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Hepatology Highlights

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Sookoian et al...Ribavirin (1-ß-D-ribofuranosyl-1,2,4-triazole-3-carboxamide) is a nucleoside analogue that has demonstrated efficacy in the treatment of viral disease with or without directly inhibiting viral replication. Thus, its demonstrable antiviral activity may be attributed to a direct reduction in levels of circulating virus and/or the promotion of T cell-mediated immunity against viral infection. In this article the authors evaluated the effects of ribavirin at different concentrations (1, 10 and 100 μM) on cytokine production of recall antigen and PHA-stimulated PBMC obtained from healthy individuals. Parallel sets of wells containing PBMC exposed to medium alone were used as negative controls. They found that the effects of ribavirin on cytokine released by human PBMC in response to PHA and TT showed a great variation among individuals. No significant changes were observed between 1-10 μM concentrations in the production of TNF-α, IFN-γ and IL-10 by both PHA and TT-stimulated PBMC. However, ribavirin inhibited TNF-α, IFN-γ, and IL-10 in both PHA and TT-stimulated PBMC at 100 μM ($p<0.05$). At this concentration, ribavirin induced an increase of 124% in the production of IL-2 by PHA-stimulated PBMC ($p < 0.05$). They concluded that ribavirin may cause diverse effects on immunoregulatory cytokine secretion with changes in the Th1/Th2 balance. This study added to previous information on the effect of ribavirin on the immune system.

Méndez-Sánchez et al. Nonalcoholic fatty liver disease is an increasingly recognized condition that may progress to end-stage liver disease. In this study the authors investigated in a short period the effects of weight reduction and ursodeoxycholic acid administration in patients hepatic steatosis assessed by ultrasound. They carried out a double-blind, placebo-controlled trial and treat twenty-seven women with a body mass index of >30 kg/m^2 with a normal diet (1,200 kcal/d) plus 1,200 mg/d of ursodeoxycholic acid or placebo. Hepatic steatosis, was assessed by abdominal ultrasound. Fasting glucose, cholesterol, triglycerides, and aminotransferases levels were determined before and after treatment. They found that body mass index decreases significantly from 34.2 ± 4.2 kg/m^2 and 33.3 ± 1.6 kg/m^2 to 31.8 ± 4.5 kg/m^2 and 30.6 ± 2.6 kg/m^2 in the ursodeoxycholic acid and placebo groups, $p < 0.001$. The hepatic steatosis index decreased from 2.3 ± 0.7 to 1.0 ± 0.6 and 2.2 ± 0.7 to 1.1 ± 0.7 in the ursodeoxycholic acid and placebo groups, $p <0.003$. Serum AST decreased significantly from 41.2 ± 5.6 to 34.5 ± 3.4 in the ursodeoxycholic acid group, $p <0.001$, and from 43.6 ± 4.2 to 35.3 ± 2.9 in the placebo group, $p <0.001$. Serum ALT decreased from 62.9 ± 6.5 to 44.0 ± 3.5 in the ursodeoxycholic acid group, $p <0.001$, and from 63.5 ± 4.5 to 44.0 ± 3.5 in the placebo group. They concluded that weight reduction producing improvements in biochemical and imaging markers of liver disease with or without ursodeoxycholic acid. In this study the authors hese data indicate the importance of weight reduction, especially considering that the cat model has similarities with NAFLD in humans.

It has been suggested that weight loss, particularly if gradual, may lead to improvement in liver histology. However, rate and degree of weight loss required for normalization of liver histology have not been established. This is the first controlled clinical trial that demonstrated the beneficial effect of weight reduction. However, it will be useful in the future to confirm these results in a longer period of reduction. Furthermore, liver tests, in particular serum aminotransferase levels, usually improve or normalize with weight loss.

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