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Multiple myeloma with t (11;14)

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Abstract

Translocation t(11;14) is one of the most common primary translocations in multiple myeloma (MM). It has emerged as a crucial genetic aberration in MM. In addition, t(11;14) showed a strong association with additional molecular and chromosomal abnormalities. Understanding the role of t(11;14) can also guide the development of targeted therapies in MM and may thus improve the outcomes of this subgroup of patients with MM. In recent studies, the impact of t(11;14) on treatment responses was evident, which emphasize its role in shaping therapeutic strategies for this specific subgroup of patients.

Keywords: Myeloma; t11; 14; BCL2; Lymphoplasmacytic

Abbreviations: MM: Multiple Myeloma; SR: Standard Risk; HRCA: High-Risk Chromosomal Abnormalities; CAs: Cytogenetic Abnormalities

Introduction

Multiple myeloma (MM) is a hematological neoplasm characterized by an uncontrolled proliferation of malignant plasma cells and complex cytogenetic abnormalities. Presence of translocation t(11;14) in MM is considered a significant genetic abnormality that plays a key role in both risk stratification and in tailoring treatment decisions. t(11;14) is present in 15% to 20% of MM. t(11;14) engages IGH in chromosome 14 and has been associated with increased cyclin D1 (CCND1) expression leading to tumor cell proliferation. Moreover, one-third of these patients harbor additional high-risk abnormalities (e.g., del13q in 37%, del(IGH) in 33%, gain1q in 20%, and del16q in 15%). Furthermore, patients harboring t(11;14) have higher levels of the anti-apoptotic protein BCL2, and a reduced expression of the proapoptotic proteins MCL1 and BCLXL. t(11;14) is predictive of the BCL2 dependency [1].

MM cells carrying t(11;14) have a unique cellular features and distinct biological characteristics that differ from t(11;14)-negative MM risk categories

- A higher incidence in younger patients (≤50 years) [1].
- t(11;14) has been reported in patients with monoclonal gammopathy of an undetermined significance, indicating that it could be an early event in the pathogenesis of the disease.
- A higher rate of light-chain-only myeloma, higher probability of having oligosecretory and non-secretory disease, myeloma bone disease, and renal impairment due to cast nephropathy compared to patients without t(11;14).
- t(11;14) is a typical characteristic of patients with IgM myeloma.
- Patients diagnosed with plasma cell leukemia or light-chain AL amyloidosis have an incidence of 40% or 50% respectively of t(11;14).
- Have an increased number of circulating plasma cells [1], more bone marrow infiltration, less protein secretion, and fewer plasmacytomas at diagnosis [2].
- Have lymphoplasmacytic morphology.
- Have a lower probability of being hyperdiploid.
- It often exhibits clinical characteristics that position them between standard- and high-risk categories.
- Often lacks traditional plasma cell markers compared to other MM cell types. The greater abundance of expression of the B-cell lineage membrane protein CD20, increased levels of the B-cell receptor CD79a and decreased expression of CD38 and CD56.

- Increased levels of BCL2. t(11;14) arose as a predictive marker of vulnerability of MM patients to BCL2 inhibition. BCL2 inhibitors, such as venetoclax, demonstrated impressive efficacy alone or in combination with other anti-myeloma drugs in patients with RRMM accompanied by t(11;14) and BCL2 overexpression [1].

- t(11;14) patients are characterized, by lower incidences of IgA, and of plasmacytomas, higher PC bone marrow infiltration and levels of serum calcium in comparison to standard risk (SR) patients.

- t(11;14) patients had a less aggressive phenotype than those with the high-risk chromosomal abnormalities (HRCA), that is typified by fewer plasmacytomas, a lower β2 microglobulin level, and a higher hemoglobin level [2].

Dynamic Role of t(11;14) in Shaping MM Prognosis

The Mayo Stratification of Myeloma and Risk-Adapted Therapy consensus guidelines (mSMART) classified t(11;14) as a standard-risk cytogenetic abnormality. Outcomes in patients with t(11;14) vary, often irrespective of their standard stratification. Patients with t(11;14) exhibit outcomes that fall between the two extremes standard- and high-risk [1]. The survival of patients with t(11;14) was not worse than that of the SR group, but is better than that of the HRCA group [2]. Moreover, multiple studies on patients with t(11;14) and MM consistently demonstrated inferior outcomes compared to hyperdiploid standard-risk patients [1]. In the era of new drugs, patients with t(11;14) have poorer PFS compared to those with normal cytogenetics [3].

Treatment of t11,14 MM

Conventional Chemotherapy

The Spanish GEM2005-MENOS65 and GEM2012 trials of transplant-eligible MM patients revealed similar responses and outcomes for patients with t(11;14) and SR cytogenetic when they received conventional chemotherapy such as chemotherapy (melphalan or cyclophosphamide with dexamethasone/prednisone) and polychemotherapy (e.g., the combination of vincristine, carmustine, melphalan, cyclophosphamide, and prednisone; VBCMP) [2].

