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Prolonged Moderate Alcohol Intake and Liver Function

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Ethyl alcohol can affect liver function in two ways: (1) by causing derangements in various metabolic processes while undergoing metabolic degradation, (ii) by exerting a pharmacological action on tissue function (Kalant and Khanna, 1969).

Though in the literature we do not find much of an attempt to distinguish between these two effects, it is now agreed that prolonged alcohol intake produces derangement of liver function. Alcohol is recognised to produce hypoglycaemia (Arky and Freinkel, 1969), and also to inhibit albumin synthesis in the liver (Rothschild *et al,* 1971). In a recent study Dattani *et al.,* (1972), using the Bromsulphthalein retention test, demonstrated that liver function impairment occurs within a few hours of ingestion of whisky, even in amounts compatible with complete self-control–as in 'social drinking'. They also demonstrated that in habitual drinkers a residual impairment may be present continuously.

Since most people who indulge in alcoholic beverages cannot give up the practice (Leevy and Smith, 1970), an agent, which could prevent or ameliorate this dysfunction, would be highly useful. Tryptophan has been shown to have some effect in affording protection against the toxic effects of alcohol (Rothschild *et al.,* 1971).

The present study was planned to observe the effect of alcoholic beverage consumed in moderate quantities by apparently normal, high-income group subjects who were not habituated or addicted to alcohol. Additionally we wished to determine the effect of Liv.52 therapy on liver function impaired by alcohol consumption.

MATERIAL AND METHODS

This study was conducted at the Lokmanya Tilak Municipal General Hospital. There were 17 subjects who gave a history of alcoholic intake over the previous two to ten years.

After taking a detailed history and doing a thorough physical examination, the following liver function tests were performed on each subject:

1. Serum proteins: albumin/globulin.
2. Icteric Index.
3. Thymol turbidity.
4. Van Den Berg test.
5. SGOT/SGPT determination.
6. Bromsulphthalein (B.S.P.) retention.

Of the 17 subjects, 7 showed no abnormality in any of the above tests and were eliminated from the study.

The remaining 10 subjects who showed abnormal B.S.P. retention were asked to continue their usual way of life, including alcohol intake and were asked to take the "medication" 2 tablets 3 times a day for 8 weeks. Weekly visits ensured the proper intake of the medication. The medication

consisted of placebo tablets for the first two weeks and Liv.52 tablets for the next 6 weeks. The liver function tests were repeated at 2 weeks, i.e. at the end of placebo therapy and 8 weeks after starting the treatment, i.e., after 6 weeks of Liv.52 therapy.

Table 1

Subject		Serum proteins	Albumin	Globulin	SGOT	SGPT	BSP Bromsulph-thalein Retention
BNM	1	5.9	2.9	3.0	63	88	12%
	2	5.9	3.0	2.9	62	94	14%
	3	6.3	3.3	3.0	43	80	5%
JNP	1	5.8	3.1	2.7	53	-	16%
	2	5.7	3.0	2.7	59	-	14%
	3	5.9	3.1	2.8	56	-	8%
ASG	1	6.3	3.3	30	63	111	18%
	2	6.3	3.2	3.1	65	106	18%
	3	6.3	3.8	3.0	42	83	6%
CNC	1	5.8	3.0	2.8	35	64	18%
	2	5.9	3.0	2.9	38	76	21%
	3	6.3	3.4	2.9	38	64	9%
PNC	1	5.6	2.8	2.8	88	124	17%
	2	5.7	2.9	2.8	92	126	16%
	3	5.6	2.9	2.7	76	131	18%
BSK	1	5.8	2.8	3.0	64	118	14%
	2	5.7	2.9	2.8	63	116	12%
	3	5.7	2.7	3.0	56	110	12%
SGP	1	6.1	3.2	2.9	64	120	18%
	2	6.2	3.3	2.9	66	110	17%
	3	6.2	3.2	3.0	62	110	18%
SNP	1	5.6	3.2	2.4	50	68	18%
	2	5.8	3.1	2.5	52	66	16%
	3	5.8	3.3	2.3	44	34	10%
SGG	1	6.2	3.8	2.4	70	110	16%
	2	6.0	3.8	2.2	72	112	17%
	3	6.4	4.0	2.4	50	84	8%
PSM	1	6.1	3.8	2.3	73	123	18%
	2	6.1	3.7	2.4	74	106	19%
	3	6.3	3.8	2.5	54	93	6%

