Gastrointestinal stromal tumors (GISTs) are a type of soft-tissue sarcoma, with 4,500–6,000 new cases reported per year in the United States. At the time of diagnosis, about 1,500 of these tumors already have metastasized (American Cancer Society, 2010; Nilsson et al., 2005; Tran, Davila, & El-Serag, 2005; Tryggvason, Gislason, Magnusson, & Jonasson, 2005). Median age at diagnosis is about 60 years and risk is slightly higher in men than in women; about 50% of all cases occur in the stomach, another 33% occur in the small intestine, and the remainder are found in the colon, rectum, or esophagus (Joensuu et al., 2002; Nilsson et al., 2005; Perez et al., 2006; Tran et al., 2005; Tryggvason et al., 2005).

Most GISTs (85%–95%) are driven by oncogenic mutations in either KIT or platelet-derived growth factor receptor alpha protein (PDGFRα). These gain-of-function mutations lead to the constitutive activation of the KIT and PDGFR receptors, which result in subsequent cell proliferation and the prevention of apoptosis (programmed cell death) (Fletcher et al., 2002). The observation that most GISTs express KIT or PDGFRα led to the development of the targeted oral therapy imatinib, a tyrosine kinase inhibitor (TKI) (Joensuu et al., 2002). Imatinib inhibits the constitutive activation of KIT and PDGFRα tyrosine kinase, which inhibits tumor growth and induces tumor regression (Hirota et al., 1998; Rubin et al., 2001).

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