Pathophysiology of Venous Thromboembolism
Specific systemic conditions predispose individuals with cancer to VTE development. These conditions were first described by Rudolph Virchow in 1859 and are known as Virchow’s Triad, which consists of hypercoagulability, vessel wall or endothelial injury, and stasis (Kesieme, Kesieme, Jebbin, Irepkita, & Dongo, 2011).

Prothrombotic mechanisms respond to the presence of a tumor, causing inflammation, necrosis, and hemodynamic changes, which can also be exacerbated by MM therapy (Kristinsson, 2010). In addition, clot-promoting molecules with procoagulant and fibrinolytic activities are secreted by tumor cells, causing an interaction with endothelial cells. This plays an important role in pathogenesis. In addition, venous stasis reduces the clearance of activating coagulation factors, causing endothelial cell damage and increasing the risk for VTE (Elyamany, Alzahrani, & Bukhary, 2014; Kesieme et al., 2011).

Immunomodulatory drugs have various anti-angiogenic and anti-inflammatory effects that can alter the interaction between the tumor cells and the bone marrow microenvironment. Although the exact reason why these drugs increase the risk of VTE is unknown, it is thought that they can enhance the expression of tissue factor and vascular endothelial growth factor and, by downregulating thrombospondin, cause cytokine-mediated activated protein C resistance (Kristinsson, Bjorkholm, Schulman, & Landgren, 2011).

Risk Factors for Venous Thromboembolism in Multiple Myeloma
VTE incidence is higher among patients diagnosed with monoclonal gammopathy of undetermined significance (an asymptomatic premalignant clonal plasma cell or lymphoplasmacytic proliferative disorder associated with an increased risk of developing MM