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RESEARCH ARTICLE

Hepatoprotective Acitivity of a Polyherbal Formaltion: A histological study.

Puja Tulsiani
Department of Pharmacology, B.R..Nahata College of Pharmacy, Mhow-Neemach Road, Mandsaur, Madhya Pradesh, 458001, India

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Puja Tulsiani
Department of Pharmacology, B.R..Nahata College of Pharmacy, Mhow-Neemach Road, Mandsaur, Madhya Pradesh, 458001, India

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ABSTRACT

The present study explores the hepatoprotective mechanism of a polyherbal preparation. Initially the polyherbal preparation was prepared by extracting the three herbs were choosen as *Glycyrrhiza glabra, Clerodendrum serratum* and *Allium cepa* with hydroalcoholic (50:50) solvent and further combined it to prepare a polyherbal preparation (F1). Further F1 at 200 and 400 mg/Kg were evaluated for acute toxicity and hepatoprotective activity in comparison with Silymarin as standard against Ethanol and Galactosamine induced liver toxicity in albino wistar rats and mice. Parameters like AST, ALT, Bilirubin, TBARS, Lipid peroxidation, ALP, GGTP and GSH were assessed. These biochemical observations were supplemented by the histopathological examination of liver sections. Experimental data suggested that the treatment with F1 protects the liver damage to a significant level from Ethanol and Galactosamine hepatotoxicity. From these results it may be concluded that the preparation significantly showed a dose dependent hepatoprotective effect.

Keywords: Ethanol; Galatosamine; polyherbal preparation (F1); Silymarin hepatoprotection.

1. INTRODUCTION

Objective of the research was to find out the safe polyherbal preparation and evaluate its efficacy against ethanol and galactosamine induced hepatotoxicity. Since Liver, is the largest organ of body, weighing 1 to 1.5 kg and detoxifies almost all the drugs thus is highly endangered and could be protected by regeneration of liver cells/ inhibition of leukotrienes/ detoxifying by conjugating the toxins with glutathione synthesisedn by various hepatoprotective agents [1, 2]. A Polyherbal preparation with the combination of *Glycyrrhiza glabra, Clerodendrum serratum and Allium cepa* confined to the temperate regions of Asia was prepared. In the regard with individual herbs traditional use for hepatoprotection, we have previously evaluated a significant hepatoprotective potential of polyherbal preparation in the light of inhibition of lipid peroxidation and subsequent normalization of (GSH)- related enzymes linked with the antioxidant defence system against Ethanol evoked liver damage in mice and Galactosamine induced liver necrosis in rats. In the present communication, we have assessed further the hepatoprotective activity of polyherbal preparation in vivo by monitoring serum enzyme level of glutamate pyruvate transaminase (SGPT), glutamate oxaloacetate transaminase (SGOT), alkaline phosphatase, gamma glutyl transpeptidase (GGTP), serum bilirubin, MDA (malonyl di aldehyde) (Babaie et al., 2007), liver weight, which are known to be the most frequent and satisfactory markers for evaluating hepatocellular damage[3,4]. By alcohol dehydrogenase, ethanol affects the metabolism of lipids, carbohydrates, proteins and purines while ethanol by cytochrome P-450 (P-450IIE1) which contributes to ethanol metabolism, tolerance and increased production of acetaldehyde, inhibition of mitochondrial electron transport chain, replacement of fatty acids in mitochondria, loss of membrane structure and integrity. Thus results in elevated levels of glutamyl transpeptidase, inhibits the glutathione peroxidase. [5,6]

2. MATERIALS AND METHODS

2.1. PREPARATION OF POLYHERBAL PREPARATION

Fresh root barks of Glycyrrhiza glabra (300) were dried and crushed in grinder and treated with petroleum ether (5 times the voume of drug) for 24 hours and further treated with 1:5 (drug: solvent) of 50:50 hydroalcoholic solvent for 52 hours[5]. *Clerodendrum serratum* 200 g while *Allium cepa* 600 g produces a yield of 6.66, 6.5 and 3.33%[6]. The extraction was carried out in a cold room (20+ 1ºC). The homogenate was then dried on water bath till semisolid dry extract was obtained which was then shade dried.

2.2. TREATMENT OF ANIMALS

Adult swiss albino rats, weighing between 100-150 g, were acclimatized to conditions in the laboratory (room temperature, 60-80% relative humidity, day night cycle) for 10 days prior to the commencement of the treatment, during which they received food and tap water ad libitum. Rats were then divided in several groups of 6 rats each. Initialy Acute toxicity study and DPPH free radical scavenging activity were performed on rats and further evaluation of hepatoprotective activity was done through carbon tetrachloride model as follows[7,8]. Mice were divided in different groups and were administered ethanol after 7 days of polyherbal treatment in all groups except model control. Further 3 doses of ethanol (0.5ml orally) were given in 12 hours duration and thereafter 10 hours, they were sacrificed. Finally the blood was withdrawn before sacrification for the serum collection and biochemical testing. [5] While in other model, after 7 days of continuous administration of polyherbal preparation orally, galactosamine was administered after 24 and 96 hours i.p. in treated groups while normal saline was given in model control group and standard drug in Standard group.[6]

3. RESULTS

In both Ethanol induced and Galactosamine induced hepatotoxicity, there occurred a rise in enzyme levels like SGOT, SGPT, ALP, GGTP, Lipid peroxidation while a fall in GSH and weight of liver of mice as: Model control> Treated 200 mg/Kg> Treated 400 mg/Kg> Standard> Control. While a decline in centrilobular necrosis and ballooning degeneration in F1 treated was observed in histology reports.

