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Management of Thrombosis in a Neuro-Oncology Patient

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Mr. B is a 27-year-old man with no significant medical history. He presented two weeks ago with new onset of generalized seizures during his sleep that awoke his wife, who stated, "He was shaking the bed." She was unable to get him to respond. The tremors lasted approximately five minutes. Mr. B was taken to a local emergency room, where a head computed tomography (CT) scan was performed, revealing a 2 cm abnormality in the right temporal lobe, with no evidence of hemorrhage. The patient was given a loading dose of phenytoin and started on IV dexamethasone 10 mg every six hours. Mr. B was transferred to a large medical center for further workup and evaluation.

After admission to the medical center, the patient underwent a staging workup, including magnetic resonance imaging (MRI) of the brain with and without contrast and a CT scan of the chest, abdomen, and pelvis to rule out a primary source of the brain lesion. No primary source was detected. A brain biopsy was performed that revealed a high-grade astrocytoma, World Health Organization grade III (Kleihues, Burger, & Scheithauer, 1993). Mr. B had a craniotomy with postoperative MRI confirming gross total resection. He was scheduled to receive six weeks of radiation therapy and oral temozolomide at 75 mg/m^2 daily for six weeks. He had completed 40 Gy out of 60 Gy of radiation therapy and was continuing on oral temozolomide when he called the triage nurse educator with complaints of right lower-extremity pain, redness, and swelling. An ultrasound of the right lower extremity confirmed a diagnosis of occlusive deep vein thrombosis (DVT). He was hemodynamically stable and otherwise had no complaints. He was started on low-molecular-weight heparin, 1 mg/kg subcutaneously every 12 hours. The clinical nurse specialist instructed Mr. B on self-injection, side effects of enoxaparin, and follow-up care. His insurance company approved enoxaparin prior to instituting the regimen. He was instructed to report any signs of bleeding and have his platelets checked twice weekly while he continued radiation and temozolomide. If the platelet count was less than 50,000/ml, enoxaparin would be held.

What is the incidence of thrombus and what are the implications of the diagnosis in the neuro-oncology population?

A thrombus is a clot that forms as a result of vascular wall injury, venous stasis, and hypercoagulability. The symptom cluster has been described as Virchow's triad (Cervantes & Rojas, 2005). Malignancy produces a hypercoagulable state. Individuals with cancer have an increased incidence of thrombus, up to seven times more than those with no malignancy (Blom, Doggen, Osanto, & Rosendaal, 2005). In the hypercoagulable population, patients with brain tumors have a particularly high incidence of venous thromboembolism (VTE) (Gerber, Grossman, & Streiff, 2006). Patients with large glial tumors have increased tissue factor production and increased levels of active coagulation factors (Sciacca et al., 2004) (see Table 1).

If a patient develops symptoms of DVT and is not treated, the risk is almost 50% that the patient will develop a pulmonary embolus, resulting in significant morbidity and mortality; therefore, assessment and timely intervention and treatment of DVT are vital in all patients (Gerber et al., 2006). In the past, anticoagulants have been administered somewhat reluctantly to patients with primary brain tumors because of a fear of intracranial hemorrhage. Growing evidence suggests that anticoagulation may be more effective than inferior vena cava filtration devices for treating VTE in patients with brain tumors and that the risk of hemorrhage with anticoagulation is relatively small (Wen & Marks, 2002).

What are the risk factors for development of deep vein thrombosis?

Kyrle and Eichinger (2005) stratified the risk of DVT as follows.

- **Low risk:** minor surgery in patients younger than age 40 with no additional risk factors
- **Moderate risk:** minor surgery and additional risk factors; surgery in patients aged 40–60 with no additional risk factors
- **High risk:** surgery in patients older than age 60 or aged 40–60 with additional risk factors (e.g., previous VTE, cancer, thrombophilia)
- **Highest risk:** surgery in patients with multiple risk factors (e.g., older than age 40, cancer, previous VTE, hip or knee arthroplasty, hip fracture surgery, major trauma, spinal cord surgery)

In patients with a first spontaneous DVT, the annual likelihood of recurrence is 5%–15%, with a cumulative recurrence rate of about 25% after four years. Risk of recurrent DVT is low in patients who develop it postoperatively (Kyrle & Eichinger, 2005).

What other factors increase the risk of deep vein thrombosis or venous thromboembolism?

