Effect of K-17.22, a Novel Phytoterapeutic Agent, on DNA Synthesis and Liver Enzyme Release in Experimental Liver Damage

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ABSTRACT ONLY

It has been suggested that a number of toxins, drug chemicals and virus infection to the liver can bring about a DNA synthesis abnormalities. Such DNA impairment are also of paramount importance in the reparative mechanisms during the recovery period after the injury. The purpose of the present investigation was to see whether K-17.22, a controlled herbal remedy which is used in clinical practice in HCV liver disease and had been successfully tested by us in previous experimental studies, could exert any beneficial effect on plasma and liver tissue parameters of hepatotoxicity as well as on liver DNA synthesis activity. Seventy-two Wistar rats were randomly put on 2-week dietary supplementation as follows: A) standard diet or B) standard diet enriched with K-17.22 (50mg/kg/day).

Afterwards, alpha-naphtyl-isothiocyanate (ANIT) liver injury model was applied. A separate group of healthy rats served as control. At one-week observation, blood samples were withdrawn to measure routine chemistry, lipid peroxide (LPO) and malondialdehyde (MDA). At sacrifice the liver was quickly excised and tissue samples were used to measure GSH, GSSG, GSH-Px and DNA synthesis rate by radioactivity counting. As compared to control, group A rats showed a significantly increased levels of GOT, GPT, ALP, T. Bilirubin, LPO and MDA ($p<0.001$). All these parameters significantly decreased, although remaining abnormal, in group B rats ($p<0.05$ vs A). Accordingly, the in vitro release of GOT, GPT and ALP from group A rats significantly decreased when aqueous solution of K-17.22 was added ($p<005$). ANIT caused a nearly 50% decrease of DNA synthesis in liver slices ($p<0.01$).

However, such effect was virtually preserved by K-17.22 ($p<0.01$ vs A). Moreover, liver concentrations of GSH significantly decreased while GSSG decreased in A group ($p<0.01$ vs control). However, such effects were prevented by the concomitant K-17.22 adminstration ($p<0.05$ vs A). These preliminary data suggest that K-17.22 does indeed exert a significant protective effect in liver toxicity model. Given that ANIT noxious mechanism is a neutrophil-generated lipid peroxidation, these data offer a potential tool in an integrative treatment of HCV chronic liver disease expecially in view of HCC transformation.