IMIDs/PIs

The introduction of novel agents in the first line treatment did not benefit the t(11;14) group as much as the other cytogenetic subgroups in terms of response, PFS, or OS, so this translocation may be considered a marker of suboptimal response to IMIDs/PIs. Patients who received a PI plus IMID combination had better outcomes than those treated with PI or IMID alone, as shown by the IMWG study, and upfront ASCT resulted in survival close to 10 years.

The expectations of less effectiveness of PIs and IMIDs in t(11;14), arises from the endoplasmic reticulum stress theory. It explains how compensatory pathways are activated when PCs accumulate unfolded or misfolded proteins to eliminate them. However, clonal PCs that harbor t(11;14) have a lymphoplasmacytic morphology, with scant cytoplasm and less rough endoplasmic reticulum. For this reason, these PCs might be less likely to accumulate proteins and less able to activate the compensatory mechanism derived from endoplasmic reticulum stress, and thereby less susceptible to drugs that inhibit it directly (e.g., PIs) or indirectly (e.g., IMIDs) [2].

Venetoclax

The overexpression of BCL2 and the high ratio of BCL2/MCL1, indicate a vulnerability to BCL2 inhibitors. Novel therapies targeting the BCL2 family, such as venetoclax, offer hope for increased responses of t11,14 MM to therapy and improved survival. Venetoclax has shown ORR of about 40% in patients with RRMM harboring t(11;14) and demonstrated synergistic effects if combined with PIs and dexamethasone, which were most improved in patients with MM and t(11;14) [1].

ASCT

Upfront ASCT in transplant-eligible patients likely remains important in t(11;14) MM showing higher ORR and longer PFS-1 in patients who underwent ASCT vs. those that did not [4].

Recommendations

- A global consensus for routine performance of FISH testing of t(11;14) at the time of diagnosis and during relapse of the disease is warranted since t(11;14) demonstrates a rather special biology in comparison to a specific risk group [1].

- Future clinical trials should additionally focus on the incorporation of BCL2 inhibitors into earlier treatment lines, and potent combinations of BCL2 inhibitors in conjunction with other anti-myeloma agents. Novel intervention strategies hold the potential to enhance the outcome in both patients with NDMM and patients with RRMM [1].

- Translocation t(11;14) frequently occurs alongside other abnormalities, with approximately one-third of affected patients exhibiting additional cytogenetic abnormalities (CAs). Recent studies highlight the need to consider the presence of coexisting CAs such as HRCAs and 1q21+, when assessing the prognostic implications of t(11;14). The presence of additional CAs, such as HRCAs and 1q21+, correlates with an even worse prognosis. Notably, 1q21+ is considered a prognostic risk factor for PFS in t(11;14) MM. Consequently, alternative therapeutic options are essential for clinical treatment of patients with NDMM with this genetic abnormality. However, few studies have explored the influence of t(11;14) on CAs [3]. t(11;14) coexistence does not provide a survival benefit for high-risk genetic patients. Patients with t(11;14) plus other high risk cytogenetic abnormalities require early and more aggressive treatment options, such as BCL-2 inhibitors [5].

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Conclusion

A better understanding of t(11;14) genetic abnormality influences the choice of therapeutic approaches. Therefore, the presence of this translocation highlights the need for precise clinical decision making. Physicians should consider t(11;14), besides other important genetic markers, when devising treatment strategies for patients with MM.

References

1. Kleber M, Ntanasis-Stathopoulos I, Terpos E (2023) The Role of t(11;14) in tailoring treatment decisions in multiple myeloma. Cancers 15(24): 5829.
2. Puertas B, González-Calle V, Sobejano-Fuertes E, Escalante F, Rey-Bua B, et al. (2023) Multiple myeloma with t(11;14): impact of novel agents on outcome. Blood Cancer Journal 13: 40.
3. Li Y, Deng J, Jian Y, Zhang Z, Chen W (2025) Is t(11;14) in newly diagnosed multiple myeloma a favorable outcome in the novel agent era? Blood Res 60(1): 11.
4. Lim KJ, Wellard C, Talaulikar D, Tan JL, Loh J, et al. (2023) The prognostic impact of t(11;14) in multiple myeloma: a real-world analysis from the Australian Lymphoma Leukaemia Group (ALLG) and the Australian Myeloma and Related Diseases Registry (MRDR). EJHaem 4(3): 639-646.
5. Liu Y, Qiu L-G, An G (2024) t(11;14) with multiple myeloma: standard risk survival but slow and poor response. Blood 144: 4709.

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