The possible mechanism of polyherbal preparation as hepatoprotective agent could be by substantially decreasing lipid per-oxidation [9] with a fall of MDA level in liver homogenate and a concurrent rise in hepatic GSH content as compared to model control mice liver against ethanol induced hepatotoxicity. The binding of acetaldehyde, a metabolite of ethanol with glutathione lead to a fall of glutathione, this was prevented thus leading to the rise of GSH content, decline in lipid peroxidation as well as targeting to the maintenance of NADPH/NADP. [10-13], Thus the target could be the maintenance of ϒ-glutamyl transpeptidase level in blood plasma.[14] The primary disturbance involves a depletion of uridine nucleotides with subsequent effect on mucopolysaccharide and sugar metabolism through Glactosamine which was also

prevented [6]. Thus F1 could be administered safely

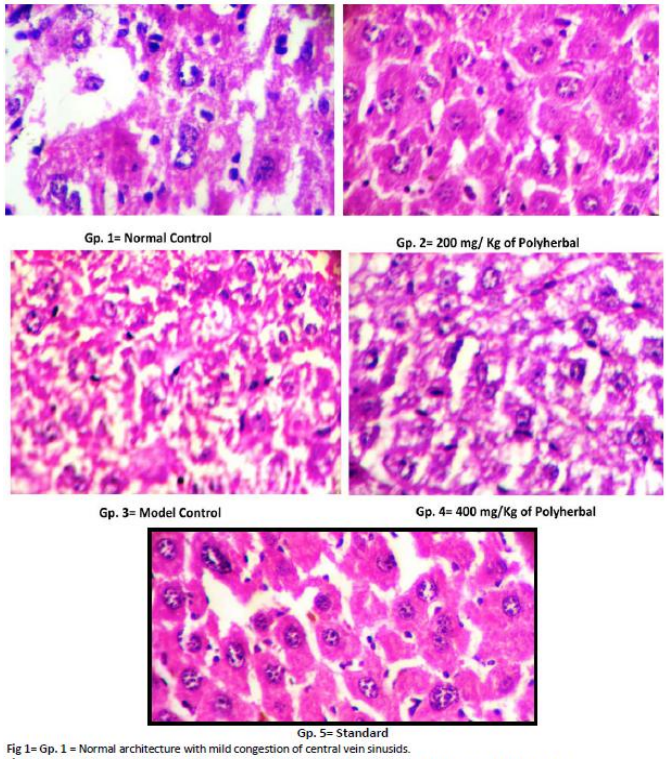

Fig 1= Gp. 1 = Normal architecture with mild congestion of central vein sinusids.
Fig 2= Gp. 2 = Normal architecture with ballooning degeneration of hepatocytes with moderate inflammation.
Fig 3= Gp. 3 = Nuclear hyperchromic with chronic inflammation and severe congestion. Thus depicting highest level of necrosis.
Fig 4= Gp. 4 = Evidence of hepatocytic regeneration with slight congestion.
Fig 5= Gp. 5 = Mild congestion and inflammation.

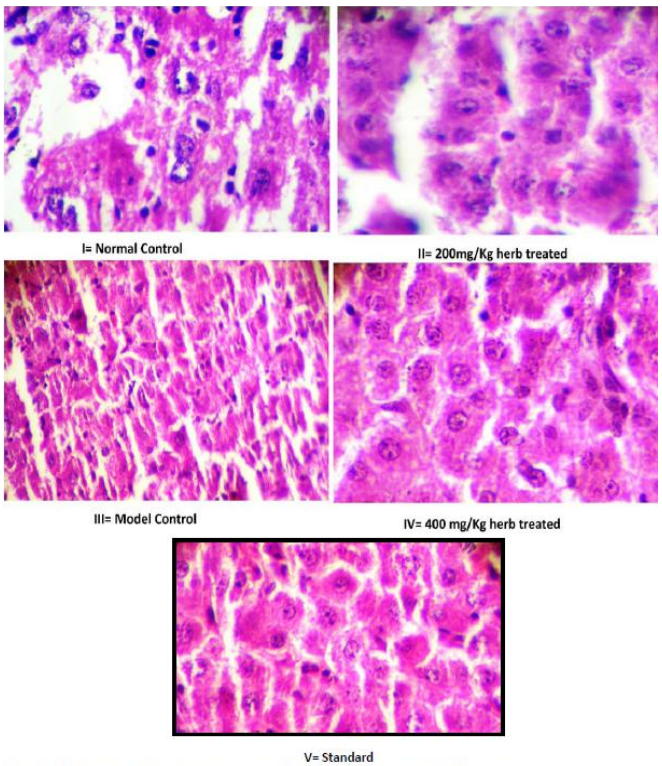

Fig 6= Gp. I= Normal architecture of hepatic section with no observation of necrosis or inflammation;
Fig 7= Gp. II= Architecture along with congestion and chronic inflammation in perisinusoidal and periportal area.
Fig 8= Gp. III= shows architecture along with congestion of central vein along with chronic inflammation in periportal a perisinusoidal area.
Fig 9= Gp. IV= Liver lobule along with congestion of central vein and portal area shows acute inflammation.
Fig 10= Gp. V= shows intact architecture: The sinusoids shows congestion with perivascular infiltration. There is slight inflammati infiltrate in between hepatocytes

and will prove in hepatotoxicity caused by Ethanol or Galactosamine. Also the preparation proved to be dose dependent hepatoprotective as well as in comparison to Silymarin as standard drug. Hence it could be oncluded that the polyherbal preparation is a free radical scavenger, Glutathione content maintainer, declining the membrane permeability and benefiting Y-glutamyl transpeptidase[15, 16].

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