Acute promyelocytic leukemia (APL), a subtype of acute myeloid leukemia (AML), was once considered the most fatal form of AML because of its significant propensity for bleeding and the subsequent high mortality rate associated with early hemorrhagic death (Coombs, Tavakkoli, & Tallman, 2015). However, advances in the understanding of the disease process and improvements in the available therapies since the 1980s have led to it being considered the most curable (Peterson, Trautman, Hoffner, & Zakrocki, 2013). APL makes up 5%–10% of all cases of AML (Peterson et al., 2013) and appears to be more common in patients of Latin American descent, representing about 20%–25% of AML cases in Latin countries (Ribeiro & Rego, 2006). In the United States, the incidence of APL diagnoses is estimated to be 600–800 cases per year. A diagnosis of APL is considered to be highly unlikely in children and is most commonly seen in adults in midlife. Cases are most commonly diagnosed in adults ranging from 20–50 years of age (Ribeiro & Rego, 2006).

A key characteristic of APL is the presence of atypical promyelocytes, both in the bone marrow and in the peripheral blood. This disease is distinguished from other forms of AML by the cytogenetic translocation of the long arms of chromosomes 15 and 17 (Wang & Chen, 2008). One of the genes responsible, the promyelocyte leukemia, or PML gene, is on chromosome 15 and is thought to be responsible for apoptosis and tumor suppression. The other gene responsible, the retinoic acid receptor-alpha, or RARα, is on chromosome 17 and is mostly responsible for myeloid differentiation. The translocation of genetic material that occurs between these two chromosomes creates a fusion between parts of the PML gene to parts of the RARα gene. This chromosomal translocation of genetic material causes neither the PML nor the RARα protein to act in its original capacity. Because of this morphology of chromosomes, blood cells cannot differentiate past the promyelocyte phase and, thereby, result in both the bone marrow and the peripheral blood being filled with promyelocytes (Peterson et al., 2013; Walker & Held-Warmkessel, 2010).

The clinical presentation of APL, like that of AML, can be nondescript, with many patients experiencing days to weeks of nonspecific symptoms followed by the occurrence of