral contraceptives or postmenopausal hormone therapy are well-known risk factors for VTE, it is interesting to note that the third-generation progestins, dezogestrol and gestodene also rank among risk factors for VTE. [9]

In order to optimize an anticoagulation regimen for patients with VTE, one should establish the importance of their specific VTE risk factors by distribution into these

three categories: 1. Reversible – major (occuring a month after surgery where general anesthesia lasted longer than 30 minutes, hospitalization longer than three days, plaster immobilization of lower limbs); 2. Reversible - minor (hormone replacement therapy, pregnancy and puerperium, an eight-hour or longer flight or travel in a sitting position [a stricter limitation is 4-6 hours] or the presence of major reversible risk factors during 1-3 months); 3. Nonreversible or permanent risk factors for VTE (malignancy, molecular thrombophilias).[11]

Long-term anticoagulant therapy is strongly recommended for patients with persistent nonreversibile risk factors, such as homozygous mutation of factor V Leiden, double heterozygotes for factor V Leiden and prothrombin G20210A mutations, protein C/S deficiency, antiphospholipid antibodies.[11] Interruption of anticoagulant therapy constitutes a greater risk for recurrent VTE in patients with previous proximal deep vein thrombosis (DVT) compared with the distal lower limb DVT.[12]

Agnelli classifies risk factors for VTE as **transient** (surgery, trauma, immobilization) or **persistent** (cancer and paralysis), but considers those individuals with idiopathic and spontaneous VTE to have no identified risk factors for thrombosis. Numerous studies identifed male gender as a risk factor for recurrent VTE [relative risk (RR) 1,6; 95% confidence interval (CI) 1,2-2,0].[13] The observation that the risk of fatal pulmonary embolism is two-three times greater after an episode of PE than after a DVT episode is also of clinical relevance.[13]

Classification based on risk for recurrent VTE

Prandoni[14] defines several groups of risk factors for recurrent VTE: 1) persistent acquired risk factors (active malignancy, especially with metastasis and treated with chemotherapy, patients with chronic nonsurgical diseases who are immobilized for long periods of time), 2) major transient risk factors (previous surgery or trauma), 3) minor transient risk factors (minor trauma, long-haul flights, estrogen therapy, pregnancy and puerperium), 4) spontaneous VTE, 5) congenital thrombophilias (with special emphasis on the deficiencies of proteins C and S and antithrombin, increase of factors VIII and IX, hyper-homocysteinemia). Although recent studies associating recurrent VTE with homozygous factor V Leiden and prothrombin 20210 remain controversial, it is indisputable that patients on one-year anticoagulation therapy regimens have a lower percentage of recurrent VTE than those on conventional three-month anticoagulation regimens. Lowering homocysteine levels with vitamin B12 supplementation does not reduce the risk of recurrent VTE. Prophylaxis of VTE in pregnancy must not be discontinued before the end of the puerperium (6 weeks after childbirth).

Patients with significant transient risk factors should be treated for three months, but the duration of treatment could be less (six weeks) if thrombosis is localized to veins in the lower legs. Patients with minor transient risk factors require treatment tailored to the degree of hemorrhagic risk for each individual.[15] Indefinite anticoagulant treatment is recommended for patients with multiple episodes of VTE. This might include an implanted vena cava filter if anticoagulants are contraindicated as well as for individuals with antiphospholipid syndrome.[14]

Table 1 lists the most important risk factors for recurrence of VTE upon anticoagulant therapy discontinuation.[15] Prospective studies in patients with VTE indicate a greater risk of recurrent VTE in patients who have high levels of D-dimer a month after termination of anticoagulant therapy.[13] Identification of these patients by D-dimer monitoring can single out those at greatest risk and help to prevent recurrent VTE.[14] In the PREVENT trial (Prevention of Recurrent Venous Thromboembolism), one group of patients with spontaneous VTE received anticoagulant (warfarin) therapy for six months, but measurement of D-dimer for seven weeks following warfarin withdrawal showed that those with increased D-dimer levels had a twofold higher recurrence rate.

