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The role of natural killer cells in liver injury

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Human hepatic lymphocytes are enriched in natural killer (NK) cells, which are defined as CD56+ CD3-and can be further subdivided into two distinct subpopulations $CD56^{bright}$ and $CD56^{dim}$ based on the relative expression of the surface marker CD56. While $CD56^{bright}$ are poorly cytotoxic but produce a broad range of cytokines (IFN-γ, TNF-α, GM-CSF, IL10), $CD56^{dim}$ NK cells are highly cytotoxic. Thus, NK cells have dual function: effector, against pathogens and tumors through their natural cytotoxicity and cytokine production; and regulatory, which is dictated by cross talk between NK cells and other types of immune cells in liver resulting in the production of variety of cytokines. As such, interaction between NK cells and activated Kupffer cells (KCs), dendritic cells (DCs) and natural killer T cell (NKT) cells results in the production of IFN-α/β, IFN-γ, IL-2, IL-12, IL-15 and IL-18. This cytokine-mediated NK cell activation contributes to hepatocellular damage during viral hepatitis. Conversely, several cytokines have been shown to inhibit the functions of hepatic NK cells. Accumulation of NK cells within the liver results in higher levels of cytotoxicity and cytokine production, but it can also enhance hepatocellular damage. In contrast, chronic liver diseases are associated with a decreased number of NK cells and impairments in NK cell cytotoxicity and cytokine production. Hence, understanding the multifaceted role of NK cells in pathogenesis of liver diseases, reviewed in here, may help us design better therapies to treat patients and translate these findings into clinical practice.

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Drug-induced modulation of T lymphocytes as a potential mechanism of susceptibility to infections in patients with multiple myeloma during bortezomib therapy

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Introduction & Aim: Bortezomib is effective in the therapy of multiple myeloma (MM), but causes infections that are different from those associated with conventional chemotherapy. It is important to identify the risk factors that facilitate infections associated with bortezomib therapy. In the present report, we sought to (1) define the features of the infections associated with this therapy and (2) identify the immune mechanisms responsible for the observed susceptibility to these infections.

Methods: We first retrospectively analyzed the clinical data of 143 patients who had received bortezomib therapy for MM. We then prospectively assessed the modulation of T lymphocyte status during this therapy, and evaluated potential relationships between infections and T lymphocyte changes.

Results: The infection rates peaked during the first cycle of bortezomib therapy (47.6%) in patients with MM ($p<0.05$ vs. subsequent cycles). Bortezomib therapy was associated with higher incidence rates of viral and fungal infections (15.8%, $p<0.05$ vs. conventional chemotherapy). In addition, patients with the IgG immunophenotype showed higher bacterial and viral infection rates (respectively, $p=0.008$ and 0.009). The T lymphocyte numbers significantly decreased after bortezomib therapy ($p<0.05$), and the same was true for the Th1/Th2 ratio ($p<0.01$).

Conclusions: Patients with MM who have decreased lymphocyte counts while on bortezomib therapy are more likely to develop bacterial or viral infections. In addition, an imbalance in T lymphocyte subsets is also associated with bacterial or viral infections in these patients.

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