Efficacy of Kamilari in alcoholic liver cirrhosis

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Abstract

Long term consumption of alcohol will produce cirrhosis of the liver. Here, the beneficial effect of an ayurvedic preparation, Kamilari, in ten cirrhotic patients has been analysed. For a period of four months they were given this herbal drug. The biochemical parameters analysed in serum were aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), cholesterol, bilirubin and protein. Treatment with Kamilari showed significant decrease in the levels of AST, ALT, ALP, cholesterol, bilirubin and increase in total protein. In conclusion it can be said that Kamilari is a hepatoprotective drug.

Ingredients of Kamilari

Thespesya poppulina, Zingiber Officinale, Piper Longum, Elattaria Cardamomum, Glycyrrhiza Glabra, Berberis Aristata, Cordia Myxa, Curculigo Orchiodes

Introduction

Alcoholism is a disease and much of the alcohol-related morbidity and mortality is due to alcoholic liver cirrhosis. Alcohol abuse mainly affects liver function because it is the site responsible for the most part of ethanol oxidation. Within the liver alcohol is acted upon by alcohol dehydrogenase to form acetate and finally to CO, and H,O. Acetaldehyde formed during the metabolism of ethanol is the toxic component, which ultimately produces liver cirrhosis.

Attempts to influence connective rissue accumulation resulting in fibrosis have focused on agents which alter the course of the inflammatory events preceding fibrosis and on agents which have direct effect on collagen synthesis or processing. Various substances such as oral testosterone - an

anabolic steroid, catechin - a flavanoid, herbal preparations such as Liv-52 etc. have been used for the treatment of cirrhotic patients.^{2,3} The present study evaluates the effect of an ayurvedic medicine, Kamilari in the treatment of patients with alcoholic cirrhosis.

Materials and Methods

Ten adult male patients of age between 40-60 years, with histories of chronic alcoholism were selected for this study. Tenhealthy adult males of the same age group without any liver disease were used as marching controls. The patients were instructed to abstain from alcohol for at least one week before and also during the study. They were advised to take Kamilari tablet (850mg) three times daily for a period of four months.

The patients were evaluated clinically and with laboratory indices of routine liver function tests for assessing the severity of liver damage. The biochemical parameters analysed were serum cholesterol, serum bilirubin, serum enzymes such as aspartate aminotransferase (ALT), alanine aminotransferase (ALT), alkaline phosphatase (ALP) and serum

proteins. 12 In liver diseases all these parameters get altered. So they were helpful in the diagnosis of liver diseases.

The above-mentioned biochemical tests were conducted before treatment and then after one month, two months, three months and finally after four months respectively. The initial values obtained in cirrhotic patients were first compared with values obtained for the normal control. The initial values obtained for the experimental group were compared with values obtained after each month for a period of four months. Statistical analysis was done using student's paired 't' test.

Results

Table 1 depicts the effect of Kamilari on the activities of serum transaminases and alkaline phosphatase. Table 2 shows the effect of the herbal drug on the serum levels of cholesterol, bilirubin and total protein in cirrhotic patients.

Before treatment, the serum transaminases and alkaline phosphatases showed significant elevation when compared to normal controls. The biochemical parameters showed significant decrease after four months of treatment with the herbal drug for four months.

The concentrations of cholesterol and bilirubin increased in the serum before treatment whereas the concentration of serum proteins gets decreased. These biochemical parameters were very close to the normal values, after treatment with Kamilari for a period of four months.

Discussion

The association between chronic alcohol abuse and cirrhosis has long been recognised.

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Table 1

Effect of Kamilari on the activities of SGOT, SGPT and serum alkaline phosphatase in alcoholic cirrhosis (N=10) (values expressed as IU/L serum)

Parameters	Normal control	Initial	1 AC			
		values	After 1 month	After 2 months	After 3 months	After
Serum alkaline	45.3 ± 1.17	65.5±2.94*	58.6±2.05		Jinonens	4 months
phosphatase		1 2	20.0 ± 2.03	54.7 ± 1.80	49.8 ± 1.70	47.9±1.43+
SGOT	52.5 ± 1.31	61.0±2.1*	59.2±2.13	55.		
SGPT	45.6±1.14	53.9±1.94*		55.7 ± 1.94	53.9 ± 1.88	52.3±1.32*
±700		J357 £ 1.74	50.2 ± 1.8	48.6±1.7	46.2±1.61	45.2+1.24*

*P < 0.05 as compared to initial readings.

Table 2

Effect of Kamilari on the serum levels of cholesterol, bilirubin and total protein

Parameters			(values expre	essed as mg%)			
	Normal control	Initial values	After 1 month	After 2 months	After	After	
Serum	190+4.75	278±9,45*	0/0 0		3 months	4 months	
cholesterol		27017,43	260±9.1	240±8.4	202±7.07	201.3±5.03+	
SGOT :	1.01±0.02	1.98±0.06*	152.005		1 1 2 2 2 2 2 2		
SGPT	7.50±0.26		1.52±0.05	1.35 ± 0.04	1.21±0.04	1.19±0.02*	
		5.16±0.18*	5.32±0.19	5.65±0.2	5.71±0.2	6.41±0.22*	

*P < 0.05 as compared to initial readings

Cirrhosis of the liver is the third most frequent cause of death in alcoholics.

A variety of agents have been used to treat this hepatic dysfunction. 2.3 Clinical experiments have shown that individuals with a morphologically normal liver developed a fatty liver when given ethanol either in addition to the normal diet or as an isocaloric substitution for carbohydrates in a variety of non-deficient diets.8 Alcohol causes injury to the organelles of the hepatocytes, especially the mitochondria and the endoplasmic reticulum.6

Intracellular enzymes like AST and ALT are tightly bound to particular organelles (such as mitochondria). In cases when the membrane integrity is lost due to the intervention of membrane destabilising agents, the enzyme activities in particular tissues increase and leak out into blood and their activity in serum increases.

Ethanol imparts hepatotoxicity through redox changes produced by the NADH generated in its oxidation via the alcohol dehydrogenase pathway. This

carbohydrates, proteins purines. 11 Ethanol is also oxidised in the liver by a microsomal system containing an ethanol inducible cytochrome P-450 which activates xenobiotics to toxic radicals and this results in acetaldehyde. 9.10 Acetaldehyde, the first merabolite of ethanol is responsible for many of the ethanol induced alterations of hepatic structure and function. The toxic effects of ethanol on hepatic cells include decreased capacity to synthesise albumin and increased lipid peroxidation causing membrane damage.

Treatment of alcoholic cirrhotic patients with Kamilari normalised the activities of serum transaminases and alkaline phosphatase, decreased concentration of cholesterol and bilirubin in serum, elevated the level of protein in serum. From the present study, it may be concluded that Kamilari has curing effect on alcohol-induced liver damage by reducing lipid peroxidation products (like acetaldehyde) or by altering the lipid composition of the plasma

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