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Hepatology Highlights

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Genistein Decreases Liver Fibrosis and Cholestasis Induced by Prolonged Biliary Obstruction in the Rat by Alfonso Leija Salas

Genistein is a soy-derived isoflavone shown to have several, unrelated pharmaceutical effects. Genistein is a potent tyrosine kinase inhibitor which results in the inhibition of either growth factor- and cytokine-stimulated proliferation of cells. Genistein has also been shown to affect the *in vitro* metabolism of stellate cells where inhibits the TGF-β1-stimulated collagen synthesis, increases cell proliferation and inhibits the intensity of *c-fos*, *c-jun*, and cyclin D1 expression collectively indicating an antifibrotic activity. This work reports the effect of genistein in the biliary obstructed rat, a well established *in vivo* model of hepatic fibrosis. Genistein administration after the establishment of a hepatic fibrosis was followed by a reduction of the hepatic content of collagen, probably related to an increased degradation of type 1. This was associated with a 5 times reduction in the serum bilirubin level as compared to untreated animals and a 3 times reduction of alkaline phosphatase activity. Although no information was provided on the mechanism(s) of the *in vivo* antifibrotic effect(s), this is an important piece of information which may open new avenues in the treatment of human liver fibrosis/cirrhosis. Having the possibility to shift the balance linking deposition and removing of scar tissue towards removal may be crucial. However, we must be careful before extrapolating what demonstrated in the predictable rat model to the much more complex one of human cirrhosis. In spite of these caveats, this study may pave the way to a controlled clinical trial where the antifibrotic effect may be assessed, particularly since new non invasive tests of fibrosis (Fibroscan as a good example) are becoming available.

Fatty liver: Experience from Western India, by Anjali Amarapurkar and Tanveer Ghansar

Liver steatosis is booming in the Western world and steatosis is the leading cause of slightly abnormal liver function tests (ALT and GGT in particular). The main etiology is obesity and steatosis is believed to be part of the so called «metabolic syndrome». This study performed in Mumbai indicates that among autopsies performed over 5 years the prevalence of fatty liver was around 16% and that alcoholism was the major risk factors associated with the fatty liver. Interestingly fatty liver linked to alcohol consumption appeared to be associated with a rather important inflammation and fibrosis, suggestive for ASH, while fatty liver related to chronic infection (TB) showed a lower degree of inflammation. In spite of the poor definition and selection of the sample and the retrospective design, the main take home message is that steatosis observed in India is clearly different from what observed in Europe and US as well indicated by the finding that obesity was present in 10 out of 195 steatosis, i.e. 5%. This figure is at least 10 times lower that what reported in Europe and US and speaks most probably for a different disorder. More worrisome is the consideration that autopsy may not be the appropriate cohort for a metabolic disorder such as steatosis since the events preceding death may affect the percentage of liver cells filled with fat. This should prompt to prospective studies in living Indian subjects where the real prevalence and risk factors of fatty liver (both NAFLD and AFLD) may be compared with those reported in Western countries. I expect that the picture will not be as different as reported here.

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