Venous Thromboembolism: Updated Management Guidelines

A review of what's new, what's the same, and the implications for nursing practice.

ABSTRACT: Venous thromboembolism (VTE) is a leading cause of death and disability worldwide. Each year, more than 10 million cases of VTE are diagnosed; studies suggest there are as many as 900,000 cases per year in the United States. The condition is estimated to cost the U.S. health care system between $7 billion and $10 billion annually. In February 2016, the American College of Chest Physicians released the 10th edition of the Antithrombotic Therapy for VTE Disease: CHEST Guideline and Expert Panel Report. After providing an overview of VTE pathophysiology, risk factors, signs, symptoms, and key clinical assessments, this article details recommendations from the new guideline, which incorporates the most up-to-date treatment options for patients with VTE. The authors highlight key changes from the 2012 guideline, particularly those related to nursing practice, patient education, care coordination, patient adherence, medication costs, follow-up appointments, and diagnostic testing.

Keywords: care coordination, embolism, non–vitamin K oral anticoagulants, patient education, treatment guidelines, venous thromboembolism

Venous thromboembolism (VTE) is a leading cause of worldwide death and disability and a growing public health concern. In the United States, there is no national surveillance system for VTE, so the national incidence is unclear. Estimates of U.S. incidence vary from 300,000 to 900,000 cases per year,[1-4] with as many as 100,000 to 300,000 cases ending in death.[4]

VTE, which includes both deep venous thrombosis (DVT) and pulmonary embolism (PE), is the second most common medical complication related to hospitalization and extended length of stay and the third most common cause of excess hospital charges and high mortality rates.[5] During 2007–2009, a discharge diagnosis of VTE was recorded for roughly 550,000 adult hospitalizations per year.[6] Because it is a condition associated with health care, VTE has received attention from the U.S. Surgeon General, the Joint Commission, the Centers for Medicare and Medicaid Services, the National Quality Forum, and the Agency

for Healthcare Research and Quality in the form of calls to action, new quality and performance measures, information sheets, consensus standards, and guides to quality improvement.[2,6-8] Despite this focus, the number of secondary diagnoses of VTE in hospitalized patients has continued to rise and a 2011 retrospective analysis of U.S. health care claims data estimated that the incidence of VTE would more than double from 950,000 cases in 2006 to 1.82 million in 2050.[9]

Hospital stays for VTE place a considerable economic burden on the U.S. health care system,[10-12] with total health care costs, including the treatment of acute and recurrent VTE as well as the treatment of resulting complications, estimated to be between $7 billion and $10 billion per year.[11] In 2011, mean hospital charges were $30,051 for DVT and $37,006 for PE, while the mean length of stay was 4.7 days for patients with DVT and 5.1 days for patients with PE.[12]

In February 2016, the American College of Chest Physicians published the 10th edition of the Antithrombotic Therapy for VTE Disease: CHEST Guideline and Expert Panel Report (CHEST Guideline).[13] This guideline incorporates the most up-to-date treatment options for patients with VTE and includes some noteworthy changes to the 2012 9th edition guideline (see Table 1[13]). These include the following[13]:

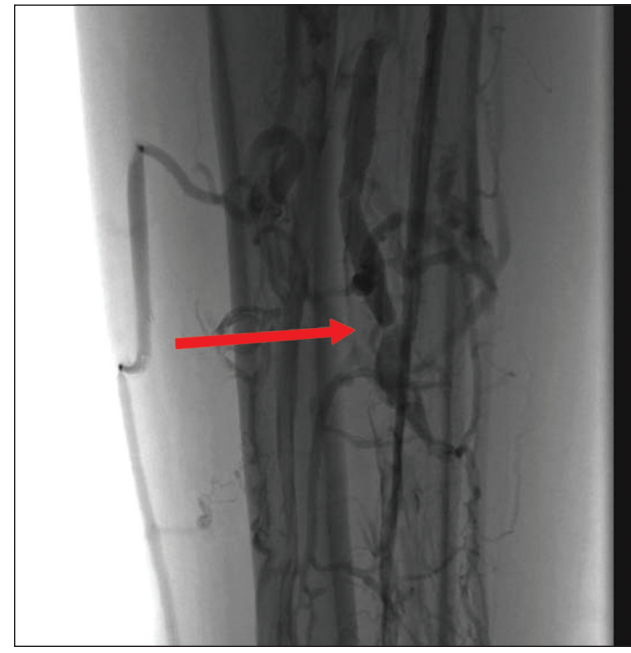
- the recommended use of non–vitamin K oral anticoagulants (NOACs) over warfarin for initial and long-term treatment of VTE in patients without cancer
- a reversal of the recommendation to routinely use compression stockings to prevent postthrombotic syndrome
- new treatment recommendations for patients with isolated subsegmental PE

