Patients with cancer have an increased risk of venous thromboembolism (VTE). Several factors contribute to the thrombogenic risk for this patient population, including stasis, hypercoagulability from treatment or the tumor itself, and vascular endothelial damage from neoplastic disease or procedures performed on patients. Treatment of VTE for patients with cancer usually involves a sequential combination of unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH), followed by oral warfarin therapy or LMWH for continued anticoagulation. The goal of this treatment is to protect against further complications with the existing thrombus or clot and to reduce or protect against further embolic events. Despite appropriate treatment with oral anticoagulant therapy, many patients with cancer suffer from recurrent thrombosis. Reasons for inadequate anticoagulant response of patients with cancer include the possibility of Trousseau syndrome, a condition characterized by persistent low-grade intravascular coagulation (Callander & Rapaport, 1993). Additionally, patients with cancer have increased bleeding complications compared with patients without malignancy (Schafer, Levine, Konkle, & Kearon, 2003).

Anticoagulation Treatment in Patients With Cancer

Anticoagulation treatment in patients with VTE and cancer may be challenging secondary to the high risk of bleeding in these patients and the high rate of recurrence even with therapeutic anticoagulation regimens (Hutten et al., 2000). Historically, treatment of VTE in patients with cancer mimics management of VTE in the general population. Treatment traditionally was initiated with UFH, with eventual conversion to a vitamin K antagonist such as warfarin. Use of UFH requires frequent testing of adjusted prothrombin time (aPTT) to determine the therapeutic level of drug, necessitating patients to undergo laboratory evaluation and venipuncture.

Understanding in the structure of heparin has led to the development of LMWH, synthetic heparinomimetics, antithrombin (AT), and anti-Xa agents that now compete with UFH for specific indications, including management of VTE. The high risk for recurrence of thrombosis in cancer has initiated re-evaluation of historic strategies of long-term anticoagulation therapy with vitamin K antagonists versus long-term treatment with LMWH (Lee et al., 2003). The indications that LMWH may have direct antitumor effects also have increased support of these agents for patients with VTE and cancer (Levine, Lee, & Kakkar, 2003). A recent study looked at patients with advanced malignancy (N = 385) receiving dalteparin once daily versus placebo (Kakkar et al., 2004). Although LMWH did not significantly improve one-year survival, a subgroup of patients with a better prognosis was observed to have impaired survival, suggesting a possible direct effect of dalteparin on biology of the tumor (Kakkar et al.).