Hairy cell leukemia (HCL) first was described in 1958 by Bouroncle, Wiseman, and Doan, who called it leukemic reticuloendotheliosis. In 1966, Schrek and Donnelly changed the name to hairy cell leukemia, describing its unique appearance. The hairy cell is a B lymphocyte; little is known about its pathology (Goodman, Burian, Kozioł, & Saven, 2003). HCL accounts for 2% of all leukemias, and 600 new cases are diagnosed each year. Men are four times more likely to be diagnosed with HCL than women, and the mean age of onset is 52 years. HCL occurs primarily in Caucasians; Jewish men are overrepresented (Goodman, Bethel, et al., 2003).

Background

HCL is a monoclonal proliferation of relatively mature B lymphocytes, typically expressing monoclonal immunoglobulin G on their cell surfaces and having unique immunoglobulin gene arrangement. Hairy cells also coexpress the pan B-cell antigens CD19, DC20, and CD22 (Goodman, Bethel, et al., 2003). B lymphocytes in adults are processed in bone marrow and manufacture antibodies. Each B lymphocyte has on the surface of its cell membrane 100,000 antibody molecules that react specifically to one type of antigen. With the disruption of the B lymphocyte in HCL, immunity is disturbed and infections are common (Guyton, 1991).

Serum levels of soluble interleukin-2 (IL-2) are high in HCL and correlate with disease activity. The abnormal cells do not produce IL-2; however, they do produce tumor necrosis factor and a B-cell growth factor (Goodman, Bethel, et al., 2003).

The cause of HCL is unknown. Genetic and viral origins have been studied without any associations noted. Patients with HCL have been found to have a higher previous occupational exposure to ionizing radiation and organic chemicals (Goodman, Bethel, et al., 2003).