Drawing largely from this guideline, this article provides an overview of VTE pathophysiology, risk factors, signs and symptoms, and key clinical assessments, with an emphasis on nursing practice, patient education, care coordination, patient adherence, medication costs, follow-up appointments, and diagnostic testing.

VTE PATHOPHYSIOLOGY

Venous stasis, as can occur with immobilization or prolonged bed rest, is a well-established risk factor for developing VTE. In fact, when patients are hospitalized for an acute medical illness, their risk of developing VTE increases by a factor of eight.[14] There are, however, numerous other risk factors, both modifiable and nonmodifiable (see *Risk Factors for VTE Development*[15-17]).

DVT is the initial presentation in two-thirds of patients with VTE.[1] The etiology of DVT was first proposed by the 19th-century German physician Rudolf Virchow as a triad of vascular abnormalities: hypercoagulability, endothelial injury, and altered venous blood flow. These abnormalities may result from medical conditions or therapies, traumatic injury, surgical procedures, dehydration, or reduced or restricted activity.



In this venogram of the lower leg, the red arrow points to a filling defect associated with deep vein thrombosis. Image © O'Connor MB, et al. *Cases J* 2009; licensee BioMed Central.

DVT most commonly develops in the lower extremities, though the study of a U.S. multicenter registry of patients with confirmed DVT found that 11% of the patients had developed upper-extremity DVT.[18] Rarely, DVT develops in cerebral, pelvic, and mesenteric veins. It is critically important to identify the size and exact location of lower-extremity DVT because thrombi in the proximal veins of the leg, defined as the popliteal vein and those above it, are strongly associated with PE if left untreated. While thrombi located in the distal lower-extremity veins are less likely to result in PE, up to 15% of distal thrombi enlarge and extend into the proximal deep veins if left untreated.[19]

PE, an obstruction within a pulmonary artery, is most commonly caused by a thrombus but can also be caused by air, fat, or a tumor. The mechanical obstruction within the pulmonary vasculature can cause pulmonary hypertension and ultimately right heart failure. In response to rising pulmonary pressures,

compensatory mechanisms activated by the sympathetic nervous system release chemical mediators that cause vasoconstriction, further reducing blood flow through the pulmonary vasculature and creating a ventilation–perfusion mismatch. The resulting hypoperfusion and hypoxemia cause both pulmonary and cardiac ischemia and potentially tissue infarction. Early recognition and appropriate treatment of PE maximize patients' chances of survival.

CLINICAL PRESENTATION

Common signs and symptoms of DVT include unilateral swelling of the affected limb in conjunction with warmth, tenderness, and redness from the inflammation that accompanies venous obstruction. A thorough assessment of the affected extremity may reveal superficial venous dilation as well. Any of these symptoms should be evaluated in the context of the patient's current medical condition and circumstances.

The most frequently reported symptoms of PE are dyspnea and pleuritic pain. Other PE signs and symptoms include coughing, palpitations, anxiety, wheezing, bloody sputum, and unilateral extremity pain. Assessment may reveal unilateral extremity swelling, fever, hemoptysis, pleural effusion, altered mental status, and hypoxia. Differences have been noted in symptoms reported by women and men, with women more likely to report anxiety, shortness of breath, and

Table 1. Major Changes in Guideline Recommendations for the Management of VTE[13]

