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Dual face functional role of IL-33 during immune cell mediated and hepato-toxic induced acute liver injury

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IL-33/ST2 axis play a protective role during acute hepatitis but little is known about the functional role of endogenous IL-33 in liver patho-physiology. We aimed to decipher the functional role of IL-33 by using IL-33 deficient mice during immune cell mediated and hepatotoxic driven liver injury. We used a genetic model of acute hepatitis by using IL-33 deficient mice in ConA (a T cell-mediated hepatitis) and CCl4 (a hepatotoxic agent-induced hepatitis) induced acute liver injury. The liver functions (AST/ALT), signature of cytokines and characterization of infiltrate cell in WT and IL-33-/-mice were carried out by biochemistry, qPCR and flow cytometry analyses. Our results demonstrated that IL-33-/- mice exhibited more severe ConA liver injury than WT mice evidencing a protective effect of IL-33 in this hepatic model while no difference was observed in CCl4-hepatitis between WT and IL-33-/- mice. The ConA-induced hepatic injury was associated with increased TNF-α, IL-1-β, IFN-γ and IL-6 cytokines in WT and IL-33-/- mice. The level of TNF-α and IL-1-β but not of IFN-γ and IL-6 was significantly higher in IL-33-/- mice than WT control. The intra-hepatic percentage of NK, NKT cells, T cells and B cells was not altered significantly between WT and IL-33-/- mice following ConA-hepatitis. In conclusion, we evidenced that the genetic ablation of IL-33 sensitized the mice to severe ConA liver injury but not CCl4-mediated liver injury. IL-33 has a limited impact on pro-inflammatory cytokines and influx of infiltrate cells during immune cell mediated liver pathology.

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HBsAg Quantification: Useful for monitoring natural history and treatment outcome

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Serum hepatitis B surface antigen (HBsAg) level reflects the transcriptional activity of the covalently closed circular DNA in the liver. In clinical practice, quantification of HBsAg (qHBsAg) is a simple and reproducible tool that may be used in association with HBV-DNA to classify patients during the natural history of HBV and to monitor therapy. There is a growing interest in serum HBsAg quantification (qHbsAg). In hepatitis B e antigen (HBeAg) positive chronic hepatitis B, HBsAg level is higher in the immune tolerance phase than the immune clearance phase, HBsAg titers are negatively correlated with liver fibrosis. In HBeAg negative patients HBsAg level <1000 IU/ml and HBV-DNA titer <2000 IU/ml accurately identify inactive carriers. In HBeAg-negative patients, combination of low hepatitis B virus (HBV) DNA and low HBsAg levels may predict low risk of HCC, and probability of HBsAg loss. During PEG-IFN treatment qHBsAg identifies patients with no benefit from therapy at week 12, allowing stopping or switched therapy so call the "week 12 stopping rule": Absence of any decline at week 12: Prediction of non response therapy should be ended or switched to nucleos(t)ide analogues, Any decline at week 24 prediction of response continue the 48 weeks therapy. During nucleos(t)ide analogues therapy the role of qHBsAg need to be clarified. Although the HBsAg decline is slow with nucleos(t)ide analogue therapy, a rapid decline may predict future HbsAg seroclearance. More recently an HBsAg titer <1000 IU/ml during at least 3 measurements during nucleos(t)ide analogue therapy might allow to end therapy with los risk of relapse.

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