The changing sands of diagnosis in hematopathology: Anaplastic massive cell lymphoma.

Hofman James*

Department of Hematopathology, Erasmus MC-University Medical Center Rotterdam, Netherlands

Abstract

The paradigm for defining new disease entities, Anaplastic Large Cell Lymphoma (ALCL), offers a model that can be used in many fields of pathology. Characteristic histologic characteristics and a specific immuno-phenotype were used to diagnose ALCL for the first time. Sinusoidal invasion and CD30 positivity, however, did not show to be completely specific. Following the discovery of a distinctive cytogenetic aberration, the genes responsible for the translocation were discovered, providing new information about the pathophysiology. The production of monoclonal antibodies to anaplastic large cell lymphoma kinases with aberrant expression, such as ALK-1, can be employed for diagnostic purposes and has enhanced characterization of the diagnostic entity with significant clinical and prognosis implications. The connection between ALCL and Hodgkin's disease, a different lymphoid cancer connected to CD30 expression. We now know that the true histologic range of ALCL is both narrower and wider than previously thought. Although the neoplastic cells may not appear big or anaplastic, the small cell and lymph histiocytic types of ALCL are recognised as being ALK-positive and part of the disease entity. On the other hand, the majority of cases of Hodgkin's-like ALCL have shown to be unconnected to ALCL and more closely related to the actual Hodgkin's disease.

Keywords: Hematopathology, Hodgkin's disease, Anaplastic large cell lymphoma.

Introduction

The Anaplastic Large Cell Lymphoma (ALCL) provides a model that is applicable to all fields of pathology and serves as a paradigm for the method used to describe novel disease entities. ALCL was initially identified based on specific immunophenotype (CD30+) and recognisable histologic characteristics. Sinusoidal invasion and CD30 positivity, however, did not show to be completely specific. Later, the t(2;5), a distinctive cytogenetic aberration, was discovered. This discovery provided information on the genes involved in the most frequent translocation of ALCL as well as insights into the aetiology. The ability to diagnose with the aid of monoclonal antibodies rose against the aberrantly expressed ALCL tyrosine kinase, or ALK, has improved the definition of the diagnostic entity with significant clinical and prognosis implications. The final histologic spectrum of ALCL is both wider and narrower than initially thought using these biologic methods. Our final definition of this condition will probably be limited to "T-cell" lymphomas with abnormal ALK expression [1].

When the previously linked to cases of "malignant histiocytosis" was discovered in ALCL in 1989, the pathophysiology of ALCL started to be understood. The translocation's genes were cloned in 1994 by Morris and colleagues, who discovered the gene on the chromosome to be a recently discovered tyrosine kinase known as the anaplastic large cell lymphoma kinase. Although it was later shown that ALK might have various translocation partners, the nucleophosmin gene served as the translocation partner in this instance. Polyclonal and monoclonal antibodies to the ALK kinase were produced to aid in the diagnosis of ALCL. Particularly useful for clinical investigations and standard diagnosis is the ALK-1 monoclonal antibody produced against a formalin resistant epitope of ALK [2].

In haematoxylin and eosin stained sections, the neoplastic cells typically contain an abundance of basophilic cytoplasm that appears gray-blue. Usually, a noticeable Golgi zone can be seen, staining in tissue slices as a clear or more eosinophilic zone. The Golgi region is surrounded by the nuclear lobes, which are frequently lobulated in the nucleus. These cytological characteristics have been referred to as "hallmark" traits by Delsol since ALCL is characterised by them. Smaller cells with comparable cytological characteristics may also be detected, despite the fact that they are normally huge cells [3]. Accurate diagnosis can be substantially aided with the identifying of these "hallmark" cells with experience. Some cells may appear to have cytoplasmic inclusions depending on the plane of slice; however, they are actually invaginations of the nuclear membrane rather than true inclusions [4]

***Correspondence to:** Hofman James, Department of Hematopathology, Erasmus MC-University Medical Center Rotterdam, Netherlands, E-mail: Hofman@jam.nl

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Understanding the molecular pathogenesis of ALCL has helped to define the disease and its link to other lymphomas, such as HD. At first, Stein et al. hypothesised that ALCL was connected to HD based on the expression of CD30. This hypothesis was strengthened by the existence of morphological and immune-phenotypic similarities between ALCL and HD. Although one study used RT-PCR to detect the NPM/ALK in HD, other studies were unable to corroborate this finding. Similar to this, some investigations came to the conclusion that the t (2; 5) NPM/ALK may not be specifically for ALCL due to the wide morphologic spectrum of ALCL. T-immunoblastic lymphomas and peripheral T-cell lymphomas have both shown promising RT-PCR outcomes [5].

Conclusion

A separate clinic-pathologic entity called ALCL has been identified based on the histologic, clinical, immunophenotypic, and molecular characteristics of the disease. Since most diseases have a unique molecular fingerprint that is related to the underlying pathophysiology, molecular analysis has been a significant technique in defining the boundaries of this and other entities. Furthermore, according to this paradigm, molecular research may be applied to improve the final morphologic definition and the diagnostic standards. Because ALCL is a highly curable form of lymphoma and has a significantly better prognosis than other types of T-cell lymphoma, a precise diagnosis has significant clinical ramifications. Our present definition of ALCL is both more specific and more general than what was first intended. Since there are considerable clinical differences between ALK+ and ALK, it is crucial to designate them as such if one chooses to include them in the wide definition of ALCL. An example of this is the molecular genetic sub classification of acute myeloid leukaemia, where unique genetic variants are regarded as sub entities having clinical and prognosis significance.

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