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Technologies for Early-Stage Cutaneous T-Cell Lymphoma Using Next-Generation Sequencing

Takuro Kanekura*

Department of Medicine, University of Cornell, Cornell, New York, USA

Description

Mycosis fungoides (MF) has a clinical presentation, histological findings, and laboratory findings that are similar to those of inflammatory skin diseases like atopic dermatitis, psoriasis, and parapsoriasis en plaque, making it difficult to diagnose early stage cutaneous T-cell lymphoma. Additionally, MF may occur simultaneously with or after these inflammatory skin conditions. Assessments of T cell clonality and clinical impressions are heavily incorporated into the current diagnostic criteria. The identification of a malignant clone is essential for making a diagnosis of early-stage MF. For this purpose, gene rearrangements of the T cell receptor (TCR) have been detected using southern blotting or polymerase chain reaction, but the results of these techniques are insufficient. TCR sequencing at high throughput has shed light on the intricate nature of the immune repertoire. As a result, his method is more sensitive and specific than current techniques, making it useful for early lesions and therapy response monitoring. Non-Hodgkin lymphomas, also known as cutaneous T cell lymphomas (CTCLs), are a diverse group of skin-tropic T cells-derived lymphomas. Mycosis fungoides (MF), the most pervasive sort of essential CTCL, represents close to half of all cases. Clinically, MF is characterized by erythematous patches, plaques, or skin tumors, and it may involve lymph nodes, the blood, or other internal organs. At first presentation, more than two-thirds of MF patients are in an early stage. Similar to many inflammatory skin diseases, MF typically manifests as an erythema that is not specific [1].

Histopathologically, MF can be identified by the formation of intraepidermal collections, also known as Pautrier's microabscesses, by the epidermotropic proliferation of pleomorphic lymphocytes ranging in size from small to medium. Although this microabscess is thought to be the histopathological sign of the disease, it only occurs in about 20% of early MF cases. Although spongiotic variants of MF have been reported, these microabscesses are typically identified as epidermotropic atypical lymphocyte infiltration without spongiosis. Skin lesions are infiltrated by a large number of non-malignant memory T cells, making it difficult to distinguish activated benign infiltrating T cells from malignant T cell clones based on histopathology, so morphologic characterization of early-stage MF may show non-specific findings. Clinical and histopathological calculations have been created to help early finding, yet the particularity and responsiveness of these calculations for early analysis in individual patients are in no way, shape or form laid out. Only careful clinicopathological correlations can lead to a definitive diagnosis [2].

Psoriasis, parapsoriasis en plaque (PEP), and chronic dermatitis such as atopic dermatitis (AD), among others, can resemble the early stages of MF. Promotion is a typical constant fiery skin problem that has a T-partner (Th) 2 sort prevailing aggregate, skin-boundary brokenness, and pruritus. AD, on the other hand, is an inflammatory disorder whose pathophysiology is comparable to AD's. Eosinophilia and elevated serum immunoglobulin E levels are frequently linked to mycosis fungoides, a Th2-type disease. Although T-cells in the affected skin and peripheral blood exhibit a Th1 cytokine profile during the early stages of MF, chemokines such as CCL17, CCL11, and CCL26 that are expressed in lesional MF skin are thought to induce a Th2 milieu in MF [3].

Based on next-generation high-throughput sequencing (NGS) technologies, assays for determining T cell clonality have recently seen improvements. The number of T cell clones in a sample, their relative proportions, and the CDR3 region sequences of each clone can be quantified by sequencing the third complementarity-determining regions (CDR3s) of TCR and TCR genes. Through the precise identification of malignant T cell clones, NGS is also a superior method for diagnosing CTCL. For detecting clonality, this method is more sensitive than previous methods. Additionally, when it comes to monitoring the recurrence and progression of a disease, NGS-based methods permit the clinician to follow specific clones. Additionally, TCR sequencing has demonstrated that the proportion of neoplastic cells in some MF lesions may be as low as one percent of the total number of T cells. The difficulties encountered during the histopathological assessment of early-stage MF are clearly explained by these data [4].

In MF, barrier dysfunction is also present. Lower levels of skin dampness, with expanded transepidermal water misfortune, have been seen in the lesional skin of CTCL, contrasted with that in typical skin. Similar to what has been demonstrated for AD, lesional CTCL skin has lower levels of filaggrin and loricrin mRNAs than normal skin. One of the most distressing symptoms for patients with MF is pruritus, which occurs frequently. As a result, it can be challenging to clinically distinguish MF from AD. Numerous studies also reported that patients with MF and AD coexisted De Masson demonstrated through the use of NGS technologies that in patients with CTCL and, more specifically, early-stage MF with a T2 distribution, a lower progression-free and overall survival rate is strongly correlated with an increased proportion of a malignant T cell clone in the skin. A tumor clone frequency of >25% was also found to be a strong predictor of disease progression and poor survival for MF patients with skin-limited disease, according to high-throughput DNA sequencing of the TCR gene [5].

NGS is useful for diagnosing early stage MF and can assess TCR clonality with greater sensitivity than current methods. In addition, this method enables the more precise diagnosis of MF recurrence or progression by allowing for the tracking of specific clones across various time points or in multiple lesions. In rundown, proof for TCR clonality from some technique is solid proof for danger. However, it is not conclusive because clonal T-cell populations have also been linked to benign conditions like reactive or autoimmune diseases [5].

***Address for Correspondence:** Takuro Kanekura, Department of Medicine, University of Cornell, Cornell, New York, USA, E-mail: kanekura@gmail.com*

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Conflict of Interest

None.

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