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Original Research Article

Assessment of renoprotective effect of *Hygrophila auriculata (*K. Schum*)* Heine against mercury-induced nephrotoxicity defects

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ABSTRACT

Background: The therapeutic potential of *Hygrophila auriculata* (K. Schum) Heine *(Syn. Astercantha longifolia)* against mercury-induced nephrotoxicity in rats was investigated, specifically assessing its capacity to restore altered mercury-sensitive indices in urine and isolated renal brush-border membrane preparations.
Methods: Toxicity was induced in male Wistar albino rats by administering mercuric chloride (HgCl2) at 1 mg/kg body weight intraperitoneally three times a week for two weeks. Following mercury induction, rats were treated with an ethanolic extract of the whole plant of *Hygrophila auriculata* at 100 mg/kg body weight orally for 10 days to evaluate its protective/reparative effects on kidney weight changes, urinary constituents (urea, uric acid, creatinine, and protein), urinary enzyme activities (alkaline phosphatase, acid phosphatase, lactate dehydrogenase, N-acetyl-β-D-glucosaminidase, γ-glutamyl transferase, and β-D-glucuronidase), and isolated brush-border membrane enzymes.
Results: Mercuric chloride administration caused significant alterations in kidney weights, elevated levels of urinary constituents, and increased activities of urinary enzymes due to cellular damage. Conversely, mercury administration resulted in a decline in the activities of isolated brush-border enzymes. Post-treatment with the ethanolic extract of *Hygrophila auriculata* significantly ameliorated these toxic effects and successfully improved the altered parameters toward normal levels.
Conclusion: The post-treatment administration of *Hygrophila auriculata* demonstrates significant efficacy in protecting and maintaining renal integrity and functional parameters against mercury-induced nephrotoxicity in rats.

Keywords: Mercuric chloride, Brush-border membrane, Nephrotoxicity, Urinary enzymes

INTRODUCTION

Mercury has a long history of inflicting harm to those who come in contact with it very frequently. The toxicity of mercury was realized by the 18th century, at a time when mercurial ointments were very popularly used to various diseases especially syphilis1. Among the various forms, mercuric chloride ($HgCl_2$) is known for its rapid and preferential accumulation in the renal cortex producing necrosis of epithelial cells in the pars recta of the proximal tubule. The proposed mechanisms for the selective straight proximal tubular (SPT) injury invivo include tubular retention of $HgCl_2$ as it progresses through to SPT sites; tubular secretion or selective accumulation of $HgCl_2$ by the SPT.[1] The basic mechanism is thought to be primarily due

to the interaction of Hg^{2+} with the sulphydryl groups of cellular proteins and enzymes. Besides, it may also bind to membrane phosphotidylserine and phosphotidylethanolamine groups thereby inducing cellular toxicity.[2] The ultrastructural changes are consistent with irreversible cell injury which includes actual disruption of mitochondria, release of lysosomal and brush-border enzymes and rupture of cell membranes.[3] It alters plasma membrane functions such as transport of ions, glucose and also mitochondrial energy status thus upsetting cellular metabolism. Several researchers have reported that mercury induced renal damage is manifested by protienuria, enzymuria and glucosuria.[4] Urinary enzyme levels have been shown to be elevated in a wide spectrum of renal diseases including glomerular and interstitial as well as tubular disease, often prior to the onset of renal failure or even before any abnormality5. Changes in urinary parameters may be due to one factor or a combination of several factors including increased plasma levels or decreased reabsorption of the compounds as well as structural lesions in the nephron.[5]

The most important part of the nephron is the proximal tubule as far as nephrotoxicity is concerned. Alterations of brush- border enzymes reflect early changes in the kidney after chemical insult. It appears logical, therefore, to look for an index of this part of the nephron, which would permit early recognition of alterations to brush-border enzyme activities5. Furthermore, reports also exist advocating complexation of Hg(II) to cellular thiols to promote ROS formation. The plant *H. auriculata* (K. Schum) Haine (Syn.) *Astercantha longifolia Nees* is known to possess antitumor, hypoglycaemic, free radical scavenging and lipid peroxidation activities.[6-8] The literature survey revealed that there are no scientific studies carried out regarding nephroprotective activity of the *H. auriculata*. Hence, the present study is focused to evaluate the efficacy of plant against mercury-induced renal defects with reference to isolated brush border enzyme activities.

