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Prevention of Mercuric Chloride induced Histopathological Changes in the Small Intestine of Mice with Liv.52

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Inorganic mercury poisoning in human beings causes cardiovascular collapse, acute renal failure and severe gastrointestinal damage. Careful survey of literature revealed that no study was aimed at preventing mercuric chloride induced gut pathology. Common Indian food items have been found to have high mercury contents[1,2]. A multiherbal hepatotonic drug had been reported to prevent pathological changes in mice duodenum and small intestine following cadmium chloride intoxication[3].

MATERIAL AND METHODS

Six months old male Swiss albino mice, obtained from the Biological Products Division, Veterinary College, Mhow (M.P.) were used. Experimentation consisted of the following grouping and treatments. Group I Controls (C). Mice on standard food and Hg-free water, *ad labitum*. Group II Mercuric Chloride Treated (P) mercuric chloride (Ranbaxy, 99.9% analyzed reagent) dissolved in distilled deionized water to prepare solutions of 1 mM 5 mM concentration. These were administered as drinking water for 100 days and 30 days respectively. Group III Mercuric Chloride Treatment + Liv.52 Administration (P + D). Mice drinking 1 mM or 5 mM mercuric chloride solutions; each mouse was given 0.5 ml Liv.52 syrup/day for 100 or 30 days respectively. (11 mM mercuric chloride solutions; each mouse was given 0.5 ml Liv.52 syrup/day for 100 or 30 days respectively. (1mM = 270 µg ml, and 5 mM = 1935 µg ml) Group IV (PT). After mercuric chloride exposure, as in 'P' group, each mouse was given 0.5 ml. Liv.52 syrup/day for next 15 days. Mice were shifted to Hg-free water during this fortnight. Group V (NR). After mercuric chloride exposure as in 'P' group, mice were allowed to recover naturally. They were maintained on Hg-free water.

In each group 10 mice were placed and experiment was set in triplicate. Animals of group C, P, P + D were killed on 31st or 101 day while animals of PT and NR were killed on 46th and 116th day. Bouin's fixed small intestine was sectioned and stained in Delafield's hemotoxylin. Photomicrographs and observation of slides have formed the basis of present results.

RESULTS

5 mM mercuric chloride administration to mice for 30 days caused disorganization and reduction in the height of villi. These pathological changes were not evident when drug was also given along with mercury exposure. Post-therapy remained ineffective as disorganized villi were observed. 1mM mercuric chloride administration to mice for 100 days caused erosion of serosa, fusion and disorganization of villi but in the presence of drug Liv.52 no adverse effects of mercury were noticed. Effectiveness of drug during post-therapy is only slightly better over natural recovery. In this experiment there occurred 50% mortality in 'P' group and 20% mortality in P + D group at 5 mM $HgCl_2$ administration.

DISCUSSION

Ingestion of mercuric chloride and inhalation of Hg-vapor results in the necrosis of gut mucosa in human beings and experimental animals[4]. In the present experiment 5 mM mercuric chloride solution had quite high Hg i.e. 1035 µg/ml. If each mouse consumed one to two milliliters of this solution then severe pathological changes in the gut and even their death is not an unexpected finding as LD_{50} for mouse in 10 mg/kg body weight[5].

The multiherbal drug Liv.52 has been found to stabilize lysosomes and to inhibit activities of acid phosphatase, cathepsin-B and deoxyribonuclease[6]. It lowers lipid peroxidation and enhances activities of cytochrome P-450, ATPase, cytochrome-C-oxidase and SDH[7-10]. Interestingly GSH and sodium selenite has already been reported to nullify mercuric chloride induced alterations in the activities of sodium potassium and magnesium dependent ATPase, acid and alkaline phosphatased, succinic dehydrogenase and protein contents in mice gut[11].

SUMMARY

Mercuric chloride was administered in drinking water to mice at 1 mM and 5 mM for 100 and 30 days respectively. Lower concentration caused mild pathological changes in the small intestine while higher concentration caused severe pathological changes. Pathological symptoms were less pronounced when Liv.52 was administered along with 5 mM mercuric chloric and Hg-induced changes were totally absent when drug was used along with 1 mM Hg Ci2 solution. After Hg-exposure at both concentrations mice were allowed to recover naturally or with drug (Post-therapy). Again, use of drug appeared useful. At least under laboratory conditions this herbal drug seems to reduce Hg-Induced pathological change in small intestine of mice.

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