Category	Prior Recommendation	Current Recommendation	Evidence Supporting This Change
Choice of long-term anticoagulants	Warfarin for patients without cancer	NOACs over warfarin for patients without cancer	Risk reduction is similar with NOACs. Risk of bleeding is less with NOACs. Greater convenience for patients with NOACs
Use of aspirin for extended therapy	Not addressed	Aspirin recommended for patients discontinuing NOAC therapy and for those who decline NOAC therapy	Moderate-quality evidence that use of aspirin reduces recurrent VTE by about 33%
Use of compression stockings to prevent postthrombotic syndrome	Recommended	Not recommended	No evidence to support the use of compression stockings to prevent postthrombotic syndrome
Treatment of subsegmental PE	Not addressed	Clinical surveillance over anticoagulation in patients with no proximal DVT and low risk of recurrence	After subsegmental PE, which is small and usually originates from an isolated DVT, the risk of recurrence is less than with a larger PE.
Outpatient treatment of acute PE	Recommended early discharge (after five days)	Outpatient treatment recommended for carefully selected patients	Treatment with a NOAC facilitates outpatient treatment for select patients. A NOAC that does not require bridge therapy should be selected to aid in this process.
Management of recurrent VTE while on anticoagulant therapy	Not addressed	For patients on oral anticoagulant therapy, switch to LMWH for one month. For patients on LMWH, increase dosage by 25% to 33%.	Low-quality evidence supports the use of LMWH for a short period because the risk of recurrent VTE decreases over time.

DVT = deep venous thrombosis; LMWH = low-molecular-weight heparin; NOAC = non–vitamin K oral anticoagulant; PE = pulmonary embolism; VTE = venous thromboembolism.

calf pain, and men less likely to report signs and symptoms of PE or DVT.[20]

CLINICAL ASSESSMENT

When DVT is suspected, screen the patient using a DVT probability scale, such as the Wells Score.[21] (See Table 2.[21]) If the patient receives a Wells score of 2 or higher, diagnostic testing, starting with a D-dimer laboratory test, should be performed as soon as possible.[22, 23]

The D-dimer test identifies the presence of the small fibrin fragments produced when the body tries to break down blood clots. While the presence of D-dimer fragments in the blood may suggest the presence of a blood clot, the test is not specific enough to serve as a diagnostic criterion because D-dimer fragments are also associated with inflammation, pregnancy, trauma, surgery, and infection. For this reason, the D-dimer test is most often used to exclude, rather than to confirm, the diagnosis of DVT.[24] A negative D-dimer test indicates that the likelihood of DVT is low. The primary noninvasive tool used to diagnose DVT is venous-compression ultrasonography, which has a sensitivity of 97% and a specificity of 94% in correctly identifying proximal lower-extremity vein thrombosis.[24] Treatment can be started immediately upon ultrasonographic confirmation of a thrombus, though if findings are inconclusive or inconsistent with clinical assessment, venography should be considered.[25]

When PE is suspected, screen the patient using a probability tool, such as the Wells Criteria for Pulmonary Embolism (available online at www.mdcalc.com/wells-criteria-pulmonary-embolism).[26] If probability screening suggests a likelihood of PE, diagnostic testing should be initiated as soon as possible. As with DVT testing, a D-dimer blood test can be performed to rule out the presence of PE.[26] Diagnostic imaging includes multidetector computed tomography angiography (MDCTA), which is highly sensitive in detecting PE and, when used in conjunction with D-dimer measurement, can safely rule out PE in patients without high clinical probability, eliminating the need for compression ultrasonography in such cases. If MDCTA scanning is unavailable or cannot be used because of patient sensitivity, a ventilation–perfusion scan can be performed, though this has a lower sensitivity in identifying PE, particularly in patients with preexisting pulmonary conditions.[27]

UPDATED GUIDELINE RECOMMENDATIONS

The 10th edition of the CHEST Guideline did not change previous recommendations regarding which patients should and should not receive extended anticoagulation therapy; however, several recommendations concerning VTE prevention and treatment were reversed or modified.[13] Except as otherwise cited, the discussion in this section is based on this report by Kearon and colleagues.

Risk Factors for VTE Development[15-17]

- Metabolic, endocrine, or respiratory disorders
- Hypercoagulable states, including genetic thrombophilia
- Active malignancy or cancer treatment
- Advanced age
- Hypertension
- Elevated triglyceride levels and low levels of high-density lipoprotein cholesterol
- Previous VTE or first-degree relative with VTE
- Recent surgery or trauma
- Pregnancy
- Estrogen use (oral contraceptives or hormone replacement therapy, for example)
- Limited mobility
- Severe obesity
- Venous stasis, as can occur with immobilization or prolonged bed rest
- Dehydration
- Inpatient status with acute medical illness
- Varicose veins with phlebitis
- Presence of multiple medical comorbidities

VTE = venous thromboembolism.

