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Perspective

Acute Myeloid Leukemia: Development of Immune-Related Prognostic Features

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DESCRIPTION

A kind of hematological malignancy known as Acute Myeloid Leukemia (AML), which is the most common acute leukemia in adults, is characterized by the proliferation and accumulation of aberrant hematopoietic progenitor cells in the bone marrow. The normal hematopoietic system is disrupted by these multiplying nonfunctional leukemia cells, which then cause potentially fatal symptoms as immunodeficiency, anemia, and thrombocytopenia. Despite significant effort, there have been very few advancements in AML treatment over the past few decades, with a 5-year overall survival rate for AML patients of only about 50%. Sequential sessions of cytarabine and daunorubicin, the most widely used clinical chemotherapy for AML, demonstrated a high rate of resistance and relapse. Although there is no consensus regarding the cause of leukemia, the theory can be categorized into three groups: (1) immunological dysregulation; (2) immune gene mutation and translocation of AML cells; and (3) bone marrow microenvironment dysregulation. Numerous immunotherapy techniques have been included into the therapeutic management of AML and have shown to have promising results as a result of the exploding body of research showing that cancer immunology influences tumor pathogenesis. AML cells contain checkpoint inhibitor receptors and ligands, which can be potential targets in therapeutic therapy, in contrast to solid tumors. Sorting out high-risk AML patients and offering them extra therapy plans is urgently needed to improve the prognosis of AML patients. Hematological malignancies of the type AML have a dismal prognosis. The limited advancements in treatment regime contributed significantly to the unsatisfactory outcomes for AML patients, even if the efficacy of therapy relies on the patient's age and leukemia type. These non-targeted cytotoxic medications are beneficial for AML patients, although chemotherapy intolerance is also a significant issue. Additionally, AML cells found refuge in the tumor microenvironment, where immune functions were mismanaged, leading to cancer cells' capacity for self-renewal and the emergence of drug resistance. Therefore, early detection of high-risk patients and provision of extra immune targeted medicines may provide positive clinical outcomes. Therefore, in this work, we created two distinct immune-based prognostic signatures to classify high-risk patients and determine which checkpoint molecules are most crucial to their survival. This study classifies the TCGA cohort patients into high risk and low risk groups using this immune-genes associated signature, and they displayed significantly varied Overall Survival (OS) and Disease-Free Survival (DFS). Additionally, a strong and favorable correlation between patient's Immune Risk scores and their cytogenetic risk classification was discovered. The Immune Risk signature statistically associated with patient's survival under various cytogenetic risk scenarios. However, Receiver Operating Characteristic (ROC) curves showed that this signature had very low prediction efficiency when we verified it in the Gene Expression Omnibus (GEO) cohort. Furthermore, there is no difference in survival between patients who were divided into various risk groups based on the signature. We classified the TCGA cohort patients into high risk and low risk groups using this immune-genes associated signature, and they displayed significantly varied OS and DFS. Furthermore, there is no difference in survival between patients who were divided into various risk groups based on the signature. Although using these created fingerprints to predict the survival of AML patients is encouraging, there are several restrictions on applying them to clinical care. Larger prospective cohorts are required to validate the predictive value of signatures because those signatures were produced from two retrospective cohorts and the number of enrollees was also limited. Further mechanistic investigations are required to determine whether the identified checkpoint molecules could function as potential targets.

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