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Efficacy of Alcoholism on Histology of Liver of Swiss Albino Mice

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***Abstract*— It is known that chronic ethanol treatment can increase the degree of liver damage by hypoxia. The amount of fat in the liver increases progressively during the period of alcohol intake. Other workers have also suggested that women are more likely to develop alcoholic liver disease than men at equivalent levels of consumption. Alcoholic liver disease is usually classified histologically into fatty liver, alcoholic hepatitis and cirrhosis. The histological features of alcoholic hepatitis included degenerative changes in the hepatocytes , hepatocytes necrosis, infiltration with polymorphonuclear leucocytes and deposition of Mallory's alcoholic hyaline (eosinophilic perinuclear cytoplasmic inclusions).**

***Keywords*— Alcoholism, Histology, Swiss Albino Mice**

I. INTRODUCTION

There are many health risks of chronic alcohol abuse, ranging from high blood pressure stroke. Heavy drinkers have an increased risk of jaundice, cirrhosis, liver failure, liver cancer, and many other conditions. The definition of heavy drinking is consuming 8 drinks or more per week for women and 15 or more for men. Even a single binge drinking episode can result in significant bodily impairment, damage or potentially death.

Earlier studies have showed that rats chronically treated with ethanol experienced an increased degree of centrilobular necrosis of the liver when exposed to hypoxia in vivo. Changes in at least two physiological parameters could be responsible for the increased damage after ethanol treatment. One factor, as by other studies, is an increased in total oxygen demand by the liver. If the oxygen demand exceeds oxygen supply from circulating blood, then cells in the regions with least access to blood (centrilobular areas) will become anoxic. A second factor is the resistance of the liver cells transient or sustained episodes of low oxygen tension.

The present work demonstrates the effect of alcohol on the histology of liver. For this study, Swiss albino mice were selected and purchased from Calcutta and from Patna of Bihar state. The albino mice were administered with alcoholic beverages like rum, country liquor, mahua and dudhia for 5 to 15 days daily and the histological slides of liver tissue were prepared and examined for any effects.

II. MATERIAL METHOD

Slide preparation begins with fixation of tissue specimen collected from liver of the albino mice. This is a crucial step in tissue preparation, and its purpose is to prevent tissue autolysis (destruction of tissue by their own enzymes) and putrefaction (decay or rotting of the tissue). Tissue samples transferred into a suitable fixative (e.g. 10% neutral buffered formalin) immediately after collection. This allows most tissues to become adequately fixed within 24-48 hours. After fixation, specimens are trimmed using a scapel to enable them to fit into an appropriately labeled tissue cassette. The filled tissue cassettes are then stored in formalin until the processing begins. Processing tissues into thin microscopic sections is usually done using a paraffin block, as follows : Dehydration is the first step, which involves immersing tissue specimens in increasing concentration of alcohol to remove the water and formalin from the tissue, Clearing is the next step in which an organic solvent such as xylene is used to remove the alcohol and allow infiltration with the paraffin wax. Embedding is the final step, where specimens are infiltrated with the embedding agent, usually paraffin wax. The tissue becomes surrounded by a large block of molten paraffin wax, creating what is now referred to as the 'block'. Once the block solidifies, it provides a support matrix that allows very thin sectioning. Tissue specimen is now ready to be cut into sections that can be placed on a slide. Wax removed from the surface of block to expose the tissue. A microtome used to slice extremely thin tissue sections off the block in the form of a ribbon.

Histochemical stains (Haematoxylin and Eosin) used to provide contrast to tissue sections, making tissue structures

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more visible and easier to evaluate. After staining, a coverslip is mounted over the tissue specimen on the slide, using optical grade glue, to help protect the specimen. Then the specimen is studied under a compound microscope.

III. RESULT

The five micron thick and double stained T.S. of liver of 5 days Rum treated mice showed the following histological variations :

1) Intralobular vein intact.
2) Clear cut section of blood vessel.
3) No change in hepatic cells.

The T.S. of liver of 15 days Rum treated albino mice exhibited the following histological characteristics :

1) Great damage of hepatic cells.
2) Disintegration of Glisson's capsule.
3) Necrosis at several places.
4) Large vacuoles in the tissue. Vacuolated cells.
5) The lesions usually concentrated in the centrilobular areas around the control vein and obliteration of the lumen appeared.
6) Sclerosis around the control vein and obliteration of the lumen appeared.

The 5 days Country liquor caused no change in the histology of liver.

The T.S. of liver of 15 days Country liquor treated albino mice showed the following histological features :

1) Enlargement of hepatic cells.
2) Enlarged Glisson's capsule.
3) No vacuolation like rum treatment.

The 5 days Mahua treatment was found not effective on the histology of liver.

The T.S. of liver of 15 days Mahua treated mice illustrated the below mentioned variations at histological levels :

1) Shrinkage of hepatocytes.
2) Vacuolization of hepatocytes.
3) Granulation of cells.
4) Increase in the no. of Kupffer cells.
5) Portal hypertension even before cirrhosis had developed.

The T.S. of liver of 5 days Dudhia treated albino mice demonstrated the following histological features :

1) Shrinkage of hepatocytes.
2) Degeneration of cells.
3) Appearance of damaged area.
4) Necrosis at certain places.
5) Appearance of phagocytic cells.
6) Disappearance of intralobular vein.
7) Loss of Glisson's capsule.

The double stained section of liver of 15 days Dudhia liquor treated mice exhibited the following characteristics :

1) Shrinked hepatocytes.
2) Maximum disintegration of hepatocytes.
3) Large no. of Kupffer cells.
4) Necrotic tissue at several places.
5) Liver cirrhosis.
6) Primary hepatocellular carcinoma, a late complication of alcoholic cirrhosis was seen.
7) Smooth hepatomegaly often seen on clinical examination.

IV. DISCUSSION

It was found that the liver responded to all types of alcohol (rum, country liquor, mahua and dudhia). The acute alcoholism caused a least or no hepatocellular variation but the chronic use of alcohol caused liver cirrhosis, damage of Kupffer cells to the great extent that animal becomes susceptible to any antigens and pathogens. The animal loses its immune capacity. The rum and dudhia were found to be more effective on liver than that of country liquor and mahua.

The disorientation of Glisson's capsule might be due to oozing out of water molecules from hepatic cells. Alcohol absorbs water from the tissue. The reduction in water content in the cell caused metabolic derailment. The proper functioning of the cells disturbed and their degradation starts. The dangerous histological abnormality like cirrhosis and carcinoma influenced most of the metabolic changes. Thus alcoholism was proved to be dangerous for haematological and histological point of view.

V. SUMMARY AND CONCLUSION

Steatosis was found in alcoholic liver but most marked are the morphological changes, with centrilobular necrosis, and ballooning of hepatocytes were due to alcoholism. The disorientation of Glisson's capsule might be due to oozing out of water molecule from hepatic cells.

Five days mahua and dudhia liquors treatment caused no significant change in the liver. The prolonged use of dudhia liquor caused liver cirrhosis, primary hepatocellular carcinoma and smooth hepatomegaly. Moreover, alcoholism caused hepatocellular necrosis, infiltration of polymorphonuclear leucocytes, lesion in the centrolobular

areas around hepatic veins, sclerosis around central vein and obliteration of lumen, portal hypertension even before cirrhosis, primary hepatocellular carcinoma and smooth hepatomegaly.

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