Each year, about 19,000 people in the United States are diagnosed with primary brain cancers. The risk of developing brain

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cancer increases with age. In addition, in the United States, 4.5 individuals in every 100,000 people younger than age 65 develop brain cancer compared to 17.8 in those aged 65 and older (National Cancer Institute, 2000). Older patients often have chronic comorbid conditions such as coronary artery disease, hypertension, or thyroid abnormalities. Atherosclerosis, congestive heart failure, and sepsis also can increase the risk for VTE (Luyendyk, Tilley, & Mackman, 2006).

What is the pathophysiology of deep vein thrombosis?

Increased tissue factor or protein secretion caused by malignancy leads to hypercoagulability (Lin, Wakefield, & Henke, 2006; Sciacca et al., 2004). The formation, propagation, and dissolution of venous thrombi represent a balance between thrombogenesis and the body's protective mechanisms, specifically the circulating inhibitors of coagulation and the fibrinolytic system. Virchow's triad (venous stasis, injury to vascular endothelium, and altered blood composition or hypercoagulability) is considered to be the major contributor to development of thrombus.

DVT develops through activation of coagulation in areas of reduced blood flow. DVT in a lower extremity usually begins in the deep veins of the calf around the valve cusps or within the soleal plexus. A few cases arise primarily in the ileofemoral system as a result of direct vessel wall injury, as seen with hip surgery or catheter-induced DVT. A vast majority of calf vein thrombi dissolve completely without therapy. Approximately 20% grow larger in situ, and propagation usually occurs before embolization. The process of adherence and organization of the venous thrombus does not begin until 5–10 days after thrombus formation. Until the process has been established fully, the nonadherent disorganized thrombus may propagate or embolize (Line, 2001).

What are the presenting symptoms of deep vein thrombosis?

Most patients with DVT are asymptomatic; however, typical symptoms include pain, swelling, and redness in the extremity. Differential diagnoses for presenting symptoms are found in Figure 1. Patients with a malignancy who report a new onset of dyspnea or pleuritic chest pain should be evaluated by physical examination, pulse oximetry, and spiral CT of the chest or a ventilation-perfusion scan to rule out pulmonary embolus.

Possible Causes of Pain or Swelling in a Lower Extremity

- Venous
  - Deep vein thrombosis
  - Superficial thrombophlebitis
  - Post-thrombotic syndrome
  - Chronic venous insufficiency
  - Venous obstruction
- Other
  - Musculoskeletal pain
  - Cellulitis
  - Baker cyst
  - Torn gastrocnemius muscle
  - Fracture
  - Hematoma
  - Acute arterial ischemia
  - Lymphedema
  - Hypoproteinemia (e.g., cirrhosis, nephrotic syndrome)

Possible Causes of Dyspnea or Chest Pain

- Musculoskeletal pain
- Pleuritis
- Costochondritis
- Rib fracture
- Pericarditis
- Angina pectoris
- Salicylate intoxication
- Hyperventilation

Figure 1. Differential Diagnoses

Note. Based on information from Kane et al., 2004.

Table 1. Risk Factors for Venous Thromboembolism

Risk Factor	Risk	Incidence (%)
Age	1.9 every 10 years from age 20–80	–
Surgery		
Hip or knee	–	48–61
Neurosurgery	–	24
General	–	19
Trauma	–	58
Femoral catheter placement	–	12
Malignancy (lung cancer, gastrointestinal tumors)	–	15
Previous thromboembolism	–	2–9
Primary (genetic) hypercoagulable states		
Antithrombin, protein C/S deficiency	10-fold	–
Factor V Leiden	4-fold	–
Prothrombin 20210A	4-fold	–
Increased factor VIII	6-fold	–
Hyperhomocysteinemia (high blood levels of homocysteine)	2.5–4-fold	–
Family history	2.9-fold	–
Oral contraceptive use	2.9-fold	–
Estrogen replacement	2–4-fold	–
Immobilization		
Bed rest of three days	–	Start of increased incidence
Confinement of one, two, and four weeks	–	15, 77, and 94 increase
Prolonged air or other confined travel	Unclear	Unclear
Pregnancy	–	0.075% of pregnancies
Postpartum	–	2.3%–6.1% per 1,000 deliveries
Antiphospholipid antibodies	2–6-fold	–
Inflammatory bowel disease	–	1.2–7.1
Obesity, varicose veins, heart attack, congestive heart failure	Variable	Variable

Note. Based on information from Kyrle & Eichinger, 2005; Meissner & Strandness, 2001; Stanford School of Medicine, 2007.

