A Nutritional Approach with Herbal Remedy K-17.22 Delays the Onset of Spontaneous Chronic Pancreatitis

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ABSTRACT

Background Chronic pancreatitis (CP) is a progressive disease and to date no validated therapeutic options are available. Recently, it has been devised an herbal formula (Yojyo-Henshiko: K-17.22, Kyotsu Inc., Tokyo, Japan), obtained from selective control of the processing preparation and which has been shown in preliminary clinical tests to significantly decrease chronic viral liver disease activity.

AIM

The aim of this study was to test the above preparation on the progression of a genetic model of CP.

METHODS

Four-week old WBN/Kob rats which were fed a specific MB-3 diet in order to promote CP within a further 12-week period. Rats were allocated into 3 groups: A) no treatment; B) K-17.22 50mg/kg-5% glucose; C) vitamin E 200mg/kg. The same schedules were applied "therapeutically" after the onset of CP (from 12th to 20th week). Rats were sacrificed at 12- and 20-week in the "prophylactic" and "therapeutic" group, respectively. Routine histology, blindly scored, was carried out. Total RNA was extracted from pancreatic tissue for PAP gene expression by RT-PCR method.

RESULTS

Unlike A and C groups, B rats pancreata didn't show any overt oedema/haemorrhage on macroscopic examination in the "prophylactic" group, but not on the "therapeutic" one. Either B and C rats preserved pancreas weight and lessened serum amylase ($P<0.05$ vs. A). Microscopic analysis showed that K-17.22 almost entirely prevented CP damage as compared to A and C rats ($P<0.001$) when used prophylactically while it significantly decreased fibrosis, inflammatory infiltrate, oedema and ductal hyperplasia in the "therapeutic" group ($P<0.05$). As compared to A and C group, B rats showed a complete suppression of PAP mRNA in the prophylactic group ($P<0.01$) and a significant decrease in the therapeutic schedule ($P<0.05$).

CONCLUSIONS

The present preliminary data suggest that K-17.22 exerts powerful protective and therapeutic effect against the progression of experimental CP by mechanisms to be elucidated as yet. These might possibly involve effects such as antioxidative, microcirculatory-enhancement, gastric and pancreatic secretion suppression and cytokine regulation. PDF format