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Original Research Article (Experimental)

Amelioration of anti-hepatotoxic effect by *Lichen rangiferinus* against alcohol induced liver damage in rats

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ABSTRACT

Background: Reindeer lichen, *Lichen rangiferinus* syn. or *Cladonia rangiferina* (L.) F. H. Wigg. (Cladoniaceae) has been traditionally reported as a remedy to treat fever, colds, arthritis as well as convulsions, liver infections, coughs, constipation, and tuberculosis. The current study is aimed at rectification of alcohol induced liver damage by the use of *L. rangiferinus* extract.
Objectives: The aim of the study was to compare some biochemical markers for liver injury and hematological indices in normal untreated rats and treated rats.
Material and Methods: The study was performed using male Wistar rats. Animals were categorized into five groups, negative control group (normal diet only), treated groups (2 groups were lichen treated along with 10% ethanol & 1 group was only ethanol treated) and positive control group (Silymarin + 10% ethanol) of six animals in each group. Biochemical markers for liver injury and hematological indices of all animals were measured using standard diagnostic tools. The animals were then sacrificed and livers were sent to the pathology lab for histopathological analysis.
Results: Lichen extract showed a significant restoration of altered biochemical parameters towards normal in both *in vitro* and *in vivo* conditions. The total phenolic and flavonoid content of the LRE was found to be 21.78 μg PE/mg of extract and 5.13 μg RE/mg of extract respectively. The IC_{50} values for atranorin and fumarprotocetraric acid were found to be 128.48 and 218.46 mg/mL respectively. Reducing power of the extract was found to be quite significant. After administration of lichen extract, endothelial cells were less injured around central vein and number of fat vacuoles was also lesser in hepatocytes.
Conclusion: Conclusively, treatment with lichen extract assuages alcohol-related damage and guards hepatic tissue from alcohol-induced toxicity.

1. Introduction

Alcohol liver disease is a highly pervasive ailment among human beings which environs a range of hepatic disorders starting from simple steatosis (fatty liver) to cirrhosis/liver failure [1]. Ethanol consumption enhances the ratio of NADH/NAD^+ in hepatocytes which causes disruption of β-oxidation of fatty acids in mitochondria leading to steatosis. Alcohol also increases the lipid transport to the liver from the small intestine leading to enhanced mobilization of fatty acids from adipose tissue which is taken up by the liver [2]. This causes damage to cell membrane of hepatocytes leading to augmented levels of transaminases (Alanine aminotransferase (ALT) and AST (Aspartate aminotransferase)) in blood stream. Alkaline phosphatase (ALP) is also present in hepatocytes which come into the circulation indicating hepatic damage. Gamma-glutamyl transferase (GGT) has a key role in preserving intracellular homeostasis of oxidative stress which protects cells against oxidative damage. It is present in cell membrane and is set free in circulation when cell membrane is damaged. Glutathione (GSH) is a powerful antioxidant in our body which prevents damage by oxidative stress which is lowered by alcohol. Alcohol intake causes accumulation of oxidative stress markers (Glutathione reductase (GR) and malondialdehyde (MDA) which causes further damage to liver [3].

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