Hodgkin lymphoma different stages and risk factors.

Goy Harris*

Department of Clinical Pharmacy, Jordan University of Science and Technology, Irbid, Jordan

Introduction

The presence of typical bi/multinucleated Reed-Sternberg cells and their variations, mononucleated Hodgkin cells, together known as Hodkin Reed-Sternberg cells, characterises Hodgkin lymphoma (HL), a rare monoclonal lymphoid cancer (HRS). In HL instances, the majority of HRS cells (85% and 100%, respectively) express CD15 and CD30. By using single cell PCR to identify Ig gene rearrangements, HRS cells have B-cell origins ; and somatic mutations in the heavy and light chain Ig genes' variable area are indicative of a B-cell origin from the late germinal or post-germinal centre [1].

While their B-cell origin, HRS cells often lack key B cell characteristics. These include particular signalling molecules connected to the B-cell lineage that are either absent or produced by a small proportion of cells, despite being derived from B-cells. HRS cells have the ability to exhibit markers that are specific to other hematopoietic lineages, such as dendritic cells, monocytes, and T-cells (5-15% of cHL cases), in addition to the variable B-cell marker expression seen by around 95% of classical Hodgkin lymphoma (cHL) patients. T-cell markers on HRS cells have been observed to be independently related with poor prognosis while being infrequently expressed.

With the exception of the relative prevalence of particular histological subtypes and the unique immune response against HRS cells in the tumour microenvironment, the biology of HL in children and adults is comparable, if not identical. Based on histologic characteristics and phenotypes, HL can be split into two major groups: (1) Classical Hodgkin lymphoma (cHL) and (2) Nodular lymphocyte predominate Hodgkin lymphoma (NLPHL). Four more subtypes of cHL exist: lymphocyte depletion (LD), mixed cellularity (MC), nodular sclerosis (NS), and lymphocyte rich (LR) (LD). NLPHL is thought to be a unique disease entity that resembles B-cell non-HL more so than cHL. As a result, the focus of this review will be cHL [2].

A cure rate of more than 95% has been reached for children with HL thanks to the development of current sophisticated treatment and imaging technologies. However, relapse/refractory (RR) individuals have a poor prognosis. Greater attention was paid to therapeutic adjustments that would minimise toxicity while maintaining high cure rates due to the subsequent malignancy and cardiac toxicity that can be brought on by HL treatment.

Therefore, finding predictive biomarkers in juvenile patients that correlate with clinical outcome will advance knowledge of HL pathology and probably have an impact on therapy strategies. In this review, we cover the key characteristics of paediatric cHL, prognostic biomarkers, existing medications, and novel targeted therapeutics. Additionally, we briefly discuss potential therapeutic side effects and secondary malignant neoplasms (SMNs) in HL long-term survivors.

Epidemiology and risk factors

With an usual male predominance, childhood HL accounts for 6% of all malignancies and has an incidence rate of 12 cases/million/year in the 0–14 age group. According to the demographics of different age groups, HL has a typical bimodal distribution, with the first, greater peak being evident for teenagers and young adults (15–24 year age group) and the second, smaller peak taking place for adults (around 59 years). The MC histological subtype, which is mostly linked to Epstein-Barr virus (EBV), is prevalent in early children and accounts for around 20% of HL, while the NS subtype is more common in adolescents and young adults and accounts for about 75% of HL.

Although EBV positive is not always associated with HL, in industrialised and developing nations, respectively, 30% and 90% of all HL cases have EBV-infected tumour cells. Latent membrane protein (LMP) 1 (lmp1), which causes constitutive nuclear factor-kappaB (NF-B) activation by imitating CD40 receptor, and lmp2A, which can replace the function of the B-cell receptor, are two important EBV genes involved in the aetiology of HL (BCR). Numerous observations in HL patients showed elevated antibody titers and EBV DNA detection, which are suggestive of a link between EBV and HL. Additionally, the fact that the NF-B pathway is more deregulated in EBV+ patients compared to EBV- patients supports a role for EBV in the development of HL [3].

Despite these results, it is unclear and less thoroughly researched in juvenile HL patients as to the predictive importance of EBV positive. Only a small number of researches have examined the direct prognostic importance of paediatric HL that is EBV positive, whereas many other studies have found no connection or improved clinical outcomes. Variations in the presence of EBV+ HL that are related to geography, age, ethnicity, and histological type may account for the disparate results.

***Correspondence to:** Goy Harris, Department of Clinical Pharmacy, Jordan University of Science and Technology, Irbid, Jordan, E-mail: harris@hotmail.com

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Insufficient social contact, fewer siblings, sparsely populated areas, immunodeficiency, viral infection, rapid prenatal growth, and familial HL (4.5% of cases) were all associated with an increased risk of juvenile HL in epidemiologic studies. A twin study of lymphomas found that monozygotic twins of HL patients had a risk of the disease that was about 100 times higher than that of dizygotic twins, despite the possibility of co-inheritance of genetic variations.

Current Staging

The current gold standard for staging HL in children is the Ann Arbor staging system with Cotswolds' adjustment. There are four stages in this method, with stages I and II denoting mild disease and stages III and IV denoting advanced disease. Stage I accounts for 19% of paediatric HL cases, stage II for 49%, stage III for 20%, and stage IV for 13%. On the basis of the illness stage and extent, disease bulk, and systemic B symptoms, HL patients are further divided into three risk groups (e.g., unexplained persistent fevers, weight loss, or drenching night sweats) [4].

Fluorodeoxyglucose (FDG) positron emission tomography (PET)-computed tomography is frequently used for the staging and monitoring of paediatric HL patients. FDG PET-CT integrates functional and anatomic tumour characteristics to help define radiotherapy (RT) margins and provide a baseline scan for later response assessment. The main purposes of FDG-PET for interim assessment following initial cycles of chemotherapy are to further categorise risks and to identify individuals who are either cured and do not need RT or require intensified treatment. The best definition and timing for response assessment are not agreed upon, though. Bilateral bone marrow aspirates and biopsies are also necessary for paediatric HL staging in patients with advanced stages. After therapy is finished, surveillance scans are advised to spot early relapse in high-risk individuals [5].

References

1. Nadali G, Tavecchia L, Zanolin E, et al. Serum level of the soluble form of the CD30 molecule identifies patients with Hodgkin's disease at high risk of unfavorable outcome. Blood. 2013;33(3):322-4.
2. Albelda SM. Role of integrins and other cell adhesion molecules in tumor progression and metastasis. Lab Invest 2018;10(2):173.
3. Ruco LP, Pomponi D, Pigott R, et al. Expression and cell distribution of the intercellular adhesion molecule, vascular cell adhesion molecule, endothelial leukocyte adhesion molecule, and endothelial cell adhesion molecule (CD31) in reactive human lymph nodes and in Hodgkin's disease. Am J Pathol. 2016;55(1):267-79.
4. Tacyildiz N, Yavuz G, Gozdasoglu S, et al. Serum levels and differential expression of intercellular adhesion molecule-1 in childhood leukemia and malignant lymphoma: prognostic importance and relationship with survival. Pediatr Hematol Oncol. 2017;16(1):1-5.
5. Pui CH, Hudson M, Luo X, et al. Serum interleukin-2 receptor levels in Hodgkin disease and other solid tumors of childhood. Leukemia. 1996; 348(9028):692.

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