EuroSciCon

Annual Summit on

Hepatology and Pancreatic Diseases

November 12-13, 2018
Paris, France

J Clin Gastroenterol Hepatol 2018, Volume:2
DOI: 10.21767/2575-7733-C3-009

NOVEL THERAPIES FOR HBV

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Alternative treatments for CHB are subject of in¬tense research worldwide, one of the most studied has been therapeutic vaccination. Important clinical trials combining therapeutic vaccination and antiviral treatments have failed in their attempt to reach the study endpoints. The rationale favoring vaccination under viral suppression is based in the observation that a decrease in HBV load seems to precede the detection of HBV specific T-cell responses. This has been evidenced, both in patients resolving natural infections and in those displaying flare-ups of hepatitis associated with HBeAg seroconversion during chronic infection. Also, the reduction in HBV load by antiviral chemo¬therapy may, therefore, increase the responsiveness of HBV-specific T cells which are hypo-responsive in cases of persistent HBV or viral antigen stimulation.

ON the other hand, taking into account the immunology of the liver, there are some theoretical disadvantages from immu¬nizing patients under long-term antiviral treatment. The induced immune response needs to migrate to the liver, in order to exert their function. However, the liver is under non-inflammatory con¬ditions, as evidenced by the sustained reduction in ALT levels in most patients under antiviral treatment. Important publica¬tions support that hepatocytes only express HLA class II in pro-inflammatory conditions. Inflamma¬tory mediators or the HBV infection itself have been proposed as eliciting agents. The elimination of the virus and the normalization of ALT during long term antiviral therapy further reduce the inflammatory mediators and, consequently, the expression of HLA class II and the CD4+ T helper activity. On the other hand, the reduction of the replication has been linked to a lower intracellular expression of viral antigens, mainly cytoplasmic HBcAg. Taken together, it is expected a reduced intracellular expression of viral antigens in the virally suppressed patients, together with the absence of HLA class II ex¬pression and a reduced presentation of viral peptides to vaccine-induced T cells by both HLA class I and II. A second opportunity appears for therapeutic vac¬cination after antiviral treatment cessation: the natu¬ral reactivation of the immune response represents a solid and effective factor that may further potentiate the vaccine-induced immune response.

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