Choice of anticoagulant therapy. The expert panel agreed that current evidence supports the use of anticoagulant therapy in treating PE or proximal DVT. For patients with cancer who develop VTE, low-molecular-weight heparin (LMWH) continues to be recommended as the primary treatment over any of the oral agents, including warfarin and the NOAC medications—dabigatran and edoxaban, which must be combined with LMWH in the acute phase of treatment, and apixaban and rivaroxaban, which need not be combined with LMWH. For patients without cancer who develop VTE, new recommendations support the use of NOAC medications over warfarin for both initial and long-term treatment, but do not recommend any NOAC over another. Research has shown that NOAC medications perform as well as warfarin in preventing recurrent VTE—and with less risk of bleeding, most notably intracranial bleeding.[28, 29] Although warfarin has a specific reversal agent, warfarin doesn't have a lower risk of a fatal bleeding event than the NOAC medications, with rivaroxaban and apixaban having the lowest bleeding risk compared with either LMWH plus warfarin or unfractionated heparin plus warfarin.[28, 29] The choice of anticoagulant is also influenced by comorbid conditions and patient preference. LMWH is the panel's recommended therapy for VTE associated with

pregnancy because the oral anticoagulants have been shown to cross the placental barrier.

Duration of anticoagulant therapy. When an anticoagulant is prescribed, the panel recommends three months as the minimum course of treatment. A decision to extend anticoagulant therapy beyond three months should not be made lightly, as it usually implies that the treatment will continue indefinitely. If the VTE was associated with surgery or another transient risk factor, the panel recommends discontinuing treatment at the end of three months. Isolated, distal DVT, however, may require no anticoagulant therapy. For patients who develop a first unprovoked proximal DVT or PE or a second unprovoked VTE and have a low to moderate risk of bleeding, extended anticoagulant therapy, with no scheduled stop date, is recommended. If the bleeding risk is considered high, further risk stratification is in order. Any patients prescribed extended anticoagulant therapy should be reassessed periodically.

D-dimer levels. The panel recommends that D-dimer levels be measured in all patients who have received anticoagulant therapy about one month after discontinuing treatment. While there are no specific recommendations regarding continuing therapy on the basis of D-dimer levels or patient sex, both factors may influence the decision. Patients who have an elevated D-dimer level have about twice the risk of recurrent VTE than patients who have a negative D-dimer test, men have a 75% higher risk of recurrence than women, and together the two factors may have greater predictive significance.

The use of antiplatelet therapy, including aspirin, in the treatment of VTE has not been addressed in previous guidelines. Extended aspirin therapy has been found to reduce the recurrence of VTE without significantly raising the risk of bleeding.[30] While the panel does not view aspirin as an acceptable substitute for warfarin or a NOAC, it recommends that patients who discontinue or decline traditional anticoagulant therapy receive aspirin therapy unless its use is contraindicated.

The risk of developing recurrent VTE decreases over the course of anticoagulant therapy. The development of recurrent VTE while receiving anticoagulant therapy at therapeutic doses is rare and should prompt diagnostic reevaluation, evaluation of adherence, and consideration of undiagnosed active malignancy. Following these assessments, the guideline panel recommends prescribing a short-term (usually one-month) course of LMWH and then resuming oral anticoagulant therapy. Any ongoing treatment that would increase the likelihood of VTE, such as hormone therapy or chemotherapy, may be discontinued.

Table 2. Wells Clinical Model for Predicting Pretest Probability of DVT[21,a]

Clinical Characteristic	Score
Active cancer (patient receiving treatment for cancer within the previous 6 months or currently receiving palliative treatment)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden for 3 days or more, or major surgery within the previous 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than that on the asymptomatic side (measured 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Previously documented DVT	1
Alternative diagnosis at least as likely as DVT	–2

DVT = deep venous thrombosis.

[a] A score of 2 or higher indicates that the probability of DVT is likely; a score of less than 2 indicates that the probability of DVT is unlikely. In patients with symptoms in both legs, the more symptomatic leg is used.

