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Editorial
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Familial Hodgkin's Disease

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Editorial

The incidence of Hodgkin Lymphoma (HL) has increased among adolescents and young adults in recent decades, but the relevant risk factors in early life are still unknown. In most developed nations, the age-specific incidence HL has a bimodal pattern, with an initial peak occurring among young adult's ages 15-39 years, followed by a second peak among older adults. In developing nations, the first peak occurs earlier, among children under age 15 years [1]. HL has long been thought to have an infectious predisponding factor due to Epstein-Barr virus infection (EBV), being most likely etiologic candidate agent [1]. EBV is a typically benign lymphotropic herpesvirus that infects more than 90% of the world's population, during childhood. The causal role of EBV in HL is supported by findings of elevated levels of its antibodies in individuals before HL onset, as well as by the molecular detection of monoclonal EBV genome and viral products in the malignant cells of 25% to 50% of HL tumors [1]. A history of infectious mononucleosis increases the risk of young adult HL by about 3-fold. Other recent studies have addressed HL by familial types, histology, age at diagnosis, and gender. Recently, Kharazmi, et al. [2] studied a cohort of 57 475 first-degree relatives of 1392 HL patients diagnosed between 1955 and 2009 in 5 European countries, aiming to determine HL familial form, incidence. The overall lifetime cumulative risk (CR) of HL in first-degree relatives of a patient with HL was estimated 0.6%, which represents a (3-fold) increased risk over the general population risk. The risk in siblings was (4.8-7.4-fold). This was significantly higher than among parents and/or children (2.1-2.6-fold). Very high lifetime risk of HL was found for those with multiple affected first-degree relatives, (13-fold) and for same- gender twins (57-fold). They also report high familial risks between some concordant histologic subtypes of HL such as lymphocyte-rich (81-fold) and nodular sclerosis (4.6-fold) and between some discordant subtypes. The familial risk in sisters (9.4-fold) was higher than in brothers (4.5-fold) or unlike-gender siblings (5.9-fold). The lifetime risk of HL was higher when first-degree relatives were diagnosed at early ages (before age 30 years) [2]. This study provided tangible risk estimates for relatives of HL patients, which can be used as a risk calculator for classical HL by oncologists and genetic counselors. More, genetic researchers speculate a non-Mendelian segregation with genomic, parental imprinting to be the likely mechanism in these familial cases [3]. The risk stratification can help families and physicians to raise their awareness towards possible early detection of another case of HL among members of the same family.

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