How is deep vein thrombosis diagnosed?

Once presenting symptoms of DVT are noted, a pulse oximetry measurement should be obtained. Assess all extremities for redness, swelling, or tenderness. If a patient reports dyspnea or oxygen saturation has decreased, a ventilation-perfusion scan or spiral CT scan should be performed emergently to determine the presence of pulmonary embolus. Supplemental oxygen should be provided if the oxygen saturation is subnormal or the patient complains of dyspnea. The sophisticated use of risk stratification models, D-dimer measurement, and duplex ultrasonography correctly identifies most DVT. If DVT is suspected in an extremity, an ultrasound of that extremity should be obtained. Ultrasonography has a fairly high (~90%) sensitivity and specificity for DVT in symptomatic patients but is much less effective (~50%) in detecting DVT in the calf veins of asymptomatic patients (Katz & Hon, 2004; Qaseem et al., 2007).

Recent interest has focused on the use of D-dimer in the diagnostic approach to DVT. D-dimer fibrin fragments are present in fresh fibrin clot and in fibrin-degradation products of cross-linked fibrin. Monoclonal antibodies specific for the D-dimer fragment are used to differentiate a fibrin-specific clot from non-

cross-linked fibrin and fibrinogen. The specific attributes of the D-dimer antibodies account for their high sensitivity for VTE. D-dimer levels may be elevated in any medical condition in which clots form, such as trauma, recent surgery, hemorrhage, cancer, and sepsis. Many of the conditions are associated with higher risk for DVT. The D-dimer assays have low specificity for DVT; therefore, they should be used only to rule out DVT, not to confirm its diagnosis. D-dimer levels remain elevated in DVT for about seven days. Patients presenting late in their course, after clot organization and adherence have occurred, may have low levels of D-dimer. Similarly, patients with isolated calf-vein DVT may have a small clot burden and low levels of D-dimer below the analytic cut-off value of the assay, which accounts for the reduced sensitivity of the D-dimer assay in the setting of confirmed DVT (Kyrle & Eichinger, 2005; Schreiber, 2002). The use of a high-sensitivity D-dimer assay in patients who have a low pretest probability (see Table 2) of VTE has a high negative predictive value; that is, it will accurately identify certain patients who have a low likelihood of VTE. D-dimer is most useful in younger patients with low pretest probability, no associated comorbidities or previous DVT, and a recent onset of symptoms (Qaseem et al., 2007; Wells et al., 2002).

What are the current treatment guidelines for management of deep vein thrombosis?

Acute DVT is considered uncomplicated if no pulmonary embolus or contraindications to outpatient management exist (Buller et al., 2004; Snow et al., 2007). Uncomplicated DVT can be managed with enoxaparin 1 mg/kg subcutaneously every 12 hours or 1.5 mg/kg subcutaneously every 24 hours (Knovich & Lesser, 2004). For complicated thrombus, including pulmonary embolus, most patients are admitted to an acute care inpatient unit and given an unfractionated IV heparin drip and subsequently converted to enoxaparin 1 mg/kg subcutaneously every 12 hours (National Comprehensive Cancer Network, 2006). Buller et al. recommended against the routine use of an inferior vena cava filter in addition to anticoagulants for the initial treatment of DVT unless a contraindication for or complication of anticoagulant treatment exists or thromboembolism is recurrent despite adequate anticoagulation. Inferior vena cava filters rarely are used in the neuro-oncology population because they have a risk of complications exceeding 50% (Gerber et al., 2006; Van Ha, 2006). Low-molecular-weight heparin is more effective in preventing recurrent thrombus in patients with malignancy (Lee et al., 2003). Newer treatments such as catheter-directed thrombolysis have not been used routinely in patients with brain tumors but, at some point, may play a role in the treatment and prevention of a thrombus.

Neuro-oncology patients who present with a generalized seizure are placed on antiepileptic therapy for at least six months. If no further seizures occur and the primary tumor is resected, patients can be evaluated and possibly weaned from antiepileptic therapy. Enzyme-inducing antiepileptics such as phenytoin have multiple drug-drug interactions. Although warfarin has been used effectively in patients with cancer diagnoses, managing warfarin administration in patients with brain tumors receiving antiepileptics is difficult because of drug-drug and drug-food interactions (Preumer, 2006) (see Figures 2, 3, and 4). As a result, enoxaparin often is the preferred method of anticoagulation (Snow et al., 2007) because it has fewer interactions with foods and medications. If surgical procedures are needed during anticoagulant therapy, enoxaparin can be stopped safely the day before surgery (sanofi-aventis, 2006).

