Hodgkin’s disease

Hodgkin’s disease (or Hodgkin’s lymphoma) is a form of cancer that involves the lymphatic system and can be distinguished from non-Hodgkin’s lymphomas by cancer cell type. The American Cancer Society estimates that there will be approximately 7,880 new cases of this disease in the U.S. in 2004, accounting for less than 1% of all cancer types, and approximately 1,320 deaths (ACS, 2004). Because of substantial improvement in effective therapy for this disease, mortality rates have decreased approximately 60% since the early 1970s (ACS, 1999).

Epidemiologic studies have shown that Hodgkin’s disease is more common among men than women and more common among whites than blacks. People of Jewish descent appear to be at higher risk of Hodgkin’s disease compared to people of non-Jewish descent (Mueller, 1996). Although the disease is relatively rare among children, two peaks in the age distribution have been observed for this cancer type. The first peak occurs in young adults usually between the ages of 15 to 40 (typically ages 25-30) and the second peak occurs in adults aged 55 years and above.

No major risk factors for Hodgkin’s disease have been found (ACS, 1999). However, the clinical and cellular features of Hodgkin’s disease suggest a chronic infectious process (Mueller, 1996). The bimodal age distribution of this disease suggests that two distinct etiologies (or causes) for Hodgkin’s disease may be involved for each group. Researchers have proposed that among young adults, Hodgkin’s disease is caused by a biological agent of low infectivity. Among individuals of older ages, the cause is probably similar to those of other lymphomas (Mueller, 1996). The virus that has been linked most specifically to this disease is the Epstein-Barr virus (EBV). EBV, a herpesvirus, is common in the general population and causes mononucleosis or “mono.” Approximately 40% to 50% of Hodgkin’s disease cases are associated with EBV (Weiss, 2000). In addition, several studies have also shown that young adults who have developed infectious mononucleosis have a significantly higher risk of developing Hodgkin’s disease (ACS, 1999). However, the absence of EBV infection in about half the cases and the high prevalence of EBV in the general population suggest that EBV may be only one of several factors in the development of this cancer. Although cytomegalovirus (CMV) and the more recently identified human herpesvirus type 6 have been considered as possible factors in the development of Hodgkin’s disease, results of antibody studies are inconsistent and these viruses do not appear to be related to risk of Hodgkin’s disease (Mueller, 1996).

Slightly higher rates of Hodgkin’s disease occur among people with reduced immunity, such as those with AIDS, people with congenital immune deficiencies, and individuals on immunosuppressant medication following organ transplants. However, Hodgkin’s disease occurs at a much lower rate than non-Hodgkin’s lymphomas among this group of individuals (ACS, 1999).

Hodgkin’s disease trends in the young adult population reveal that the disease has become increasingly associated with populations both of middle to higher socioeconomic status and small family size. These factors are consistent with susceptibility to late infections with common childhood viruses, supporting the theory that Hodgkin’s disease is associated with an infectious agent (Mueller, 1996). Occupational exposures to workers in the chemical industry and woodworkers have also been suggested in several epidemiologic studies to be associated with the development of Hodgkin’s disease. However, specific chemical exposures related to the development of this disease have not been identified and results of studies investigating occupational exposures are inconsistent (Mueller, 1996). Based on an examination of medical
and scientific literature, the American Cancer Society concludes that although the exact cause remains unknown, Hodgkin’s disease does not seem to be caused by genetic, lifestyle (e.g., dietary), or environmental factors (ACS, 1999).

References