METHODS

Animals

Healthy male Wistar albino rats weighing 120±30 g purchased from Tamil Nadu Veterinary and Animal Sciences University, Chennai, were used for this study. Animals were housed in polypropylene cages and were provided certified rodent pellet diet and water ad libitum. They were maintained at 27 °C to 37°C with 12 h light and dark cycle. All animal experiments were performed in accordance with the strict guidelines prescribed by the Institutional Animal Ethical Committee (IAEC) after getting necessary approval.

Collection of plant material

The plant is widely distributed throughout India, Srilanka, Burma, Malaysia and Nepal. The plants were collected from Red Hills, Tamil Nadu. The plant specimen was authenticated by Dr. S. Jayaraman, Plant Anatomy Research Center, Chennai, Tamil Nadu, India. The study was conducted from January 2018 to September 2019 at the Department of Pharmacology and Environmental Toxicology, University of Madras, Chennai, where a voucher specimen was deposited

Preparation of plant extract

The whole plant was shade dried and coarsely powdered. The powder was then extracted with ethanol (60-70˚C) using Soxhlet extractor. The extract was dried under reduced pressure using rotary flash evaporator. The percentage yield of alcoholic extract was 12% w/w. The extract was stored in refrigerator for further studies.

Experimental design

Rats were divided into 4 groups with 6 animals in each group. The experimental design was as follows:

Group I rats served as controls and were treated with normal saline (0.9% NaCl). Group II rats were administered with HgCl2 (1 mg/kg b.w., i.p.) three doses per week for two weeks. Group III rats were administered with HgCl2 dosage as in group II and then extract of H. auriculata (100 mg/kg b.w, p.o) for ten days. Group IV rats were treated with H. auriculata extract alone.

Collection of rat tissues

After experimental period, the animals were sacrificed by cervical decapitation under ether anesthesia. The kidneys were excised immediately and washed in ice-cold saline for the isolation of brush-border membrane from renal cortex.

Isolation of kidney brush-border membrane

The method of Malathi et al was adopted for isolation of brush-border membrane from kidney cortex using 2mM Tris–HCl (pH 7.0) and 1M $CaCl_2$ in a refrigerated ultracentrifuge.[9] The pellets obtained were suspended in appropriate buffer and used for the assay of various enzymes. Alkaline Phosphatase (ALP) and acid phosphatase (ACP) were measured by the procedure of King.[10] The procedure of Kawai and Anno was followed for the determination of β-glucuronidase (β-Glu) activity.[11] The activity of γ-glutamyl transferase (γ-GT) was estimated according to the procedure of Orlowski and Meister.[12]

Collection of rat urine

Another set of control and experimental rats (Group I–IV) were housed individually in metabolic cages for 24 h urine collection. The rats were provided with water but no feed was given. The urine collected on ice was free from faecal contamination. Urine samples were centrifuged and

aliquots separated. One portion was acidified with concentrated hydrochloric acid and used for the analysis of urea, uric acid, creatinine and protein. The remaining was dialyzed at 4º C against distilled water for 3 h and later was used for the determination of various enzymes and protein.

Assays in urine

Urea was assayed by the method of Natelson et al.[13] Uric acid and creatinine were estimated by the method of Caraway and Owen et al respectively.[14] Protein content was determined by the method of Lowry et al.[15,16] The method adopted for the estimation of lactatedehydrogenase (LDH) was that of King and N-acetyl-β-d-glucosaminidase (NAG) was determined by the method of Maruhn.[17,18] The activities of ALP, ACP, γ-GT and β-Glu in urine were carried out as mentioned above in the previous section.

Statistical analysis

The data obtained was subjected to One-way ANOVA and Tukey's multiple comparison test was performed using SPSS statistical package (Version 7.5). Values are expressed as mean ± S.E.M. Value $p<0.05$ was considered significant.

RESULTS

Body and kidney weights

Table 1 Shows, $HgCl_2$ administration resulted in a significant decreased ($p<0.01$) in body weight of group II animals when compared with control animals. On the other hand, the kidney weights were significantly increased ($p<0.001$) in group II $HgCl_2$ induced animals when compared with group I control animals. Whereas on treatment with *H. auriculata* (group III) the body weight and kidney weight were reverted towards normal when compared with control animals which shows the influence of *H. auriculata* against $HgCl_2$ induced toxicity.

