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Injurious Effects of Ethyl Alcohol on Liver Function and Protective Effect of Liv.52

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SUMAMRY

Injurious effects of chronic alcohol intake on liver function have been well known and experimentally alcohol has been shown to produce liver damage in rats. Liv.52 pretreatment has been shown to protect rat liver from alcohol induced in injury. Using Bromsulfalein excretion test, Sain et al.*, demonstrated impairment of liver function after acute ingestion of alcohol. Using whole body linear scanner and body segment counter it was possible to determine the Rose Bengal I^{131} uptake by the liver accurately in human volunteers. By this test it was possible to demonstrate impairment of liver function with ingestion of moderate quantity of alcohol in a single session. After pretreatment with Liv.52 or placebo in parallel groups in a randomised fashion it was observed that Liv.52, but not placebo, prevented the depression of liver function with alcohol.*

The stimulation of albumin synthesis by the liver and increase in appetite caused by Liv.52 suggest the removal of acetaldehyde as one of the mechanisms of beneficial action of Liv.52 on the liver.

INTRODUCTION

Injurious effects of excessive and prolonged alcohol intake on the liver have been known for a long time[1]. The course of alcoholic liver disease is prolonged and variable and hence evaluation of beneficial effects of therapy has been difficult. Using I^{131} labelled Rose Bengal and whole body linear scanner body segment counter Harshe *et al.*[2] demonstrated that, even a single episode of social drinking causes reversible depression of liver function (Sain *et al.*[3]). Joglekar and Balwani[4] showed that pretreatment of rats with Liv.52 protects the liver from the alcohol induced injury. Chronic alcoholics show higher levels of acetaldehyde, which may be responsible for anorexia and cellular injury to the liver. The present study was planned to see if Liv.52 would reduce the injurious effects of single ingestion of alcohol on the liver function and if it has any effect on the acetaldehyde levels in persons consuming alcohol regularly.

MATERIAL AND METHODS

Part I

Twenty middle aged male subjects who consented to participate in the study after being informed of the details and purpose of study were included in the study. The biochemical and haematological tests and complete physical examination did not reveal any abnormality. On the day of the study volunteers reported to the study laboratory in the division of Clinical Pharmacology. Two volunteers reported at a time. One received 100 ml whisky in soda water in two divided doses at an interval of 30 minutes. The other received lime juice similarly. Two hours after consuming the drinks, the Rose Bengal uptake by the liver was determined. From the 2nd day, onwards the volunteers received either placebo or Liv.52 tablets in the dose of 3 tablets twice a day in a double blind random fashion for 14 days. On the 15th day the volunteers received whisky in the same way as on day 1 and the Rose Bengal uptake of the liver was similarly estimated.

Determination of I^{131} Rose Bengal Uptake of the Liver:

The subject received 25 μci of I^{131} Rose Bengal intravenously and at 25 minutes the volunteer passed under a shadow shield detector on a moving bed from head to foot. The counter printed the

counts in every one inch of the length of the body and the cumulative total body count. The count over the liver segment divided by the whole body count, multiplied by 100, was expressed as per cent liver uptake of Rose Bengal.

Part II

Six middle aged male subjects who had normal biochemical and haematological tests and who gave informed written consent were included in the study. They were regularly consuming moderate amounts of alcoholic beverages for at least 3 years. On the day of the study they reported at 5 p.m. to the study laboratory in the Clinical Pharmacology Unit. Venous blood sample was collected as a control and then they consumed 100 ml of whisky with soda in two portions 30 minutes apart. Blood samples were again collected immediately after cessation of drinking and one and two and a half hours thereafter. Acetaldehyde and alcohol in blood were determined by gas chromatography. From day 2 each volunteer received either Liv.52 tablets or a placebo in double blind random fashion for 14 days in the dose of 3 tablets twice a day. On the 15th day the study, as on day 1, was repeated.

RESULTS

Table 1: I^{131} Rose Bengal Liver Uptake in Subjects Receiving Lime Juice and Subjects receiving Whisky

Group	No. of volunteers	Mean age (Years)	Mean weight (Kg)	Percent I^{131} Rose Bengal uptake	*p*
Lime juice	10	42.6	59.4	70.65 ± 1.97	<0.05
Whisky	10	43.8	61.3	64.86 ± 2.3	

Table 2: I^{131} Rose Bengal Uptake after Ingestion of Whisky in subjects pre-treated with Liv.52 and subjects treated with Lime Juice

Group	No. of volunteers	Mean age (Years)	Mean weight (Kg)	Percent I^{131} Rose Bengal uptake	*p*
Placebo	9	41.9	58.6	63.2 ± 2.68	<0.05
Liv.52	11	44.3	61.8	71.6 ± 3.1	

Table 3: Blood acetaldehyde levels before and after treatment of Liv.52 or Placebo after ingestion of whisky

Group	No. of volunteers	Mean age (Years)	Mean weight (kg)	Acetaldehyde levels μg/ml			
				0	1	2	3.5 hours
Pre-treatment	6	49.4	61.8	Nil	4.6	5.1	4.3
Placebo	3	51.6	63.1	Nil	4.8	6.1	6.0
Liv.52	3	47.3	60.2	Nil	2.6	3.1	1.8

DISCUSSION

The study shows the protective effect of Liv.52 on the acute deleterious effect of alcohol on liver function. Liv.52 also reduces the level of acetaldehyde in the blood after alcohol ingestion without differences in the blood alcohol levels, indicating rapid disposal of acetaldehyde. This mechanism probably is responsible for preventing cellular injury by alcohol and also may be responsible for the strong appetite stimulating action of Liv.52. By removing anorexia, Liv.52 may ensure adequate nutrition and remove the contribution of malnutrition to aggravate liver injury. Thus efficacy of Liv.52 to prevent and favourably influence the course of alcoholic liver disease appears to be due to its action in promoting rapid disposal of acetaldehyde.

REFERENCES

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