1 = Initial values; 2 = After 2 weeks of placebo therapy; 3 = After 6 weeks of Liv.52 therapy

Table 2: Showing Mean B.S.P. retention, Serum Albumin, SGOT and SGPT before and after Liv.52

	Before treatment	After 6 weeks treatment with Liv.52	Comments
B.S.P. retention mean ± SD	16.7 ± 1.8	10 ± 4.4	Significance $t = 4.7$ $p<0.05$
Serum albumin	3.19	3.35	$t=2$ $p<0.05$ (paired t test)
SGOT	62.3	52.1	N.S.
SGPT	102.9	78.6	N.S.

Note: There were no abnormalities and alteration in Icteric Index, Thymol turbidity and Van Den Berg tests

From the tablets it is seen that serum proteins were lowered mainly due to lowering of serum albumin. There was marked increase in the BSP retention and SGOT and SGPT values were raised. After two weeks of placebo therapy there was no alteration in these values but after 6 weeks of Lvi.52 treatment there was significant reduction in BSP retention in 7 out of 10 subjects despite their continuing to drink moderately. There was also a small but significant rise in serum albumin in 6 of the 10 subjects.

DISCUSSION

Even moderate amounts of alcohol ingested occasionally or regularly may produce derangement of liver function. This derangement is reversible in the early stages if complete abstinence is observed. But most people cannot do this. The immediate and short-term effects are possibly a consequence of the metabolism of alcohol itself, whereas more protracted changes like lowering of serum albumin levels may be due to the toxic effect of alcohol on the liver cells.

Joglekar *et al.,* (1963, 1966) have demonstrated in animal models that Liv.52 protects the liver from the injurious effects of chemical agents like carbon tetrachloride and allyl alcohol. They also demonstrated the beneficial effect of Liv.52 when it was administered after the damage was caused.

We, therefore, decided to investigate whether Liv.52 would have a similar beneficial effect on impaired liver function due to alcoholic beverages.

In this study subjects with liver function impairment were given a placebo for the first two weeks after which the liver function tests were repeated and it was found that they remained abnormal. For the next 6 weeks the subjects received Liv.52. The liver function tests repeated at the end of this period showed a significant improvement in B.S.P. retention and serum albumin.

SGOT and SGPT levels were reduced but the differences were not significant mainly because of wide variation in initial values. During the entire period of study the subjects continued their drinking habits as usual.

Though hypoglycaemia and bromsulphathalein retention can occur as an acute effect of alcohol, lowering serum albumin levels occurs only after prolonged exposure and can be considered a direct toxic effect on the liver.

Joglekar *et al.,* (1970, 1971) have shown that Liv.52 has an anabolic effect as well as anti-catabolic effect. Published studies in cases of malnutrition have demonstrated that Liv.52 helps raise the serum protein levels more rapidly (Pai *et al.,* 1971; Dayal *et al.,* 1970; Khetarpal *et al.,* 1972).

In this study we observed a small but significant rise in serum albumin levels with Liv.52 therapy.

All the subjects in this study were well-to-do and in apparent good health. The lowering of serum albumin in these subjects was not due to nutritional deficiency. But it is known that nutritional deficiencies aggravate liver damage by alcohol. Since nutritional deficiencies are very common in our country and since the use of alcoholic beverages is increasing very rapidly, the impairment of liver function may also become widespread. This degree of impairment of liver function may not produce gross ill health but may make the person more susceptible to certain diseases and also to drug toxicities.

In the present study Liv.52 was seen to exert a beneficial effect on the liver function despite continued use of alcohol.

SUMMARY

The present study has shown that social drinking over some length of time causes impairment of liver function as shown by lowering of serum albumin and enhanced retention of bromsulphthalein in a majority of indulging subjects. In a large majority of such subjects, improvement is brought about by Liv.52 despite continued drinking.

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