Urinary constituents

Table 2 Shows the parameters determined in the urine of control and experimental animals. $HgCl_2$ toxicity induced rats (group II) showed a constellation of disorders of renal tubular function witnessed by increased urea, uric acid, creatinine and protein. Significant ($p<0.001$) reversal was observed in group III animals treated with *H. auriculata*.

Urinary enzymes

Urine analysis of some of the markers of renal injury is highlighted in Table 3.

Units – ALP, ACP and β-D-Glucuronidase -µmoles of p-nitrophenol liberated/mg protein/min, γ-Glutamyl transpeptidase - (IU/l), LDH: µmoles of pyruvate liberated/mg protein/min, Cathespin-nmoles tyrosine formed/hr/mg protein, N acetyl- β D Glucosaminidase: µmoles of phenol liberated/mg protein/min. a-compared with group I and b-compared with group II. HgCl 2 administered rats (group II) showed a significant ($p<0.001$) increase in the activities of urinary enzymes such as ALP, ACP, LDH, γ- GT and β- Glu indicating tubular and lysosomal damage. Effective suppression of the enzyme leakage in the urine was achieved with the treatment of H. auriculata. Brush-border membrane enzymes.

Table 4 Shows Rats exposed to $HgCl_2$ demonstrated alterations in the specific activities of the isolated renal brush border enzymes (ALP, ACP, γ- GT and β- Glu). All the four enzymes were significantly ($p<0.001$) decreased in renal brush border of group II animals, when compared with control animals. Whereas on treatment H. auriculata (group III) the enzyme activities were significantly ($p<0.001$) reverted towards normal, when compared with $HgCl_2$ administered group II animals. There were no significant changes in the enzyme activities of (group IV) *H. auriculata* alone treated animals when compared with control animals.

Units – β-D-Glucuronidase -µmoles of p-nitrophenol liberated/mg protein/min, γ-Glutamyl transpeptidase - (IU/l). LDH: µmoles of pyruvate liberated/mg protein/min, Cathespin-nmoles tyrosine formed/hr/mg protein, N acetyl- β D Glucosaminidase: µmoles of phenol liberated/mg protein/min. a-compared with group I and b-compared with group II.

Table 1: Body and kidney weight of control and experimental animals.

Particular	Group I (Control)	Group II ($HgCl_2$)	Group III (HgCl2+ *H. auriculata*)	Group IV (*H. auriculata*)
Body weight (gm)	186±13.56	153±14.78^{a***}	174±15.37 ^{a**} ^{b**}	182±16.74 ^{aNS}
Kidney weight (gm)	0.94±0.097	1.53±0.14 ^{a***}	1.27±0.18 ^{a**} ^{b**}	1.01±0.12 ^{aNS}

Each value represents mean±SEM of six individual animals. Significance at ***p<0.001, **p<0.01, * p<0.05. NS - Not significant. a-compared with group I and b-compared with group II.

Table 2: Levels of urinary constituents of control and experimental animals.

Particular (mg/dl)	Group I (Control)	Group II ($HgCl_2$)	Group III ($HgCl_2$+ *H. auriculata*)	Group IV (*H. auriculata*)
Urea	23.27±2.01	47.56±4.63 ^{a***}	37.41±3.4 $^{a**\ b***}$	22.79±2.14 $^{a\ NS}$
Uric acid	4.26±0.40	6.30±0.58 ^{a***}	5.12±0.54 $^{a**\ b***}$	4.25±0.39 $^{a\ NS}$
Creatinine	9.65±0.92	14.67±1.38 ^{a***}	11.47±1.2 $^{a**\ b***}$	9.62±0.97 $^{a\ NS}$
Protein	8.36±0.81	15.27±1.45 ^{a***}	9.76±.089 $^{a**\ b***}$	8.32±0.79 $^{a\ NS}$

Each value represents mean±SEM of six individual animals. Significance at ***p<0.001, **p<0.01, * p<0.05. NS - Not significant.

Table 3: Activities of marker enzymes in urine of control and experimental animals.

