Thursday, June 05
19:00-21:00 (room A)

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A guiding light: illuminating current treatment concepts in CLL and T-NHL

Co-chairs: M.J. Hallek (Cologne, DE) and M.J. Keating (Houston, USA)

ANTI-CD52 antibody treatment: a guiding light to improved patients outcomes

M.J. Keating (Houston, USA)

Enlightening treatments in CLL: prolonging survival in high-risk patients

S. Stilgenbauer (Ulm, DE)

Consolidation with antibody therapy: closer to full disease eradication

M.J. Hallek (Cologne, DE)

Advances in the treatment of tnhl: the role of anti-cd52 antibody therapy

F. d'Amore (Aarhus, DK)

ILLUMINATING CURRENT TREATMENT CONCEPTS IN CLL AND T-NHL

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Chronic lymphocytic leukemia (CLL) is the most common leukemia in the Western world. Finding a cure for CLL remains a central goal in clinical research, and novel strategies that exploit the efficacy of targeted treatment with monoclonal antibodies are therefore of great interest. This symposiumwill focus on current and emerging approaches to treatment with CD52-specific antibodies in CLL and T-cell non-Hodgkin's lymphoma (T-NHL), offering a forum for discussion with key hemato-oncology specialists.

Professor Michael Keating will explore the role of CD52 antibody therapy within the framework of current clinical guidelines for CLL management.[1] Interpreting findings from recent and ongoing clinical trials of first-line, consolidation and salvage treatment with immunotherapy, he will discuss how CD52-specific antibodies can be employed in clinical practice to improve the quality of response and duration of survival in patients with CLL. The pivotal phase III CAM307 study of first-line treatment with alemtuzumab[2,3] and the ongoing phase II CFAR study of fludarabine-based immunochemotherapy in previously untreated patients with high-risk disease[4] will feature centrally in his presentation.

A subanalysis from the CAM307 study showed significant improvements in response to first-line CD52 antibody therapy versus chlorambucil in a group of patients with either del(11q) or del(17p) mutations ($p<0.0001$).[3] These aberrations and the mutational status of IgVH are strong predictors of rapid disease progression, treatment resistance and survival duration in patients with CLL.[5] Professor Stephan Stilgenbauer will discuss data from recent clinical trials to highlight the encouraging survival outcomes that have been observed in patients with a poor-risk genetic profile, who receive treatment with CD52 antibody-based regimens.[6,7]

The eradication of minimal residual disease (MRD) is recognized as a crucial predictor of survival and an important clinical goal for all patients with CLL.[8] Treatment with single-agent CD52-specific antibody therapy was shown to induce MRD-negative remissions in 20% of patients with relapsed/refractory CLL and was associated with improved overall and treatment-free survival.[9] Results from several studies also suggest that MRD-negativity can be attained when this antibody is administered as consolidation for patients with CLL, who achieve incomplete initial responses to chemotherapy.[10-12] Professor Michael Hallek will present an overview of data from clinical trials investigating the utility of CD52 antibody consolidation following fludarabine-based induction therapy.

T-NHL cells also express high levels of CD52,[13]making targeted therapy with an CD52-specific antibody a logical treatment for this rare but often difficult to treat malignancy.Drawing on recent clinical data, as well as his own experience as a member of the Nordic Lymphoma Group, Dr Francesco d'Amore will look at different investigational regimens with CD52 antibody therapy in T-NHL. The concepts underlying the initiation of two different phase III studies that have recently started recruitment – combined CD52 antibody therapy plus CHOP versus CHOP alone in previously untreated younger (ACT-1)[14] or elderly patients (ACT-2)[15] – will also be discussed.

References

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