DVT management varies with thrombi location and patient symptoms. If thrombosis is limited to the muscular veins of the calf and the patient is not at risk for extension and has no severe symptoms, the risk of anticoagulation may outweigh the potential benefits. For these patients, the panel recommends serial ultrasonographic imaging for two weeks over anticoagulation therapy unless imaging reveals extension, in which case anticoagulation therapy should be initiated and continued for at least three months. Since roughly 15% of untreated distal thrombi extend into the proximal veins of the leg, patients with distal DVT should be either monitored by ultrasound or treated with anticoagulation therapy. Factors that increase the risk of thrombotic extension include

- size of the thrombus (greater than 5 cm in length, greater than 7 mm in diameter, or involving multiple veins).
- a markedly positive D-dimer test.
- inpatient status.
- active cancer.
- location of the thrombus near proximal veins.
- history of VTE.
- no reversible provoking factor.

The current guideline does not recommend catheter-directed thrombolysis, using such agents as tissue plasminogen activator or streptokinase, to dissolve a thrombus unless the patient is developing venous gangrene. It further recommends against using an inferior vena cava filter in patients who are receiving anticoagulant therapy.

Postthrombotic syndrome is a common complication of DVT. Between 23% and 60% of patients diagnosed with DVT develop postthrombotic syndrome within two years of DVT onset.[31, 32] Signs and symptoms include edema, skin discoloration, pain, and in severe cases, ulceration. The pain and swelling caused by this syndrome can substantially affect quality of life. While earlier versions of this guideline recommended the use of compression stockings to prevent the development of postthrombotic syndrome, the current guideline cited a more recent, large, multicenter, placebo-controlled study, which found that routine use of compression stockings did not reduce the incidence of postthrombotic syndrome. While the current guideline does not recommend routine use of compression stockings to prevent postthrombotic syndrome, it acknowledges that these stockings may reduce symptoms in affected patients.

Upper-extremity DVT. The panel also recommended that upper-extremity DVT, like lower-extremity DVT, be treated with anticoagulant therapy rather than thrombolysis, unless the patient is "most likely to derive benefit" from thrombolysis because she or he has

- had severe symptoms for fewer than 14 days.
- a thrombus involving most of the subclavian and axillary veins.
- a low risk of bleeding.
- good functional status.
- life expectancy of at least one year.

For patients with upper-extremity DVT who receive thrombolysis, catheter-directed thrombolysis is preferred over systemic thrombolysis. After undergoing thrombolysis, the guideline recommends that patients receive anticoagulant therapy at the same dosage and duration as patients who did not undergo thrombolysis.

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Management of PE. The advances in computed tomography pulmonary angiography have increased the diagnosis of PE that is confined to the subsegmental pulmonary arteries. Current evidence suggests that subsegmental PE is usually small, having originated from a small, isolated thrombus. Based on low-quality evidence, the guideline recommends that patients with subsegmental PE but no proximal DVT and little risk of recurrent VTE receive ultrasound surveillance of the lower extremities but no anticoagulant treatment, and that those with proximal DVT or a high risk of recurrent VTE receive anticoagulant therapy.

Outpatient treatment of PE. Patients who have no identified risk factors for bleeding and are both hemodynamically stable and expected to adhere to the prescribed therapy should be offered the option of home treatment or early discharge, rather than the standard five days of inpatient treatment.

A screening tool, such as a simplified Pulmonary Embolism Severity Index (PESI), can help clinicians identify patients for whom home treatment would be appropriate. The simplified PESI helps clinicians evaluate such patient data as age, oxygen saturation level, heart rate, blood pressure, and medical history in order to estimate the severity of PE.[33] Patients with a score of 0 would be considered "low risk" and may be suitable candidates for home treatment.[33] The panel recognized the value of screening tools such as the simplified PESI, but emphasized that, though screening tools aid in clinical decision making, they do not replace clinical judgement. PE treatment with a NOAC that does not require LMWH in the acute phase, such as rivaroxaban or apixaban, is best suited to outpatient treatment.

The updated recommendations support the use of systemic thrombolysis only for patients with acute PE who are not at high risk for bleeding and are experiencing hypotension (systolic blood pressure below 90 mmHg for more than 15 minutes). The longer the duration of hypotension or other signs of shock, the greater the indication for systemic thrombolysis. If the patient's condition deteriorates with routine anticoagulation therapy, the need for systemic thrombolysis should be reevaluated.