Particulars	Group I (Control)	Group II ($HgCl_2$)	Group III ($HgCl_2$+ *H. auriculata*)	Group IV (*H. auriculata*)
γ-glutamyltranspeptidase	3.24±0.31	4.98±0.37 ^{a***}	3.84±0.32 $^{a**\ b*}$	3.14±0.31 $^{a\ NS}$
Cathespin D	0.02±0.01	0.98±0.09 ^{a***}	0.41±0.04 $^{a**\ b**}$	0.03±0.01 $^{a\ NS}$
ALP	0.04±0.02	1.68±0.14 ^{a***}	0.44±0.11 ^{a***b***}	0.05±0.03 $^{a\ NS}$
ACP	0.03±0.04	1.27±0.13 ^{a***}	0.46±0.07 $^{a**\ b***}$	0.07±0.04 $^{a\ NS}$
β-glucuronidase	0.34±0.02	0.94±0.08 ^{a***}	0.51±0.04 $^{a**\ b**}$	0.32±0.03 $^{a\ NS}$
LDH	0.21±0.01	0.42±0.03 ^{a***}	0.34±0.03 $^{a**\ b**}$	0.24±0.03 $^{a\ NS}$
N-acetyl βD-glucosamidase	0.52±0.03	1.26±0.14 ^{a***}	0.61±0.5 $^{a**\ b***}$	0.51±0.04 $^{a\ NS}$

Each value represents mean±S.E.M of six individual animals. Significance: ***p<0.001, **p<0.01, *p<0.05, NS - Not significant.

Table 4: Activities of some marker enzymes in kidney of control and experimental animals.

Particulars	Group I (Control)	Group II ($HgCl_2$)	Group III ($HgCl_2$+ *H. auriculata*)	Group IV (*H. auriculata*)
γ-glutamyltranspeptidase	1.77±0.15	0.94±0.08 ^{a***}	1.36±0.14 $^{a*\ b**}$	1.64±0.14 $^{a\ NS}$
β-glucuronidase	1.69±0.13	1.14±0.16 ^{a***}	1.42±0.16 $^{a*\ b**}$	1.58±0.17 $^{a\ NS}$
N-acetyl β D-glucosamidase	1.76±0.14	1.04±0.14 ^{a***}	1.37±0.15 $^{a*\ b**}$	1.72±0.16 $^{a\ NS}$
Cathespin D	2.24±0.23	1.41±0.16 ^{a***}	1.91±0.18 $^{a**\ b**}$	2.12±0.25 $^{a\ NS}$
LDH	1.62±0.12	1.31±0.14 ^{a***}	1.54±0.16 a $^{a**\ b*}$	1.63±0.17 $^{a\ NS}$

Each value represents mean±S.E.M of six individual animals. Significance: ***p<0.001, **p<0.01, * p<0.05, NS - Not significant.

Table 5: The levels of glycoproteins in kidney of control and experimental animals.

Parameters (mg/ g wet tissue)	Group I (Control)	Group II ($HgCl_2$)	Group III ($HgCl_2$+ *H. auriculata*)	Group IV (*H. auriculata*)
Hexose	19.67±1.87	24.98±2.15 ^{a***}	21.10±2.12 $^{a**\ b**}$	18.42±1.75 $^{a\ NS}$
Hexosamine	2.65±0.24	6.54±0.67 ^{a***}	4.31±0.39 $^{a**\ b**}$	3.21±0.37 $^{a\ NS}$
Sialic acid	0.46±0.03	0.67±0.07 ^{a***}	0.42±0.03 $^{a**\ b*}$	0.44±0.02 $^{a\ NS}$

Each value represents mean ± S.E.M of six individual animals. Significance: ***p<0.001, **p<0.01, * p<0.05, NS - Not significant. a-compared with group I and b-compared with group II.

DISCUSSION

Irrespective of the dose and time of exposure, the kidney serves as the major target site of toxicity, leading to ultrastructural and morphological abnormalities.[19] After the experimental period, the body weight of $HgCl_2$ administered rats was found to be lesser than that of the control animals.[19] These findings are in accordance with recent toxicological evaluations emphasizing heavy metal-induced wasting syndromes.[20] Previous evaluations have shown that increase in renal mass due to $HgCl_2$ induction is a result of tissue fluid retention and an adaptive response to maintain normal renal function.[21-22] Furthermore, mercury selectively damages the pars recta segment causing widespread cellular necrosis, proliferations of smooth endoplasmic reticulum, mitochondrial swelling, and calcification.[24] In our investigation the rats treated with H. auriculata after $HgCl_2$ administration does not show

increase in kidney weight, but body weight was gained, which was contrary to what we have observed in $HgCl_2$ administered rats.[4] Besides tubular damages, $HgCl_2$ can also injure the glomerular cells or induce autoimmune reactions leading to functional decline as observed via increased levels of urea, uric acid, creatinine, and protein after the HgCl2 exposure.[19,25]

