Assessment of Mercuric Chloride Intoxication in albino Rats on the Basis of Hepatobiochemistry, Serum Biochemistry, Histochemistry & Histopathologh
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ABSTRACT
Background: Exposure to any xenobiotic can disturb the metabolic activities of particular organs. Hence present study is designed to find changes in hepatic biochemistry, histochemistry and histopathology besides serum enzyme levels after acute and sub-acute treatment with mercuric chloride, a heavy metallic compound, in albino rat (Rattus norvegicus).

Materials & methods: Thirty albino rats were divided into two groups, one for acute and other for sub-acute study. Each group were divided into 3 sub-sets (6, 12 and 24 hrs) for acute and (7, 14 and 28 days) sub-acute treatments, with 5 rats in each. The controls were also taken with similar references. Mercuric chloride was given orally 0.926 mg/kg body wt. for acute and 0.330 mg/kg body wt. for sub-acute sets (LD₅₀ = 9.26 mg/kg body wt) determined by probit analysis. The control groups received distilled water only.

Results: The result revealed that mercuric chloride caused fall in glycogen content, while elevations in cholesterol, total lipid, free fatty acids and protein levels after acute and sub acute treatment with mercuric chloride, while serum biochemistry showed significant increases in alanine aminotransferase, aspartate aminotransferase and alkaline phosphatase. On the other hand histochemical results also revealed reduction in the presence of glycogen and protein in hepatic lobules after acute and sub-acute mercuric chloride intoxication. Histopathological study showed pyknotic, degenerative and bi-nucleated nuclei, vacuolization and hepatocellular fluid accumulation inside centrilobular region at 100X fields.

Conclusion: On the basis of hepatobiochemical, histochemistry, histopathological and serum biochemistry alterations it becomes clear that mercuric chloride exhibit liver toxicity to a considerable extent. So there is an urgent need to develop a novel hepatoprotective formulation against mercuric chloride toxicity.

Key words: Mercuric chloride intoxication, Cholesterol, Total lipids, Alanine aminotransferase, Alkaline Phosphatase, Glycogen, Hepatic.

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