The expert panel notes that the role of catheter-directed thrombolysis in the treatment of PE is

Table 3. Non–Vitamin K Oral Anticoagulants Used to Treat Acute VTE[34-38]

Generic (Trade) Name	Drug Category	Antidote	Precautions
Dabigatran (Pradaxa)	Direct thrombin inhibitor	Idarucizumab (Praxbind)	• Not recommended for patients with mechanical heart valves • Not recommended for patients with liver disease • Can be used in patients with renal impairment with dose adjustments • Avoid use in patients with renal failure; dosing recommendations cannot be provided for patients with a creatinine clearance < 15 mL/min or for those receiving dialysis treatment • Strict medication adherence is critical • Discontinue at least 24 hours before invasive or surgical procedures; can be restarted after the procedure as soon as medically appropriate
Rivaroxaban (Xarelto)	Factor Xa inhibitor	None	• Not recommended for patients with mechanical heart valves • Can be used in patients with renal impairment with dose adjustments • Avoid in patients with liver disease • Strict medication adherence is critical • Discontinue at least 24 hours before invasive or surgical procedures; can be restarted after the procedure as soon as adequate hemostasis established
Apixaban (Eliquis)	Factor Xa inhibitor	None	• Not recommended for patients with mechanical heart valves • Can be used in patients with renal impairment with dose adjustments • Strict medication adherence is critical • Discontinue at least 24 hours before invasive or surgical procedures; can be restarted after the procedure as soon as adequate hemostasis established
Edoxaban (Savaysa)	Factor Xa inhibitor	None	• Not recommended for patients with mechanical heart valves and moderate to severe mitral stenosis • Not recommended for patients with liver disease • Can be used in patients with renal impairment with dose adjustments • Discontinue at least 24 hours before invasive or surgical procedures; can be restarted after the procedure as soon as adequate hemostasis established

VTE = venous thromboembolism.

supported only by low-quality evidence. In general, systemic thrombolysis is recommended over catheter-directed thrombolysis unless the patient has failed a trial of systemic thrombolysis or is unlikely to survive the few hours necessary for the thrombolysis to be effective. In such situations, if the requisite expertise and resources are available to perform the procedure, the panel recommends catheter-assisted thrombus removal—that is, mechanical intervention, with or without catheter-directed thrombolysis. The panel determined that patients who have developed chronic thromboembolic pulmonary hypertension may receive some benefit after undergoing a pulmonary artery thromboendarterectomy by an experienced surgical team, but emphasized that patient selection requires careful evaluation by specialists in pulmonary hypertension diagnosis and management.

MEDICATION CONSIDERATIONS

The introduction of NOACs in the 10th edition guideline represents a major change in anticoagulation therapy for VTE. To provide safe and effective patient care, it is important for nurses to know about warfarin, LMWH, and the various NOACs used in VTE treatment (see Table 3[34-38]), and to be familiar with the benefits and risks associated with each.

NOAC advantages. NOACs have the following advantages over warfarin:

- Since food does not affect their metabolism, they produce a predictable anticoagulant effect with no need for dietary restrictions.
- They are given in fixed doses, requiring no routine international normalized ratio (INR) monitoring.[39]
- They are as effective as warfarin, cause fewer bleeding complications, and are more convenient to administer.[39]

Unlike NOACs, warfarin has a slow onset of action; when initiated to treat VTE, warfarin must be overlapped with a rapidly acting parenteral anticoagulant bridge therapy for at least five days.[40] With their rapid onset of action, rivaroxaban and apixaban require no anticoagulation bridge therapy.[40] Without the need for bridge therapy, initiating NOAC therapy is simpler than warfarin therapy, and it may reduce hospital length of stay, number of hospital admissions, and cost.

Reversal of anticoagulant effect. One of the main concerns with NOAC treatment is the lack of antidotes should bleeding occur or should the patient require an invasive surgical procedure.[40, 41] Currently, the only NOAC with an antidote is dabigatran for which idarucizumab, a humanized monoclonal antibody fragment, can be used to reverse the anticoagulant effect.[34, 35]

Idarucizumab administration. The recommended dose of idarucizumab is 5 g, provided as two separate 50-mL vials, each containing 2.5 g. When administering idarucizumab, it is important to keep the following steps in mind[35]:

- Do not mix with other medications.
- Once the solution has been removed from the vial, administration should begin within an hour.
- The IV line must be flushed with a 0.9% sodium chloride injection solution before infusion.
- No other infusion should be administered through the same IV access.
- Dabigatran treatment can be reinitiated 24 hours after idarucizumab administration.