What are deep vein thrombosis prevention guidelines?

Currently, postoperative neuro-oncology patients are given enoxaparin 40 mg subcutaneously daily with or without sequential compression device support because of their increased risk of VTE postcraniotomy

Table 2. Pretest Probability Rule for Diagnosing Deep Vein Thrombosis

Clinical Characteristic	Score[a]
Active cancer or cancer treatment within previous six months	1
Paralysis, paresis, recent immobilization of lower extremities	1
Recently bedridden for more than three days or major surgery within 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of deep venous system	1
Entire leg swollen	1
Calf swelling 3 cm larger than asymptomatic side	1
Pitting edema confined to symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis at least as likely as deep vein thrombosis	–2

[a] Adding the score will result in the clinical pretest probability of deep vein thrombosis (low ≤ 0, intermediate 1–2, high ≥ 3).

Note. Based on information from Wells et al., 2002, 2003.

Drugs and Foods That Increase International Normalization Ratio or Bleeding Risk

Acetaminophen
Alcohol
Amiodarone
Anabolic steroids
Antifungal medications (i.e., "azoles")
Aspirin and salicylates
Cephalosporins
Chloral hydrate
Cimetidine
Clofibrate
Cranberry juice (CYP2C9 inhibitor)
Danazol
Diflunisal
Disulfiram
Fluvoxamine
Ginkgo biloba
Heparin
Hydroxymethylglutaryl-coenzyme A reductase inhibitors
Isoniazid
Macrolides
Metronidazole
Nalidixic acid
Nonsteroidal anti-inflammatory drugs
Omeprazole
Paroxetine
Penicillin
Propafenone
Quinidine
Quinolones
Sulfinpyrazone
Tamoxifen
Tetracycline
Thyroid hormone
Ticlopidine
Trimethoprim-sulfamethoxazole
Vitamin E

Drugs That Decrease International Normalization Ratio or Increase Clotting Risk

Barbiturates
Binding resins
Carbamazepine
Oral contraceptives
Penicillin
Rifampin
Vitamin K
American ginseng
St. John's wort

Drugs With a Variable Effect on International Normalization Ratio or Bleeding Risk

Allopurinol
Corticosteroids
Phenytoin

Figure 2. Warfarin Drug-Drug Interactions

Note. Based on information from Gage et al., 2000; Greenblatt & von Moltke, 2005; Sanoski & Bauman, 2002.

(Goldhaber, Dunn, Gerhard-Herman, Park, & Black, 2002). In at-risk nonsurgical patients, prophylaxis can be achieved by physical methods (e.g., early ambulation postoperative, graduated compression stockings, intermittent pneumatic compression) or with anticoagulant drugs (e.g., unfractionated heparin, warfarin, low-molecular-weight heparin). Physical methods alone are indicated in low-risk patients and those with contraindications to anticoagulants, but individuals at moderate to high risk for thrombosis need anticoagulation. Unfractionated heparin and warfarin are inconvenient for patients and medical staff because of frequent injection schedules and required laboratory monitoring of clotting times; therefore, low-molecular-weight heparin is the drug of choice (Kyrle & Eichinger, 2005). The National Comprehensive Cancer Network (2006) offers evidence-based practice guidelines on the prevention, diagnosis, and treatment of VTE.

Antibiotic or Antifungal Drugs
Azoles[a]
Carbenicillin
Cephalosporins
Clarithromycin
Erythromycin
Isoniazid
Metronidazole
Quinolones
Tetracycline
Trimethoprim-sulfamethoxazole

Analgesic or Anti-Inflammatory Drugs
Acetaminophen[b]
Aspirin
Allopurinol
Propoxyphene
Nonsteroidal anti-inflammatory drugs[c]
Sulfinpyrazone
Zafirlukast

Antiarrhythmic Drugs
Amiodarone
Propafenone
Quinidine

Miscellaneous
Alcohol
Anabolic steroids
Chloral hydrate
Cimetidine
Clofibrate
Disulfiram
Heparins
Omeprazole
Phenytoin[d]
Simvastatin
Tamoxifen
Thyroxine

[a] Including fluconazole, itraconazole, ketoconazole, and miconazole

[b] The effect is dose dependent.

[c] Many, including celecoxib and rofecoxib, raise the international normalization ratio.

