Click to prove you're human

إرشادات علاج dvt 2018 pdf download

As a library, NLM provides access to scientific literature. Inclusion in an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice . 2021 Feb 23;56(1):6–16. doi: 10.5045/br.2021.2020083 Venous thromboembolism (VTE), which includes pulmonary embolism and deep vein thrombosis, is a condition characterized by abnormal blood clot formation in the pulmonary arteries and the deep venous vasculature. It is often serious and sometimes even fatal if not promptly and appropriately treated. Moreover, the later consequences of VTE may result in reduced quality of life. The treatment of VTE depends on various factors, including the type, cause, and patient comorbidities. Furthermore, bleeding may occur as a side effect of VTE treatment. Thus, it is necessary to carefully weigh the benefits versus the risks of VTE treatment and to actively monitor patients undergoing treatment. Asian populations are known to have lower VTE incidences than Western populations, but recent studies have shown an increase in the incidence of VTE in Asia. A variety of treatment options are currently available owing to the introduction of direct oral anticoagulants. The current VTE treatment recommendation is based on evidence from previous studies, but it should be applied with careful consideration of the racial, genetic, and social characteristics in the Korean population. Keywords: Venous thromboembolism, Deep vein thrombosis, Pulmonary embolism, Anticoagulants Venous thromboembolism (VTE) includes pulmonary embolism (PE), which is potentially fatal and causes chronic thromboembolic pulmonary hypertension, and deep vein thrombosis (DVT), which causes leg swelling and postthrombotic syndrome. In addition, VTE may develop in a splanchnic vein and in other atypical locations. Currently, cancer-associated thrombosis (CAT) is highlighted due to the increased prevalence of cancer. Although the incidence of VTE in Asian populations, including that of Korea, is lower than that in Western populations [1], the age- and sex-standardized annual incidence rate (ASR) of VTE per 100,000 individuals in the Korean population has increased steadily starting in 2004 [2], and this increase has been maintained, leading to an ASR of 29.2% in 2013 [3]. Among cardiovascular diseases, PE is the third most common cause of death, after heart attack and stroke [4]. The fatality rate is approximately 6% after DVT and approximately 12% after PE [5]. Vitamin K antagonists (VKA; warfarin) and low-molecular-weight heparin (LMWH) have been used for the treatment of VTE. However, direct oral anticoagulant (DOAC) therapies have also been developed; DOACs have been commonly used for the treatment of VTE since the mid-2000s because they do not require regular hematologic monitoring, and fixed-dose oral intake is convenient. In Korea, dabigatran was first covered by the National Health Insurance System in May 2015, and rivaroxaban, apixaban, and edoxaban were subsequently approved for VTE treatment. Overall, VKA has been rapidly replaced by DOACs in the market [3]. Therefore, the development of Korean guidelines for VTE treatment is needed for Korean clinicians, although the results of clinical trials for Korean patients with VTE [6] are still scarce. The members of the Thrombosis and Hemostasis Working Party under the Korean Society of Hematology decided to develop Korean recommendations for VTE treatment. All members are clinical or laboratory hematologists, and most are affiliated with tertiary teaching hospitals. The group divided the specific topics pertaining to VTE treatment among the members; subsequently, the members wrote the manuscript, referencing recent guidelines, meta-analyses, and the results of pivotal clinical trials while reflecting on domestic circumstances in Korea. After completion of the manuscript draft, independent members in the field of thrombosis and hemostasis reviewed the primary manuscript. All members reviewed the completed manuscript several times. This recommendation is not a manual for VTE treatment. Physicians must consider each clinical situation and the patient’s condition or status when making decisions. The diagnosis or prophylaxis of VTE is beyond the scope of our recommendation. This recommendation may aid in clinical decision-making for Korean patients with VTE. Medical anticoagulation consists of 3 periods: acute (the first 5–21 days), long-term (3–6 mo), and extended period (thereafter; Fig. 1). In the acute period, anticoagulants are loaded via parenteral heparin lead-in or a higher dose of oral anticoagulants is administered. For long-term treatment, the general anticoagulant treatment period for postoperative VTE is 3 months because of the low risk of recurrence, but is prolonged to 6 months for unprovoked VTE. Extended treatment can be considered in some cases with a high risk of VTE recurrence. Overview of anticoagulant therapy for venous thromboembolism. a)Doses can be modified according to organ function, body weight, or concomitant medications. Abbreviations: INR, international normalized ratio; LMWH, low-molecular-weight heparin; UFH, unfractionated heparin. Abbreviations: INR, international normalized ratio; LMWH, low-molecular-weight heparin; UFH, unfractionated heparin.For the treatment of VTE, heparin and VKA have been used widely for a long time, although DOACs are quickly replacing them because of their convenience of use. However, in certain situations or for patients who have already been using older anticoagulants for a long time, heparin and VKA can continue to be used [7]. LMWH is also frequently used, especially in cases of CAT. Compared to unfractionated heparin (UFH), it is more likely to show a predictable dose-response relationship. It has a longer half-life, allowing it to be administered subcutaneously once a day or twice a day, with a lower risk of heparin-induced thrombocytopenia and osteoporosis. Because LMWH is excreted mainly by the kidney, it should not be used in patients with severe renal impairment. Conversely, UFH has a shorter half-life and can thus be useful in patients who require an immediate anticoagulant effect to support rapid conversion. VKA is absorbed in the gastrointestinal tract and metabolized in the liver. It exhibits moderate anticoagulant effects by inhibiting vitamin K between 48 and 72 h after the start of administration. Because protein C and protein S, which are considered natural anticoagulants, may also decrease during the priming of VKA, the risk of thrombosis and skin necrosis increases in the first 2 or 3 days of VKA use. Therefore, during this period, VKA should be administered with heparin. The effect of VKA is affected by the patient’s liver function, concurrent medication, and food intake; therefore, careful monitoring is required. Dabigatran directly inhibits factor IIa, while rivaroxaban, apixaban, and edoxaban inhibit factor Xa. Several large-scale phase 3 studies have compared DOAC therapies with VKA, confirming that DOACs are not inferior in terms of antithrombotic efficacy and bleeding [8-11]. Thus, DOACs have been used instead of VKA for the treatment of VTE. The major drug absorption sites of rivaroxaban, dabigatran, and edoxaban are the stomach and the proximal small intestine. Therefore, in patients with previous total gastrectomy, these drugs should be taken with caution because they may not be absorbed efficiently. Unlike other DOACs, apixaban is mostly absorbed in the distal small intestine and ascending colon and can be safely used in patients who have undergone gastrectomy; however, it should be avoided in patients who have previously undergone resection of the ascending colon [12]. Dabigatran is produced as a capsule formulation. Thus, when the capsule of dabigatran is opened and the contents are taken alone, its bioavailability rises rapidly. Opening, crushing, or chewing dabigatran capsules is prohibited. For the use of DOACs in cases of CAT, see the section ‘Treatment of cancer-associated venous thromboembolism’. Anticoagulation in renal insufficiency: Because the main excretion site of DOACs is the kidney, the anticoagulant effect of DOACs increases in the presence of renal insufficiency, and the likelihood of bleeding complications also increases. Since patients with renal insufficiency were not included in the clinical trials of DOACs (for example, a creatinine clearance of