Table 2. Risk factors for recurrent VTE[15]

When anticoagulation therapy has already been administered
Advanced Age
Immobilization
Malignancy
Chronic obstructive pulmonary disease
Enlargement or dyskinesia of right heart ventricle
After the termination of anticoagulant therapy
Male gender
Body overweight
Signs and symptoms of PE before DVT
Low levels of HDL
Absence of recanalization of lower limbs veins on ultrasound scan

However, a meta-analysis of 1888 patients with spontaneous VTE suggests that the problem is not that simple. That study reported that 3.5% of patients have an annual risk for recurrent VTE despite normal D-dimer levels measured upon discontinuation of anticoagulant therapy.[15] In a separate meta-analysis of idiopathic VTE studies, the recurrence rate was 7.2% for patients who had normal D-dimer values measured one month after discontinuation of anticoagulant therapy. Some reports suggest that elevation of D-dimer one or two months after therapy is associated with significant risk of spontaneous recurrent thrombosis [hazard ratio 2.0, 95% confidence interval (CI) 1.01 to

3.9].[16] The risk of recurrent VTE is 10% per year in men who have had spontaneous VTE with elevated D-dimer, whereas the risk of spontaneous VTE, or VTE caused by defined factors with negative D-dimer, in women is about 2%. Consequently, the benefit of long-term anticoagulant therapy in these women remains vague. [16]

According to Agnelli, the main predictors of recurrent thromboembolism are D-dimer levels and the presence of residual thrombosis.[13] The hazard ratio for recurrent VTE was 2.4 in patients with persistent residual thrombosis (shown by venous ultrasonography) compared with those who had vein recanalization.[13] The same author notes that recurrent VTE can be as high as 29% in patients positive for anti-cardiolipin antibodies after a first episode of VTE compared with 14% of those without the antibodies ($p < 0.01$). The PREVENT study established the efficacy of prolonged anticoagulant therapy in patients with factor V Leiden and prothrombin G20210A mutations by showing that the annual incidence of recurrent VTE was reduced from 8.6% to 2.2% per year.[13]

The Vienna Prediction Model for Recurent VTE identifies the risk of recurrent VTE in relation to sex, clinical presentation and laboratory values of D-dimer.[14,17] Besides the aforementioned risk factors, this model indicates that a number of other abnormalities can be involved, including elevated factor VIII, factor IX, increased hematocrit, low levels of apolipoprotein AI, HDL and vitamin B6, and FSAP Marburg (Marburg I polymorphism of factor VII activating protease), overweight, pregnancy and puerperium, even chronic renal disease, are associated with increased risk for recurrent VTE.[17-27]

Duration of anticoagulant therapy for secondary prophylaxis depends upon the category of VTE

According to Goldhaber,[9] the recommended duration of anticoagulant therapy for a first attack of PE and/or DVT related to an identified risk factor for VTE (surgery, trauma, oral contraceptives, pregnancy, hormone replacement therapy) is from three to six months with a target International Normalized Ratio (INR) 2-3. For patients with a first episode of upper limb DVT or isolated lower leg DVT with identified risk factors, a three-month course of anticoagulant therapy with an INR of 2-3 is advised. For a second attack of VTE provoked by an identified risk factor, most clinicians recommend doubling the duration of anticoagulant therapy; a few of them favor so-called lifelong anticoagulation therapy, or indefinite treatment. The ACCP (American College of Chest Physicians), NCCN (National Comprehensive Cancer Network) and ASCO (American Society of Clinical Oncology) reached a consensus that patients with malignancies should be treated with low-molecular-weight heparin (LMWH) during the first from three to six months and then indefinite anticoagulation therapy (vitamin K antagonists or LMWH). Table 3 shows the recommended duration of anticoagulant therapy for secondary prophylaxis of VTE[15]

Table 3. Optimal duration of therapy for secondary prophylaxis of VTE[15]

CATEGORY OF VTE	GUIDELINES FOR DURATION OF ANTICOAGULANT REGIME
First episode of PE or proximal DVT related to an identified risk factor	3-6 months
First episode of upper-limb DVT or isolated lower-leg DVT related to an identified risk factor	3 months
Second episode of DVT related to an identified risk factor	Uncertain
Third episode of DVT	Indefinite duration
DVT in malignancy	Indefinite duration until malignancy is resolved
Spontaneous PE/ proximal DVT of lower limb	Consider indefinite duration
First unprovoked DVT of calf	3 months
Second unprovoked DVT of calf	Uncertain

