Interleukin-2 and Lymphokine-Activated Killer Cell Therapy in Patients with Relapsed B-Cell Lymphoma Treated with Rituximab

To the Editor: In their article, Berdeja et al. (1) conclude that the antibody-dependent cellular cytotoxicity activity can be augmented by adoptively transferring in vitro generated lymphokine-activated killer cells, which may overcome the resistance to anti-CD20 monoclonal antibody therapy in patients with non-Hodgkin’s lymphoma who are rituximab refractory. We feel that the conclusion is overstated and that it may mislead clinicians into using this regimen as a standard first-line therapy.

Briefly, several early clinical investigations about the combination of interleukin-2 with rituximab have been reported (2, 3); however, although the addition of interleukin-2 augmented natural killer cell expansion and antibody-dependent cellular cytotoxicity, all these studies failed to confirm that interleukin-2 enhances the clinical efficacy of rituximab in low-grade non-Hodgkin’s lymphoma patients because the encouraging response rate observed was comparable to results with single-agent rituximab (4, 5). Furthermore, although antibody-dependent cellular cytotoxicity is an important effector mechanism for in vivo action of rituximab, other multiple complex mechanisms are involved in response and resistance, such as the cellular microenvironment and monocyte Fc receptor polymorphism, which are not affected by interleukin-2 treatment (6).

Finally, it should be noted that in patients who achieved a partial remission or a stable disease, it is not possible to determine the relative contributions of interleukin-2 and lymphokine-activated killer cells because these results might simply reflect a response at a much higher given dose of rituximab (375 mg/m² on 3 consecutive days), which may overcome adverse pharmacokinetic parameters.

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References
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