[d] This drug raises the international normalization ratio initially, but later it may lower the ratio.

Note. Several drugs (e.g., rifampin, barbiturates, anticonvulsants) may reduce warfarin's effects; thus, the international normalization ratio may increase when a patient stops taking the medication.

Figure 3. Drugs That Increase Warfarin's Effect

Note. Based on information from Bristol-Myers Squibb, 2006; Buller et al., 2004.

Asparagus
Avocado
Broccoli
Brussels sprouts
Cauliflower
Cabbage
Chick peas or garbanzo beans
Dark green, leafy vegetables
Egg yolks (more than four per week)
Green tea
Kale
Lentils or dried peas and beans
Liver
Onions (more than 1/4 C per serving)
Spinach
Soybeans
Seaweed

Figure 4. Foods Rich in Vitamin K

Note. Based on information from Greenblatt & von Moltke, 2005.

What are the nursing implications for patients diagnosed with deep vein thrombosis?

Neuro-oncology nurses are in a pivotal position to assist patients with brain tumors in recognizing the signs and symptoms of DVT and in practicing preventive measures when they are in at-risk situations. Awareness of evidence-based guidelines will help nurses direct patients to appropriate prevention and treatment of VTE disease. Patients who develop DVT while undergoing cancer treatment should be educated carefully (see Figure 5) on anticoagulant therapy, drug interactions, avoiding foods high in vitamin K, moderation of alcohol consumption, and international normalization ratio (INR) monitoring. Patients who are on oral anticoagulation therapy require regular blood tests to determine prothrombin time and INR. Dose adjustments are made according to INR results, which generally should be maintained from 2.5–3.5 for therapeutic effect. Injection techniques for low-molecular-weight heparin therapy and the importance of maintaining frequent contact with oncology nurses during anticoagulant therapy also should be reviewed with patients. A current medication list should be documented in the medical record and evaluated for possible drug-drug interactions with warfarin. A dietary review should include discussions about foods high in vitamin K that may interact with warfarin and moderation of alcohol consumption. Nurses in oncology settings should ensure that resources are available so that patients reporting symptoms of DVT are evaluated and treated quickly to prevent progression of DVT to pulmonary elbolus, which has a high mortality rate.

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Prevention
Ambulate at least three times a day.
Avoid prolonged bed rest or periods of immobility during the day.
Take rest breaks during long plane or car trips.
Be aware of the signs and symptoms of thrombus, including shortness of breath, chest pain, back pain, swelling, or redness of or pain in the extremity.
Symptoms need prompt evaluation via the emergency department or an urgent outpatient evaluation.

Treatment
Review subcutaneous injection teaching by demonstration and reinforce with written referral materials.
Review potential side effects of enoxaparin such as bruising or hematoma and transient burning pain during injection.
Review administration of warfarin, including routine international normalization ratio monitoring, drug-drug interactions, and avoiding foods rich in vitamin K.
Counsel patients that alcohol consumption affects how the liver metabolizes warfarin and can dangerously raise the international normalization ratio; therefore, limit alcohol intake to no more than one or two drinks per day.
Provide instructions for emergency care in the event of a bleeding episode.

Figure 5. Patient and Caregiver Teaching Points

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Clinical Highlights: Venous Thrombosis

Definition

Venous thrombosis is the formation or presence of a blood clot in a vein.

Incidence

Approximate annual deep vein thrombosis (DVT) incidence rates by age group are as follows: 2–3 per 10,000 aged 30–49, 5 per 10,000 aged 50–59, 10 per 10,000 aged 60–69, and 20 per 10,000 aged 70–79. The incidence of DVT is very strongly related to age and is comparable overall in men and women (Fowkes, Price, & Fowkes, 2003). The exact incidence of DVT is unknown because most studies are limited by the inherent inaccuracy of clinical diagnosis. Existing data that underestimate the true incidence of DVT suggest that about 80 cases per 100,000 people occur annually. DVT develops in approximately 1 in 20 people, and 600,000 are hospitalized annually for DVT in the United States (Hirsch & Hoak, 1996). In hospitalized patients, the incidence of venous thrombosis is considerably higher and ranges from 20%–70%. Death from DVT is attributed to massive pulmonary embolism, which causes 200,000 deaths annually in the United States (Schreiber, 2006). Patients with cancer are seven times more likely to develop clinically significant venous thrombooembolism (VTE) compared to the general population; in patients with metastatic disease, the risk may be as high as 20-fold (Blom, Doggen, Osanto, & Rosendaal, 2005).