Aggressive use of anticoagulant therapy after the first six months of treatment remains debatable. Many physicians continue the standard anticoagulant regimen with a target INR 2-3, whereas others consider a low-intensity anticoagulant regimen with a target INR 1.5-2 to be effective and safe.[15] Three studies achieved a 90% risk reduction in patients with standard anticoagulation therapy and a target INR of 2.5 (range 2-3) with the extended regimen, while a low-dose regimen (INR 1.5-2) resulted in 60% risk reduction.[16] Any decision on anticoagulant therapy cessation in individual patients should take into account that the annual incidence of major bleeding in patients on long-term anticoagulant therapy is 1.5-2%, and that case fatality rate or frequency of major bleeding episodes with fatal outcome is greater than the frequency of recurrent VTE. Consequently, for certain patients with high hemorrhagic risk, unconventional oral anticoagulant therapy with a target INR 1.5 to 2 should be considered.[14]

Persistent dysfunction or right ventricular enlargement after acute PE, residual DVT, non-recanalised DVT (confirmed by venous ultrasonography), low HDL, male sex and body overweight are considered risk factors for recurrent VTE. In contrast, the finding of a persistent thrombus on chest computed tomography (CT) has no predictive value for the recurrence of pulmonary emboli (PE) since about half of PE appear as persistent defects in chest CT recordings six months after the initial event.[9,15] Also, most thrombophilias do not increase the risk of recurrent VTE.[15] Clus-

tered data from 10 studies (3104 patients enrolled with a first episode of VTE) indicate an odds ratio for recurrent VTE to 1.72 (95% CI 1.27 to 2.31) in those with prothrombin mutation G20210A and a ratio of 1.41 (95% CI 1.14 to 1.75) with factor V Leiden mutation.[13] Meta-analyses indicate that the incidence of recurrent VTE is higher immediately after discontinuation of anticoagulant therapy, but it tends to decrease over time. In addition, the onset of recurrent VTE nine months after discontinuation of anticoagulation therapy does not depend on the prior therapy duration.[13]

Recommended duration of primary prophylaxis anticoagulant therapy depends upon VTE category

Based on official recommendations, primary prevention of VTE depends upon the type of previous surgery. In addition to selecting the appropriate type of thromboprophylaxis (mechanical, medication, or combined) and the type and dose of anticoagulant agents, it is necessary to consider the duration of treatment and to tailor it to the specific requirements of a particular surgical procedure. This would apply as well for protection against VTE in nonsurgical ("medical") patients, too. The National Institute for Health and Clinical Excellence (NICE) clinical recommendations (2010) advise thromboprophylaxis over a period of 28-35 days for patients with elective hip surgery or hip fractures and 10-14 days for patients with elective knee surgery, while major surgery for abdominal or pelvic malignancy requires thromboprophylaxis for 28 days from the day of the intervention.[4, 28] American College of Chest Physicians (ACCP) guidelines recommend continuing thromboprophylaxis up to 28 days, continuing after hospital discharge for those with malignancies and for other high-risk patients after general or gynecological surgery. [28]

Thromboprophylaxis is advisable for individuals with reduced mobility, such as those who have had general, gynecologic, urologic, thoracic surgery, coronary artery bypass graft or bariatric surgery as well as those with major trauma or spinal cord injury. It should be continued until the patient has regained mobility, usually about five - seven days.[28]

Where there is lower limb immobilization in a cast, the physician should prescribe the appropriate thromboprophylaxis after evaluating the risk and benefit in each patient.[28] ACCP (2008) recommends thromboprophylaxis for acutely ill patients admitted to the hospital due to congestive heart failure, severe respiratory diseases, and for those who are "bedridden" or who have additional risk factors for VTE, such as: active malignancy, previous VTE, sepsis, acute neurologic disease or inflammatory bowel disease.[28] The ACCP also advises tailoring thromboprophylaxis according to the type of cancer surgery and bedridden patients.[28] The ACCP guidelines from the CHEST 2008 do not recommend pharmacotherapy for prevention of thrombosis caused by venous catheters or as routine thromboprophylaxis in patients receiving hormone or chemotherapy; similarly, thromboprophylaxis is not recommended as a means of increasing survival rates in patients with malignancies.[28]

