



Hepatology Highlights

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What is the reason of elevated alanine aminotransferase level in HBeAg negative patients with low viremia: NAFLD or chronic hepatitis? By K Demir *et al.*

The discovery of an elevation (usually mild) of serum ALT activity is becoming more and more the main reason for referral of a patient to a Liver or GE center in the Western countries.

Usually the work up reveals the presence of one or more stigmata of the metabolic syndrome and a bright liver at the ultrasonography examination. The diagnosis of fatty liver (FL) is then rather simply reached, the distinction between AFLD and NAFLD depending on the daily amount of alcohol the subject reports. The diagnosis of FL may be complicated when other reasons for an increased level of ALT exist. In this paper Demir and colleagues analyze the basis for an increased level of ALST in 49 subjects with past history of HBV infection. Histological diagnosis of chronic hepatitis was found in about ¼ of the cases while in the remaining ¾ steatosis was present. This was taken as indication that fatty liver is by far the most frequent reason for a moderate increment of ALT and as suggestion that biopsy should be performed only when a clear cause of liver damage is present, FL excluded. This conclusion somehow fits with several guidelines recommended by different liver societies by must be taken with caution. While is true that NALFD is a benign disorder and much attention should be focused on the metabolic alteration at the basis of the fatty infiltration of the hepatocytes (diabetes and insulin resistance in particular), this is not the case when necro-inflammation is associated with FL. It is becoming increasingly evident that the prognosis of the NASH and ASH is different from NAFLD, AFLD or chronic hepatitis due to viral infection. Surprisingly in this series

no clear difference was present between subjects with NAFLD and chronic hepatitis as far as the indicators of metabolic syndrome are concerned. This suggests a consistent overlapping in the population studied and supports the importance of a diagnosis based on liver biopsy in these subjects.

***In vitro* study on the mechanisms of action of a novel phytotherapeutic compound against human hepatoma cells.** By F. Marotta *et al.*

YHK (panax pseudo-ginseng, Eucommia Ulmoides, polygonati rhizome, glycyrrhiza licorice, panax ginseng) is a multi-derivative compound which as been suggested to be possibly active as anticancer agent *in vitro*. This study further assesses the effects of increasing doses of YHK in a cell line originally derived from a human liver tumor such as HepG2. YKH added to the cells increases the rate of apoptosis together with an increased release of LDH in the medium and reduced detoxifying activity assessed by the classic MTT method. Of notice was the observation that cells exposed to the plant mixture arrested or reduced the growth. Although these results point mainly to a dose-dependent hepatotoxic toxic effect of YHK, they are interpreted as evidence for a role of the compound in the treatment of liver tumors. This conclusion is clearly speculative and by far too stressed for several good reasons. The first is that HepG2 cells are not a valid model for hepatocellular carcinoma and *in vivo* models may be much better suited to prove (or disproved) the assumption that YHK is useful in liver cancer. The second is the lack of any evidence on the mechanism(s) of the activity of the compound, a background we need to unravel before this herbal mixture may be used in mammals.



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