Chronic Myelogenous Leukemia

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Chronic myelogenous leukemia (CML) represents about 14% of all leukemias and occurs with a frequency of about 1 in 100,000. It is rare in children. Symptoms include fatigue, weight loss, sweating, and abdominal discomfort from an enlarged spleen. The white blood cell count can range from 100–600 ul. CML has three phases: the chronic phase, accelerated phase, and blast phase. Most patients are diagnosed during the chronic phase. Ionizing radiation has been implicated in some cases of CML, but in most individuals no cause is known. The Philadelphia chromosome, an acquired genetic mutation represented by a translocation of chromosome 22 and chromosome 9, drives the leukemic changes in CML. Imatinib mesylate, a tyrosine kinase inhibitor, was approved in 2002 for the treatment of all phases of CML. Because of its effectiveness, imatinib has become the treatment of choice for most patients with CML. Stem cell transplantation also is an option for eligible patients. It is the only curative treatment for CML. Two drugs under study for patients who cannot tolerate or who become resistant to imatinib are BMS-354825 and AMN107. Oncology nurses who are knowledgeable about new therapies for CML can be effective resources for their patients.

Largely unknown as a disease entity until the mid-1800s, leukemia often was mistaken for other illnesses such as infection and dropsy. In 1827, Velpeau wrote about a patient who presented with symptoms of fever, abdominal discomfort, and fatigue. It probably was the first documented case of leukemia. Unfortunately, Velpeau's observations were not acknowledged at the time (Henderson, Lister, & Greaves, 2002).

About 20 years later, Virchow recognized Velpeau's work and used the term "weisses blut" or "white blood." About that time, Barth and Craigie sent blood samples of patients with symptoms similar to Velpeau's patient to Bennett and Donne, pioneers in clinical microscopy, providing further awareness of leukemia as a disease entity. That provided further insight into the developing awareness of leukemia as a disease entity. In 1878, Neuman suggested that the bone marrow was not only the place where blood cells were formed but also where leukemia originated. He was the first to use the term myelogenous to describe leukemia. Then in 1960, a discovery that would greatly influence later treatment of chronic myelogenous leukemia (CML) was made by Nowell and Hungerford, who described an abnormality in two patients with chronic myelogenous leukemia that involved a shortened chromosome 22. The abnormality was named the Philadelphia chromosome (Beutler, Lichtman, Coller, Kipps, & Seligsohn, 2001).

Epidemiology and Incidence

CML represents 14% of all cases of leukemia (Leukemia and Lymphoma Society, 2004). The worldwide incidence rate of CML is about 1 case per 100,000 of population, with a slightly higher rate in men (Henderson et al., 2002). About 3% of childhood leukemias are CML, and 10% of cases of CML occur in children aged 5–20 years (Beutler et al., 2001). See Figure 1 for age-specific incidence rates.

Causes and Risk Factors

CML is the result of an acquired mutation to a single blood cell. The cause of the change in most cases is not known; however, ionizing radiation has been implicated in some cases. The acquired change in the cell produces the Philadelphia chromosome, which is a result of a translocation between chromosome 22 and chromosome 9 (Beutler et al., 2001).

The gene that breaks off from chromosome 9 is called ABL, for Abelson who was the scientist who discovered the gene. Likewise, the gene that splits from chromosome 22 is named BCR, for breakpoint cluster region. The mutated ABL gene fuses with what is left of the severed BCR gene on chromosome 22 and forms an abnormal fusion gene called BCR-ABL. The abnormal BCR-ABL gene produces a protein called tyrosine kinase, which causes

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