Pathophysiology

Increased tissue factor or protein secretion caused by malignancy leads to hypercoagulability and immobility (Lin, Wakefield, & Henke, 2006). Virchow's triad (venous stasis, vessel wall injury, and hypercoagulable state) is the primary mechanism for the development of venous thrombosis (Brotman, Deitcher, Lip, & Matzdorff, 2004), although the relative importance of each factor continues to be debated. DVT develops through activation of coagulation in areas of reduced blood flow. A vast majority of calf vein thrombi dissolve completely without therapy, and approximately 20% propagate proximally (Line, 2001).

Differential Diagnosis

Current clinical practice emphasizes the diagnosis of DVT, owing to the serious risk of pulmonary embolus. Many

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musculoskeletal and venous disorders may present with a similar clinical picture and require careful evaluation to avoid inappropriate investigation and management (Kane, Balint, Gibney, Bresnihan, & Sturrock, 2004). A prediction rule for diagnosis of DVT can help to determine the probability of VTE before further testing (Qaseem et al., 2007; Wells et al., 2002, 2003).

Diagnosis

When symptoms of DVT exist, a pulse oximetry measurement should be obtained. Assess all extremities for redness, swelling, or tenderness. If the patient reports dyspnea or oxygen saturation has decreased, a ventilation-perfusion scan or spiral computed tomography scan should be performed emergently to determine the presence of pulmonary embolus. The sophisticated use of risk stratification models, D-dimer measurement, and duplex ultrasonography correctly identifies most DVT. If DVT is suspected in an extremity, an ultrasound of the affected extremity should be performed (Katz & Hon, 2004; Qaseem et al., 2007).

Treatment

DVT management options include anticoagulant therapy or insertion of an inferior vena cava filter. Inferior vena cava filters rarely are used in the neuro-oncology population because the risk of complications exceeds 50% (Gerber, Grossman, & Streiff, 2006; Van Ha, 2006). Anticoagulant therapy can be given orally or by subcutaneous injection of low-molecular-weight heparin (LMWH). Enoxaparin is an LMWH commonly used in inpatient and outpatient settings (Bates & Weitz, 2006; Snow et al., 2007). The dose is based on weight and established at 1 mg/kg subcutaneously every 12 hours (Buller et al., 2004). A multisite randomized trial comparing twice-daily standard enoxaparin administration with once-daily 1.5 mg/kg showed equivalency between LMWH dosing regimens and a dose-adjusted continuous infusion of unfractionated heparin in patients with symptomatic DVT (Merli et al., 2001). Patients who are unable to take enoxaparin for fear of injections or financial reasons can receive warfarin; however, international normalization ratio (INR) values must be monitored at least two to three times per week during the initial weeks of dosage adjustment until the warfarin dose and INR are within the recommended therapeutic range of 2.5–3.5 (Bristol-Myers Squibb, 2006).

Patient Education

Nurses should counsel patients and their caregivers at initial diagnosis about the increased risk of thrombosis associated with any malignancy. Recommendations for the prevention of thrombus should be reviewed. Many neuro-oncology patients have memory deficits because of tumor location or treatment, which is a consideration. Patients with primary high-grade gliomas should be instructed to call a triage nurse or oncologist with any worrisome symptoms for an evaluation. If a patient is on warfarin, INR should be checked frequently and the patient should receive a list of all medications or foods that may interact with the drug. Patients also should be counseled to notify their dentists, surgeons, and other healthcare providers that they are on warfarin or other anticoagulant therapy. Patients on anticoagulant therapy should be aware of bleeding precautions, and an emergency plan should be in place in the event of heavy bleeding (epistaxis, rectal, or vaginal bleeding).

Nursing Implications

Patient and family education on prevention, prompt reporting of signs and symptoms of DVT, assessment, and follow-up are of utmost importance. Patients who develop DVT while under care for cancer should be educated carefully about anticoagulant therapy, drug and food interactions, INR monitoring, injection technique for LMWH therapy, and the importance of keeping in close contact with an oncology nurse during anticoagulant therapy. A current medication list should be included in the medical record and evaluated for possible drug-drug interactions. A dietary review should include discussions about foods that may interact with warfarin. Nurses who receive calls from patients reporting symptoms of DVT should ensure adequate resources so that patients are evaluated and treated quickly to prevent progression of DVT to pulmonary embolus, which has a high mortality rate.

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