Urinary excretion of nitrogenous waste products (urea and uric acid) and creatinine were increased significantly in mercury administered rats coupled with proteinuria.[19] Elevation of urinary urea and uric acid might be a consequence of impaired solute transport in the proximal tubules.[19] Rejection of solutes in the proximal tubule would retard water reabsorption and ultimately enhance fluid delivery to the distal nephron, increasing the driving force for urea and uric acid reabsorption.[19] Creatinine serves as an index for renal function, and increased urine creatinine levels reflect underlying tissue damage, filtration failure, and altered glomerular filtration rate.[19,20]

Proteinuria is a sensitive indicator for renal damage.[25] $HgCl_2$ treated rats developed heavy proteinuria, indicating extensive renal damage due to the direct local toxic effect on the kidney, reflecting a loss of normal glomerular permeability and diminished proximal tubular reabsorption capacity.[24,25] Tubular injury is heavily attributed to increased oxidative stress imposed by mercury, accelerating lipid peroxidation and depleting endogenous antioxidant defenses.[23] Renal enzyme excretion increases markedly when tubular cells disintegrate or when tubular permeability is disturbed.[24] Urinary excretion of enzymes such as NAG and LDH acts as a main indicator for tubular degeneration caused by nephrotoxic agents.[20,24]

Early change in the kidney after chemical insult results in alteration of the brush-border enzymes.[21] ALP, a membrane-bound enzyme, shows high activity in the brush-border membrane, and its alteration affects membrane permeability and disrupts metabolite transport.[21,24] A decrease in ALP activity in renal tissue correlates inversely with an increase in its activity in the urine due to leakage from damaged proximal tubules.[19,21] Similarly, alterations in ACP, gamma-GT, and lysosomal enzymes reflect the severity of membrane breakdown and cellular necrosis under heavy metal exposure.[24,25]

The renal integrity assessment following treatment with ethanolic extract of *H. auriculata* showed marked improvement with respect to the enzyme activities thus indicating the membrane stabilizing effect.[22] The basic mechanism underlying the nephroprotection of H. auriculata may involve chelation with Hg to prevent its interaction with cellular sites and/or maintenance of membrane integrity by providing endogenous sulfhydryl groups.[19,22] Treatment with *H. auriculata* resulted in modulating the activity levels of the various enzymes in a direction opposite to that of mercury, bringing them to levels near normal to that of control rats and minimizing adverse toxic side effects.[22] From this study, it may be suggested in general that *H. auriculata* may be used as an effective protective agent against nephrotoxicity.[19,22]

Limitations

This study is limited by the absence of direct oxidative stress markers (MDA, GSH, SOD), tissue mercury accumulation tracking, and histopathological imaging to visually confirm proximal tubular repair. Furthermore, the brief 10-day timeline and lack of a standard pharmaceutical reference group (e.g., DMSA) restrict evaluations of long-term recovery and comparative efficacy. Moving forward, subsequent studies will integrate these precise biochemical assays, metal-tracking metrics, and ultrastructural imaging techniques over extended timelines to fully validate the molecular clearance mechanisms and clinical translational potential of *H. auriculata*.

CONCLUSION

In conclusion, administration of mercuric chloride induces significant nephrotoxicity, characterized by body weight reduction, increased kidney weight, elevated urinary nitrogenous waste products (urea and uric acid), proteinuria and marked alterations in both urinary and brush-border membrane enzyme activities. These toxic effects are primarily driven by severe oxidative stress, lipid peroxidation, and direct cellular necrosis within the proximal tubules. However, post-treatment with the ethanolic extract of *H. auriculata* successfully reverses these pathological changes, restores membrane integrity, improves antioxidant defenses, and brings functional parameters back toward normal control values. Therefore, this study conclusively demonstrates that H. auriculata possesses potent renoprotective properties and acts as an effective therapeutic agent against heavy-metal-induced nephrotoxicity.

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