Dosing schedules. Nonadherent patients should not be prescribed short-acting NOACs because missed doses can be more harmful than missed doses of warfarin, which has a half-life of several days.[40] Patients who prefer once-daily medications can be prescribed rivaroxaban and edoxaban (dabigatran and apixaban require twice-daily dosing).

Comorbid conditions. Patients with upper gastrointestinal symptoms may have better outcomes with

Patient Teaching Points

Current Treatment

- Importance of medication adherence
- Importance of follow-up appointments
- What to do if you miss a dose of a NOAC
- Signs and symptoms of bleeding to report to your health care provider:
  - Increased bruising
  - Overt bleeding
  - Dizziness, feeling faint
  - Increased shortness of breath
  - Intolerance of cold

Preventing VTE Recurrence

- Stay hydrated
- Avoid oral contraceptives and hormonal therapy
- Exercise, stay active
- Perform leg exercises during travel
- Symptoms to report to your health care provider that may indicate recurrence:
  - Swelling of one extremity
  - Calf pain
  - Numbness or tingling of one extremity
  - Acute shortness of breath
  - Pleuritic chest pain
  - Palpitations
  - Wheezing
  - Bloody sputum
  - Anxiety

NOAC = non–vitamin K oral anticoagulant; VTE = venous thromboembolism.

rivaroxaban or apixaban than with dabigatran. Up to 10% of patients prescribed dabigatran develop dyspepsia, which can lead to early discontinuation.[40] In the updated CHEST Guideline, LMWH remains the anticoagulant of choice for patients with malignancies and for those with mechanical heart valves.[13,42]

Promoting adherence. The effectiveness of VTE treatment depends on patients taking their medications as prescribed. Nurse-led anticoagulation clinics can play an important role in improving care coordination and reducing ED visits and hospitalizations.[43-45] The personalization of dosage routines and the use of text-messaged and e-mailed patient reminders can be beneficial.[46,47] Smartphone medication adherence applications are another strategy for improving adherence.[48] Although routine INR monitoring is not required with NOAC treatment for VTE, periodic, scheduled patient follow-up is necessary to ensure safe and effective treatment. Follow-up visits allow nurses to reinforce patient education and foster a long-term trusting relationship with the patient. When patients feel safe, they are more likely to be forthcoming when discussing adherence issues and missed doses. Encourage patients to bring their prescriptions to follow-up appointments so pill counts may be used as a measure of medication adherence, and consider incorporating adherence into written treatment contracts.[49] Patients may need to be reminded that over-the-counter medications, such as aspirin and other nonsteroidal antiinflammatory drugs, are linked to an increased risk of bleeding. Patient education, emphasizing the need for strict adherence to prescribed NOAC treatment, should be ongoing.

Financial implications. Practical issues may affect NOAC use in the outpatient setting. The preauthorization requirements of some insurance companies, variable patient copays, and high cost of the drugs may present financial barriers for some patients, which may in turn contribute to medication nonadherence. Before prescribing NOACs, health care providers should verify insurance coverage and patient copays. If the patient is eligible for financial assistance through a pharmaceutical company's patient assistance program, arrangements should be made before the treatment is prescribed.[41]

Patient education should be provided upon discharge and reinforced throughout treatment (see *Patient Teaching Points*). In addition to oral instructions, written educational materials should be provided to patients and caregivers for home use. Ensure that patients understand the

- rationale for drug treatment.
- importance of medication adherence in achieving therapeutic anticoagulation.
- consequences of nonadherence, including risk of recurrent VTE.
- signs and symptoms of recurrence.
- nonpharmacologic methods for preventing recurrent VTE.

Successful VTE management is a collaborative process that includes health care providers, their patients, and their caregivers. Patients who are actively involved in their health care and follow their prescribed plans of care have improved health outcomes and lower health care costs. While NOACs may reduce the overall costs associated with treating VTE, there are challenges associated with their use. Nurses play a pivotal role in ensuring that these new anticoagulants improve patient outcomes through

- ensuring that patients have a voice in their medication selection.
- developing effective patient education programs.
- follow-up in the outpatient setting. ▼

For four additional continuing nursing education activities on the topic of venous thromboembolism, go to www.nursingcenter.com/ce.

Sarah Hudson Roberts and Sherry Motes Lawrence are assistant professors at the University of South Alabama College of Nursing in Mobile. Contact author: Sarah Hudson Roberts, sroberts@southalabama.edu. *The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.*

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