Thromboprophylaxis should be initiated as soon as possible for patients with burns and additional risk factors for VTE (one or more of the following: advanced age, morbid obesity, extensive burns, particularly in lower extremities, concomitant lower extremity injuries, the use of femoral venous catheters and prolonged immobility). For travellers on long-haul flights for more than eight hours (even over 4-6 hours), the ACCP emphasizes the importance of general measures, such as maintaining adequate hydration, avoidance of tight clothing around the waist and lower extremities and exercising the lower-leg muscles. If these travelers have additional risk factors for VTE, they should also wear lower-leg elastic stockings that provide 15-30 mm Hg pressure at the level of ankle. Alternatively, they could be given a prophylactic dose of LMWH prior to the flight.[4, 29-31]

Thromboprophylaxis is recommended for pregnant women and those who gave birth in the last six weeks (without surgery). This is particularly important if they have one or more of the following risk factors presented in Table 4.

Table 4. Risk factors for VTE in pregnancy and puerperium.[4]

Reduced mobility for three days or more
Active malignancy or malignancy treatment
Age over 35 years
Obesity (BMI before pregnancy or in early pregnancy over 30 kg/m^2)
Admission to the intensive care unit
Dehydration, major blood loss or transfusion
Comorbidities (cardiac, metabolic, endocrine and respiratory diseases)
Acute infective diseases and inflammatory conditions
Positive family history of VTE in first-degree relatives
Ovarian hyperstimulation
Hyperemesis gravidarum, multiple pregnancy, preclampsia
Varicose veins with phlebitis
Known thrombophilia

Thromboprophylaxis is also advised for nonsurgical patients, *i*.e. those with acute medical illness, stroke, malignancy, central venous catheters or those who are confined to bed for longer than three days. Thromboprophylaxis is indicated for patients with stroke, especially for those with excluded hemorrhagic stroke or ruptured cranial and spinal vascular malformations. Despite the generally lower risk of hemorrhagic transformation of stroke or hemor-

rhage in other locations, mitigating factors such as reduced mobility, previous VTE, dehydration, presence of comorbidity like malignancy should influence the decision for prophylactic treatment.[4]

Because of the high incidence of arterial cardiovascular events in patients with previous spontaneous VTE antiplatelet agents should be considered as part of the regime for long-term secondary prevention of VTE.[13] In addition, to prevent VTE, other general, non-pharmacological measures can be used, such as weight reduction, prevention of dehydration, and mechanical means (elastic stockings, compression devices such as intermittent pneumatic compression or foot pumps). [14] Temporary anticoagulant therapy should be considered in a setting of inflammation, immobilization, estrogen therapy etc. A number of studies indicate that up to 40% of patients with previous VTE develop recurrent VTE. It should be noted that recurrent VTE occurs more frequently in those with spontaneous VTE than in patients with clearly defined risk factors.[14]

The decision to terminate anticoagulation therapy requires individual assessment of each patient, including their D-dimer values and ultrasound findings in lower limb veins. A balanced approach takes into account the risk of hemorrhage. [14] The choice of an anticoagulant regime must include assessment of the risk of venous thrombosis caused by heparin-induced thrombocytopenia type II while selecting an adequate non-heparin anticoagulant. [15-17, 28, 32-37]

Management of bleeding associated with oral anticoagulants

Because the use of any anticoagulant (old and new) may be complicated by the potential of bleeding, the clearance mechanisms, and the half-life of each of these agents one should understand in order to plan strategy for rapid reversal.[38] Options for reversing anticoagulation include: (1) withholding anticoagulation therapy (observation); (2) administering a specific reversal agent (e.g. oral or intravenous vitamin K if the bleed-related to a vitamin K antagonist); and (3) administering supplemental clotting-factor substitutes (e.g. fresh frozen plasma or prothrombin complex concentrates). However, appropriate supportive and symptomatic treatment is also needed (e.g. mechanical compression or surgical intervention).

Dabigatran and rivaroxaban have relatively short half-lives (dabigatran 12-17h, rivaroxaban 7-11h), in majority of patients with minor or mechanically controlled bleeding, observation and supportive care is the preferred strategy. In the event of a bleed or the need to take a patient emergently to surgery, there are pharmacodynamic parameters that can be measured to determine the approximate level of anticoagulation. For example, fordabig atran monitoring includes following: ecarin clotting time (ECT), thrombin time (TT) and activated partial thromboplastin time (aPTT), which, being relatively insensitive especially at high plasma concentrations, is not suitable for precise quantification of anticoagulant effect. Anticoagulation reversal agent for dabigatran is recombinant factor VIIa (rF-VIIa).

Epilogue

Anticoagulation is a common intervention in the prevention and treatment of thrombosis in multiple clinical settings. Its duration, both in primary and in secondary prevention, depends upon the risk for recurrent VTE as well as the risk for bleeding and present comorbidities.[7] Therefore, determining the length and type of an anticoagulant regimen must be guided by achieving the proper balance between the benefit of therapy and the risk of hemorrhage.[13] New oral anticoagulants (direct thrombin inhibitor and factor Xa inhibitors) may present simpler and safer treatment and prevention of VTE. Their immediate onset of anticoagulant effect, convenient administration, and lack of needed regular anticoagulation monitoring are of interest both for the patients and medical profesionals. Dabigatran is the first oral thrombin inhibitor approved for the prevention of stroke and systemic embolism in patients with non-valvular atrial fibrillation and one or more risk factors for stroke. Dabigatran has also been approved in several countries for the prevention of venous thrombosis in patients undergoing total knee or hip replacement. The RE-NOVATE study on the prevention of venous thromboembolism (VTE) after hip arthroplasty and RE-MODEL study on VTE prophylaxis after knee arthroplasty showed non-inferiority of dabigatran compared with enoxaparin adminstered in European doses of 40 mg daily, while the RE-MOBILISE study after hip arthroplasty confirmed dabigratan inferiority compared with enoxaparin at the North American dose of 30 mg twice daily. However, in the treatment and prevention of VTE, more data should be accumulated to show their ultimate place in therapy.

Author's contribution

The paper is designed and writen by its NMA. VK directed and supervised this project. IŽ and LjJ provided assistance in sourcing relevant literature and writing parts of the paper.

Conflict of interest

All authors declare no conflict of interest related to this paper.

Acknowledgements

This work was supported in part by the Serbian Academy of Sciences and Arts and by Research Grant No. 173008/2011 from the Ministry of Science, Republic of Serbia. We thank Jelena M. Antonijević, and Dr. Sci. Valentina Đorđević, for language advice and consulting.

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Faktori rizika za venski tromboembolizam i trajanje antikoagulantne terapije

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APSTRAKT

Utvrđivanje reverzibilnih i ireverzibilnih faktora rizika za venski tromboembolizam (VTE), preduslov je za određivanje adekvatnog režima tromboprofilakse. Podaci ukazuju da najveći procenat plućnih embolija nastaje u nehirurških bolesnika. U hirurških bolesnika VTE se u velikom broju javlja posle otpusta iz bolnice. Ova saznanja nameću potrebu za adekvatnom zaštitom od VTE obolelih od inflamatornih oboljenja, akutnih bolesti i drugih nehirurških oboljenja, kao i optimalizacijom antikoagulantnog režima posle hirurških intervencija. Utvrđivanje faktora rizika za VTE, određivanje prisustva stečene i urođene trombofilije, okultnog ili do tada neprepoznatog maligniteta pomoći će definisanju antikoagulantnog režima u hirurških i nehirurških bolesnika u sekundarnoj prevenciji VTE. Praćenje vrednosti D-dimera, ultrazvučna procena rezidualne venske tromboze, ehokardiografski parametri uz druge relevantne kliničke podatke ukazuju na rizik od nastanka rekurentnog VTE. Ove procedure omogućavaju utvrđivanje optimalne dužine antikoagulante terapije u svakog bolesnika ponaosob, sa posebnom pažnjom na prisutne komorbiditete i hemoragijsku tendenciju.

KLJUČNE REČI

Venski tromboembolizam, tromboprofilaksa, rekurentna tromboza, rizik od krvarenja.